The principles of surgery : clinical, medical, and operative an original analysis of pathology systematically conducted, and a critical exposition of its guidance, at the bedside and in operations ... / by Frederick James Gant.

Contributors

Gant, Frederick James, 1825-1905.

Publication/Creation

London : J. Churchill, 1864.

Persistent URL

https://wellcomecollection.org/works/szfqam2z

License and attribution

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

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

wellcome collection

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

Digitized by the Internet Archive
in 2014

https://archive.org/details/b20390208

60667

THE

PRINCIPLES OF SURGERY;

CLINICAL, MEDICAL, AND OPERATIVE.

An Original Analysis of Pathology Systematically Conducted,

AND

A CRITICAL EXPOSITION OF ITS GUIDANCE, AT THE BEDSIDE AND IN OPERATIONS.

REPRESENTING THE

PRINCIPLES OF THE EARLIEST AND MOST EXACT DIAGNOSIS, ETIOLOGY, PROGNOSIS, AND THERAPEUTICS, MEDICAL AND OPERATIVE.

BY

FREDERICK JAMES GANT, F.R.C.S.,

SURGEON AND PATHOLOGICAL ANATOMIST TO THE ROYAL FREE HOSPITAL;
FORMERLY CIVIL STAFF SURGEON TO HER MAJESTY'S MILITARY HOSPITALS, CRIMEA AND SCUTARI;
AND ASSISTANT CURATOR OF THE MUSEUM OF ANATOMY AND PATHOLOGICAL ANATOMY,
UNIVERSITY COLLEGE, LONDON.

LONDON:

JOHN CHURCHILL AND SONS, NEW BURLINGTON STREET.

MDCCCLXIV.

113~9359

M19231

WELLCOME INSTITUTE LIBRARY
Coll. welMOmec
Call No. WO100 1864 G21p

LONDON:
SAVILL AND EDWARDS, PRINTERS, CHANDOS STREET,
COVENT GARDEN.

PREFACE.

THE foundation and object of this work—as severally stated in the title-page, and its contents as detailed in the following Prospectus—are conspicuously different from those of other works entitled "Principles" or "Principles and Practice" of Surgery, and Systematic works of similar purpose if not so named; all of which, however excellent and valuable in their way, are systematic compilations of "General Pathology and Therapeutics."

The distinctive Scholastic aspect alluded to is further explained in the Historical "Introduction,"—which briefly defines the past epochs of Surgery and the character of the present age.

I have, therefore, no apology to make for this innovation, as if its design were that of works hitherto published and had already been accomplished. But I do submit its merits to the judgment of others.

Unlike any course of General Pathology and Therapeutics, the Principles I have advanced are coextensive with Surgery (and Medicine concurrently) in its four Departments:—Diagnosis; Etiology, with regard to 'internal' causes and their operation; Prognosis; and Therapeutics—medical and operative. "All of them may be called Principles when compared with a thousand other judgments which are formed under the regulation of these primary propositions."* Collectively, they represent and express the Guidance of Pathology at the Bedside and in Surgical Operations and manipulations; and *Clinical* Pathological Anatomy, as I have named this latter science, when thus applied during

* "Principle:" Johnson's Dictionary.

life, is shown to give birth to Principles, and of the *earliest* and *most exact* standard, in each of the aforesaid Departments of Surgery.

Until recently, Pathological Anatomy stood alone, as mere Morbid Anatomy, dead among the Sciences. Is its *Power* yet understood?

The Principles *thence* derived impart a Preventive and Conservative character to the Practice of Surgery,—as also explained in the "Introduction,"—extending to that of Medicine. Conservatism—as therein defined—presupposes the existence and operation of, what may be aptly named, an innate Restorative Power,—manifested by the *natural* course and tendency of injuries and diseases, individually, to, or towards, recovery. This also is a branch of Pathological knowledge only just dawning on Rational Medicine, and which I have endeavoured to develope as another prominent feature of these Principles. But does Pathology impart the Conservative character to our proceedings *during* Surgical Operations? Assuredly, if not obviously, it does this also.

The Operations of Surgery were formerly regarded as *purely* Anatomical performances. The "System of Operative Surgery, founded on the basis of Anatomy," by Sir Charles Bell, was a type of the period to which I refer. At length the influence of *diseased* and *living* conditions became apparent, and, like a dissolving view, the purely Anatomical era passed away, and the Pathological epoch began. Slowly, as out of a mist, the Pathological Man, clad in the tattered garb of disease, approached, and crept by stealth into the Anatomical theatre. The teaching of the Schools and their books on Operative Surgery thenceforth underwent *some* improvement, and advanced *somewhat* nearer to the requirements of the Practical Surgeon.

Still, however, freighted with relics and mummeries of the dissecting-room, these works abounded with details of the purely Anatomical method, and were garnished, not to say adorned, with pictures that bespoke their antiquated origin. The rude designs

of the Gothic style were visible beneath the facings of the modern building; and in the construction of even the most recent manuals of Practical Surgery in this country, the basis is, I conceive, too purely Anatomical.

The alliance of 'Surgical Operations' with a series of 'Dissections' is scarcely less unnatural than would be a marriage of the living with the dead; and as ill-assorted is the association of dissecting instruments with *surgical armamentaria*. Yet such are the unavoidable inconsistencies that the Anatomical method would impose upon us.

Books of the class to which I allude would be less inconsistent were they intended solely for the use of those who may require to gain a *mechanical* knowledge of Operative Surgery, so far as this can be learnt in the dissecting-room, by handling instruments, making incisions of various shapes, and performing other simple mechanical operations on parts presenting their healthy relations to each other, and bereft of their living appearances and conditions; but the guidance of (pure) Anatomy acquires a positively *fatal* tendency when practices on the dead subject, and matured perchance in the dissecting-room, are imported into Surgical works, and are ostensibly converted into (Surgical) Operations on parts in a diseased state, and as found in living patients.

This evil inclination to the guidance of Anatomy alone is gradually eradicated, and the application of Pathology during Surgical Operations and manipulations acquired, by repeated observation of the peculiar relative position, physical properties, and living conditions presented by textures and parts, as successively seen and felt during Surgical operations, and not as they are exposed by dissection in the dead subject. True it is, that certain exceptional operations—*e.g.*, amputations and the ligature of arteries for aneurism—are executed at a distance more or less remote from the diseased or injured part; but even such operations have been fashioned too much after the dead model—an error as grave as were a sculptor to presume to chisel the proportions of the living body by glances at a dead subject. In short—

to conceive another metaphor—Surgical Operations, learnt from practice on the *dead* subject, are as if an artist were to study a landscape in the *winter*, when the outlines only of the face of Nature are seen: the fulness of verdure is wanting to completely realize the picture; every ice-bound form appears stiff and lifeless, and every rivulet is still; while the order and regularity of *healthy* forms and arrangement of parts, as contrasted with the disorder and disorganization wrought by *disease* or inflicted by *injury*, is as if this same landscape—lifeless—presented the normal undulations of hill and dale, instead of the chaotic disarrangement of relative position, &c., left by an earthquake.

The overshadowing influence of *living* conditions in *all* cases; and their combination, in *most* Surgical operations, with *morbid* conditions of relative position of parts, and physical properties of the textures concerned,—are indeed modifying, if not effacing characters, which correct the purely Anatomical impressions of the Student, and can alone safely guide the Operating Surgeon.

Now this Pathological method of operating represents the *least* amount only, no less than the kind, of instrumental and manipulative proceedings necessary; and thus fulfils the Conservative character *during* Surgical operations.

If, then, the Practice of Surgery (as an Art) be guided, regulated, and determined by Pathology; the Principles which collectively express this Guiding Power, in the preparatory and absolutely essential knowledge of Diagnosis, Etiology, and Prognosis, tend—especially by virtue of their earliness and exactitude—to elevate Surgery (and Medicine, unitedly,) to the rank and dignity of a Science.

Such being the design of this work, its Educationally distinctive purpose is obvious.

While, however, professing to discover and inculcate Principles, properly so called; the Logical processes of Analysis, Comparison, Induction, and Generalization, by which they are discovered, exhibit the course of all Physical Investigation,—the

very root of Medical Education, in contrast with that of the present system of Medical Teaching and competitive Examination, —*i.e.*, the cultivation of only *one* faculty of the mind, that of memory, and perhaps also the power of observation. The faculties which are peculiarly elicited and strengthened by the process of Physical Investigation, are overlooked; or if *their* cultivation be ostensibly provided for, by virtue of such branches of knowledge as Comparative Anatomy, Botany, &c., those mental faculties may be *equally well* educated and trained by the systematic acquisition—otherwise by memory alone—of Medicine and Surgery; more time being thus gained for *Clinical* observation; on the one hand, without prolonging the *duration* of Medical Education; on the other, without unnecessarily extending the *boundary* of such Education,—thus relieving the high-pressure work now imposed on Students in Medicine.

Moreover, this philosophic view of Medicine would no less tend to form that *Scientific* character of mind, which is itself the almost distinctive prerogative of the Medical Profession. And now perhaps, more than ever, *this* distinction should be cherished, to regulate the otherwise profligate, as well as prolific, propagation of 'Special' branches of Practice, and to nourish their otherwise sickly growth, when dismembered from the parent trunk of Medicine.

I am, indeed, not without hope that this innovation will supply a systematic course of Discipline for the investigation of Medicine as a Science; while, as the Principles of Surgery (and of Medicine unitedly), it will prove also a faithful Guide to the Student and Practitioner.

F. J. Gant.

March, 1864.

*** Having so frequently had occasion to consult original works and monographs on Pathology and Surgery, in the valuable library of the Royal College of Surgeons, I gladly avail myself

of this opportunity to publicly express my acknowledgments to Mr. Chatto, the Librarian, for aiding me with his extensive bibliographic knowledge, and for his obliging attention during a series of years.

The drawings from which the wood-engravings in this work were chiselled are from the pencil of Mr. Walter G. Searson. They cannot fail to extend his repute as an Anatomical and Microscopic Draughtsman. No less will the engravings themselves, by Mr. Hart, assuredly sustain his already well-known and deservedly-high reputation.

PROSPECTUS OF CONTENTS.

THE PRINCIPLES OF SURGERY.

Introduction. (p. 1—18.)

Historical Review of Medicine and Surgery—Origin of Pathological Surgery—The distinctive foundation and object of this Work—Principles are attained by the analysis of General Pathology, and are coextensive with all Diagnosis—Etiology, respecting internal causes and their operation—Prognosis and Therapeutics: such Principles are *earliest* and *most exact:* their Preventive and Conservative character explained; they extend to Medicine, and tend to elevate Medicine and Surgery unitedly to the rank of a *Science;* while, by their character, they are eminently Practical.

The Elements of Pathological Anatomy; being the foundation of the Principles advanced in this Work, are systematically described—The author's Tabular view of General Pathology. (p. 19—142.)

PATHOLOGICAL SURGERY.

THE PRINCIPLES OF CLINICAL SURGERY, OR THOSE OF DIAGNOSIS, ETIOLOGY, AND PROGNOSIS.

Prelude. (p. 143—150.)

The Surgeon at the bedside—The Preliminary Questions to be determined on behalf of Diagnosis—Etiology, respecting internal causes and their operation; and Prognosis—Their Pathological character, and mutual relation to rational Therapeutics—The *earliest* and *most exact* solution of the Questions alluded to, and the establishment of corresponding Principles, in each of these Departments of Surgery, by the Analysis of Pathology, will become apparent in the course of this Work—The immediate bearing of these Pathological

Principles on the Preventive and Conservative Practice of Medicine and Surgery re-explained and enforced.

The Analytical Method of Investigation explained by reference to the Principles of Diagnosis.

THE PRINCIPLES OF DIAGNOSIS,

OR OF THE 'EARLIEST' AND 'MOST EXACT' DETECTION AND DISCRIMINATION OF THE STRUCTURAL CONDITION, SITUATION, AND EXTENT OF DISEASES AND INJURIES.

NEGATIVE PRINCIPLES.

CHAPTER I. (p. 151—168.)

Anatomy and Physiology are severally incompetent Guides to an Early and Exact Diagnosis:

This Principle illustrated by the Diagnosis of Wounds, and by that of Burns.

CHAPTER II. (p. 169—188.)

Pathology—Functional Disturbances only—is an insufficient guide to an early and exact Diagnosis.

Functional Disturbance does not *invariably* accompany, and does not at the *earliest* period accompany, injury or local disease; and the same disturbance of Function is no measure of the structural lesion existing, may also accompany the same injury or disease simultaneously in different *parts* of the body, or may accompany very different *kinds* of injury or disease. Functional conditions, therefore, are only symptoms—*i.e.*, casual coincidences, and not exact signs of morbid conditions.

This *negative* Principle illustrated by the Diagnosis of Injuries of Mechanism—*e.g.*, fractures, aneurisms, dislocations, respectively; also by Diseases of Nutrition—*e.g.*, Inflammation of internal parts, as strangulated herniæ and ileus; tumours of various kinds; and degenerations of textural structure.

POSITIVE PRINCIPLES.

CHAPTER III. (p. 188—210.)

Pathological Anatomy advanced as the Guide, during life, to the *earliest* and *most exact* Diagnosis.

The Diagnostic value of this fundamental Principle illustrated by the diagnosis of the several varieties of Fracture and Aneurism, respectively.

Chapter IV. (p. 210—224.)

Pathological Anatomy applied during life to supply the earliest and most exact Diagnosis.

The Diagnostic value of Clinical-Pathological Anatomy illustrated by the Diagnosis of the varieties of Dislocation.

Chapter V. (p. 224—243.)

Clinical Pathological Anatomy, and its Diagnostic Guidance.

Pathological Anatomy applied during Life, through Physical Signs, to supply the earliest and most exact method of Diagnosis.

The value of Physical Diagnosis illustrated by the Diagnosis of Morbid Products of Nutrition—*e.g.*, Fatty Tumours, Cysts, and Cystic Tumours, respectively.

The *insufficiency* of Physical Signs as a Guide to the earliest and most exact Diagnosis.

Physical Properties are most readily recognised during life at the earliest period of disease (or injury); and, therefore, at a time when the morbid condition itself is least complicated, and most remediable by the simplest measures; but such properties are nevertheless most equivocal (or inexact) signs of disease. Unaided Physical Diagnosis therefore fails in the practice of Medicine and Surgery.

This general Principle illustrated by the diagnosis of Morbid Products of Nutrition, as, quasi-malignant varieties of non-malignant Growths—*e.g.*, recurring-fibroid and fibro-nucleated Tumours, respectively.

Chapter VI. (p. 243—250.)

The Diagnostic Guidance of Clinical Pathological Anatomy (continued).

The application of Pathological Anatomy during life (continued) by Structural characters; and the *superior* value of their guidance to supply the earliest and most exact Diagnosis.

Changes of minute Structure can be detected at the earliest period, during life, by puncturing Growths and Deposits (situated near the surface of the body), and examining with the microscope these and all other Morbid Products found in Discharges and Secretions issuing from internal parts and organs.

Moreover, the alterations of Structure thus detected are more constant, in any given case, than alterations of Physical Properties, and are therefore more exact signs of the morbid condition in question; and being also discoverable at the earliest period,—before the supervention of complications,—are thereby best adapted to supply the chief Method of Diagnosis, whenever such minute alterations of Structure are available.

The value of this general Principle illustrated by the earliest and most exact Diagnosis of Morbid Products of Nutrition, as Growths and their respective varieties; *e.g.*, Fibrous Tumours—including the *fibro-nucleated* and *recurring-fibroid varieties:* Cartilaginous Tumours—including the *myeloid* and *recurring varieties;* Cancer—*encephaloid, scirrhus,* and *colloid.*

The Law of Structural Retrogression applied.

Chapter VII. (p. 250—267.)

THE DIAGNOSTIC GUIDANCE OF CLINICAL PATHOLOGICAL ANATOMY (CONCLUDED).

The application of Pathological Anatomy during life (concluded) by Chemical conditions.

The *potential* value of Chemical Pathology, to supply the earliest and most exact Diagnosis.

Alterations of Chemical Composition are, indeed, the most essential condition of Disease, but also the least readily detected and identified. This general Principle illustrated by Deposits of lymph,—forming false tissues, pus, tuberculous or scrofulous matter; and by the present state of Pathological Chemistry in general.

The *practical* incompetency of Chemical Analysis to supply the earliest and most exact Diagnosis; illustrated by the Diagnosis of several kinds of Blood-diseases—*e.g.*, Scurvy; symmetrical diseases, and diseases having seats of election.

The *present* value of Chemical Analysis to supply the earliest and most exact Diagnosis, illustrated by the diagnosis of certain Blood-diseases—*e.g.*, Cholera, Gout, and Rheumatism.

THE PRINCIPLES OF ETIOLOGY,

OR OF THE 'EARLIEST' AND 'MOST EXACT' DETECTION AND DISCRIMINATION DURING LIFE OF THE CAUSES OF DISEASES AND INJURIES, INDIVIDUALLY AND OF THE OPERATION OF SUCH CAUSES.

Prelude. (p. 268—275.)

The causes of injuries and diseases are 'external' or 'internal;' and either may be predisposing or immediate in their operation.

Internal causes are 'local' or 'constitutional' morbid conditions.

The earliest and most exact Etiological knowledge respecting the *operation* of internal causes can be concluded only by special Clinical observation of their Functional manifestations; but such knowledge implies the previous detection and identification of these causes, and a prior Diagnosis therefore of similar quality.

Hence the guidance of Clinical Pathological Anatomy to Etiology in respect of 'internal' causes.

Pathological Anatomy being the guide to the earliest and most exact detection and discrimination, during life, of Diseases and Injuries, respectively, suggests the Principle of Prevention in the Practice of Medicine and Surgery.

CHAPTER VIII. (p. 275—431.)

LOCAL MAY PROCEED FROM CONSTITUTIONAL MORBID CONDITIONS.

This general Principle of Etiology illustrated by the manifestations of Blood-diseases in their appropriate 'textures.'

1. In many textures—Pervading Blood-diseases—Syphilis, Scrofula, Scurvy, Purpura. Growths—malignant. Degenerations.
2. In special textures.

Skin, Mucous Membranes, or both.

Fevers—Non-infectious: Inflammatory, hectic, gangrenous typhoid.
Infectious: eruptive (exanthematous), typhus, typhoid, relapsing, measles, scarlatina, small-pox, chicken-pox, erysipelas, plague.

Mucous Membranes.

Diseases, infectious—Hooping-cough, Influenza, Cholera (Asiatic).

Cellular texture—Subcutaneous.

Cellulitis, Carbuncle, Boil.

Fibrous textures and Synovial Membranes.

Rheumatism, Gout.

The Pathological Principles which guide the Preventive view of these Diseases.

Chemical Pathology *applied* to determine how far it is possible, in the present state of knowledge, to more early and exactly detect and discriminate these Blood-diseases, so as to prevent their local manifestations, by Rational Preventive measures, more appropriate and effectual than those which Experience alone may have suggested.

Pathological Anatomy of these local manifestations *applied* to more early and exactly detect and discriminate the Blood-diseases from which they respectively proceed, so as to prevent those of *infectious* origin being propagated.

Preventive Measures.

Local may proceed from Constitutional morbid conditions; concluded by Injuries and Diseases of the Central Nervous System, in relation to disorder of the Heart and circulation; the Lungs and respiration; the Chylopoietic viscera and the process of digestion; the Organs of Excretion—kidneys and bladder, skin—and the functions severally of these organs; diseases of Nutrition; organs of Reproduction and their functional derangements.

CHAPTER IX. (p. 432—445.)

CONSTITUTIONAL MAY PROCEED FROM LOCAL MORBID CONDITIONS.

This general Principle of Etiology, and the Principle of Prevention also, in a measure, illustrated by Injuries of Mechanism, and their constitutional effects —Shock (collapse, prostration), Reaction; Prostration with excitement, and Tetanus; respectively following Wounds, Burns, Fractures, and Dislocations.

The Pathological Principle which guides the Preventive view of these Constitutional Disturbances.

This presupposes a sufficiently early and exact detection and discrimination—Diagnosis of Injuries regarded as internal causes in operation.

Preventive Measures.

CHAPTER X. (p. 446—658.)

THE ETIOLOGICAL PRINCIPLE ADVANCED IN THE PRECEDING CHAPTER FURTHER ILLUSTRATED BY DISEASES OF NUTRITION, THOSE OF OTHER FUNCTIONS, AND BY CONTAGION; SEVERALLY, IN RELATION TO THEIR CONSTITUTIONAL CONSEQUENCES.

Various (Constitutional) Disturbances of the Blood, Nervous, and Vascular Systems, *i.e.* Fevers—inflammatory, hectic, and gangrenous typhoid—arise from inflammation, suppuration, and mortification, respectively.

Various (Constitutional) Blood-diseases arise from other perversions of Nutrition besides that of inflammation; also from those of the Digestive process, Excretion, and Respiration, respectively.

Various (Constitutional) Blood-diseases arise from Contagion, illustrated by—
Contagious matter derived from Animals:—Hydrophobia, Snake-bites, Malignant Pustule, Glanders, Vaccinia.
Contagious matter derived from the Human Species:—Hospital Gangrene, Puerperal Fever, Primary Syphilis—Chancres and Buboes.
The Pathological Principles which Guide the Preventive view of these Diseases.
This presupposes a sufficiently early and exact detection and discrimination —Diagnosis, of each one.
Preventive Measures.

Chapter XI.

LOCAL MAY PROCEED FROM LOCAL MORBID CONDITIONS; OR, THE RELATION OF DISEASES OF NUTRITION—*e.g.*, INFLAMMATION AND OF LOCAL NERVOUS AND MUSCULAR AFFECTIONS, RESPECTIVELY, TO THOSE OF PARTS (CONTINUOUS) CONTIGUOUS, AND REMOTELY SITUATED IN THE BODY. (p. 659—677.)

The Pathological Principles which guide the Preventive view of some of these Morbid Conditions.
Pathological Anatomy applied, during life, to most early and exactly detect and discriminate the structural condition, situation, and extent of diseases (and injuries), regarded as internal causes, in order to the Prevention of their consequences,—by the extension of such local morbid conditions to textures and organs (continuous and) adjoining, and the sympathetic affections of organs remote; also the disorganizing results in the texture or part first affected. Illustrated by inflammatory diseases of the joints, thus considered.
Preventive Measures of some of these morbid conditions.

CONSTITUTIONAL MAY PROCEED FROM CONSTITUTIONAL MORBID CONDITIONS; OR, THE RECIPROCAL RELATION OF MORBID CONDITIONS OF THE NERVOUS AND VASCULAR SYSTEMS. (p. 677—689.)

General theory of Fever propounded by reviewing Constitutional Irritation—Shock with reaction, as compared with Inflammatory fever, Eruptive fevers, and Endemic fever—intermittent and remittent.
The Pathological Principles which guide the Preventive view of these Diseases concluded.
This, as usual, presupposes a sufficiently early Diagnosis.
Preventive Measures, concluded.

SUMMARY OF ETIOLOGY, AS GUIDED BY PATHOLOGY. (p. 689—695.)

The association of 'internal causes'—their various co-operations and orders of succession in the animal economy.

The Preventive view of such causes is consummated by the earliest and most exact detection of their manifold relations.

THE PRINCIPLES OF PROGNOSIS,

OR OF THE 'EARLIEST' AND 'MOST EXACT' FOREKNOWLEDGE OF THE COURSE AND TENDENCY OF DISEASES AND INJURIES, INDIVIDUALLY, TO, OR TOWARDS, A FAVOURABLE OR AN UNFAVOURABLE ISSUE.

Prelude. (p. 696—700.)

The earliest and most exact foreknowledge respecting the course, tendency, and issue of injuries and diseases, can be *concluded* only by the experience derived from special Clinical observation of their continued Functional manifestations; but such Prognosis implies a previous Etiology, in respect of internal causes, and a prior diagnosis therefore, each of similar quality. Hence the guidance of Clinical Pathological Anatomy indirectly to Prognosis.

Chapter XII. (p. 700—721.)

THE PROGNOSTIC GUIDANCE OF CLINICAL PATHOLOGICAL ANATOMY.

Persistence, or not, of the immediate cause or causes, and therefore the question of an 'internal' cause, or more, being in operation, is the *immediate* foundation of the earliest and most exact Prognosis, unfavourable or favourable respectively.

The Prognostic value of this fundamental Principle illustrated by the career of injuries and diseases, from the whole range of General Surgical Pathology.

Chapter XIII. (p. 722—728.)

THE PROGNOSTIC GUIDANCE OF CLINICAL PATHOLOGICAL ANATOMY (CONCLUDED).

The earliest and most exact Prognosis of an injury or of a disease is *regulated* by the kind and extent of *structural* alteration that the organ or texture has undergone; and, moreover, by the period during which such alteration of structure has been in operation as an internal cause of functional disturbances of the System. Nevertheless, an acute disease, or a recent injury, is, *cæteris paribus*, more unfavourable than a chronic lesion, to which the system has become habituated.

The Prognostic value of the foregoing fundamental Principle illustrated by Injuries—wounds, other than simple (incised); fractures and dislocations, respectively, other than simple; and by aneurism—diffused. Also by diseases of Nutrition—results of inflammation; Growths—malignant.

CHAPTER XIV. (p. 729—747.)

THE SUPERIOR PROGNOSTIC GUIDANCE OF PATHOLOGY.

Pathology declares the course, tendency, and issue of injuries and diseases by their continued Functional manifestations.

Principle I.—The comparative Functional importance of any organ or texture *determines* our Prognosis, unfavourable or favourable, as to the course and tendency of any morbid condition of structure it may have undergone.

Sub-Principle I.—Organs and textures which fulfil Functions by virtue of their vital endowments (and chemical composition) suggest an unfavourable Prognosis.

The Prognostic value of this Principle and sub-Principle illustrated by the course and tendency of Blood-diseases—*e.g.*, Syphilis, Scrofula, Gout, and Rheumatism; also, by disorders of the Nervous System—*e.g.*, Constitutional Irritation; by disorders of the Nervo-Muscular System—*e.g.*, Tetanus; and by disorders of Secretion and Nutrition.

Sub-Principle II.—Organs and textures which fulfil Functions by virtue of their physical properties and mechanism suggest a more favourable Prognosis.

The Prognostic value of this sub-Principle illustrated by the course and tendency of Injuries—*e.g.*, incised flesh Wounds; reduced simple Fracture and Dislocation.

Principle II.—Local disease or injury, *per se*, suggests a favourable Prognosis.

Principle III.—Local disease or injury, sustaining, or sustained by, some Constitutional disorder, suggests an unfavourable Prognosis.

The Prognostic value of this Principle illustrated by the course and tendency of unreduced Fractures and Dislocations; also by persistent Inflammation; and by poisoned Wounds.

Principle IV.—Constitutional diseases, implying each some morbid condition of a texture or textures, of general distribution, as well as of predominant Functional influence throughout the system, suggest an unfavourable Prognosis.

The Prognostic value of this general Principle illustrated by the course and tendency of the same diseases of the Blood, disorders of the Nervous System, Nervo-muscular System, and of Nutrition, already adduced in illustration of the first Principle advanced in this chapter.

Modes of Dying.

Chapter XV. (p. 748—826.)

THE RESTORATIVE POWER,—ITS EXISTENCE, OPERATION, AND RESOURCES,—MANIFESTED BY THE NATURAL COURSE AND TENDENCY OF INJURIES AND DISEASES, INDIVIDUALLY, TO, OR TOWARDS, RECOVERY.

This Principle illustrated by selections from the whole range of General Pathology.

The Restorative Power an innate property of the living body—Noticed by Hippocrates and by subsequent clinical observers—Van Helmont, Stahl—"Expectant cure" of the French School—German School—Acknowledgment of this Power in the practice of Medicine and Surgery, opposed by certain natural prejudices, specified—Prejudices remarked by John Bell and Bigelow—Mistrust of Nature by the influence of authority—Teaching of the Schools, Medical journals, Systematic works, and Medical literature generally—These fetters are shaken off by independent observation and reflection—Concessions of matured experience.

Definition of the Restorative Power, not identical with the physiological power of maintenance in the state of health and the prevention of disease—The power of reparation a phase of the Restorative Power, and not identical with that of nutritive maintenance, as alleged by Mr. Paget—His reasons answered, and the distinction of these two Powers illustrated—The power of reparation not necessarily identical with that of original formation, as alleged by Mr. Paget—His reasons answered.

The Restorative Power, including its reparative phase, not to be predicated by any knowledge of Physiology—Evidence of the existence, operation, and resources of this Power discovered only, and its conservative value appreciated only, by independent Pathological observation.

Indirect and inferential evidence, in eight general propositions, by Sir John Forbes.

The author's direct evidence in serial illustrations of the natural course and tendency of injuries and diseases, severally to, or towards, recovery:—

Healing of Wounds:—immediate union, primary adhesion, suppurative granulations, scabbing—Modelling process of Macartney—Healing of Fractures, simple, compound—Healing of Dislocations, reduced, simple, unreduced, and natural formation of a new joint; healing of compound dislocation—General conclusions respecting the healing of non-contused, as compared with contused, lesions; and of subcutaneous, as compared with open, lesions.

Development of new capillary blood-vessels—Spontaneous cure of aneurism—Natural modes of cure specified and exemplified—Healing of wounded arteries, when incised or lacerated—either kind of lesion being partial or complete.

Inflammation not a salutary mode of action—John Hunter's statement examined and disproved—The author's view—Inflammation is essentially a morbid condition, but a manifestation of restorative power, by virtue of its purpose or intention, whenever an extra and pressing demand is made on Nutrition, either to construct some new mechanism, or to restore some loss of structure—False membranes and other false tissues—Adhesive inflammation and its uses—Suppurative granulations with cicatrization, its uses—Sloughing, its uses.

Non-inflammatory lymph-deposits—Degeneration and quiescence of tubercle, grey and yellow—Disintegration and destruction of texture around tuberculous matter—its dislodgement and evacuation—a manifestation of restorative power—Obliteration of the cavity thus formed—Professor Bennett's observations—Natural cure of phthisis—New Growths, their natural history—Arrest of Cancer, different modes specified and illustrated.

Blood-diseases are "self-limited;" their natural course and tendency, to, or towards, recovery—Exanthematous Fevers, each a natural effort to expel some blood-poison—Cardiac reserve force and reaction from the shock of injury, analogous to reaction as a restorative effort against febrile oppression of the nervous system—Illustration of the self-sufficiency of the cardiac reserve force—Nervous reserve force as a restorative effort, illustrated by involuntary muscular actions connected chiefly with the functions of Respiration and Digestion—The respiratory efforts of sighing and yawning, to restore the balance of respiration whenever disturbed—Coughing and expectoration, a deep expiratory effort to clear the air-passages and recover a due supply of air—Sneezing, a similar effort on behalf of the nasal passages—Involuntary muscular actions associated with digestion—Vomiting a restorative effort, by which substances unfit for assimilation, or poisonous, are ejected—Diarrhœa, feculent, serous, mucous, bilious, an analogous effort to clear the intestinal canal—This canal also an excreting organ to eliminate blood-poisons—Peyer's glands in typhoid fever.

Hunger and Thirst, considered as sensations indicative of the food requirements of the system in disease—The quantity required regulated by instinctive desire—a sure guide—Loathing, and ravenous appetite—Instinctive choice of this or that kind of food—an infallible guide—Food-fancies in protracted illness, and of the moribund, suggested by the inward want of that special kind of food which the particular disease has rendered necessary—Systemic wants and stomach capabilities alike suited by these selective suggestions of Nature—Remarkable instance by Dr. William Hunter.

Excreting organs manifest restorative power by extra excretion, readjusting excretion, compensatory or vicarious excretion, and supplementary excre-

tion; double work—Examples of these restorative operations by the kidneys and skin respectively.

Organs of reproduction, in the female, considered during the active period of their career—Menstruation—'Ovular' theory doubtful—The author's objections—An excrementitious discharge is probably one physiological purpose of this function—is supported by pathological evidence—Vicarious menstruation—examples of this compensatory discharge—Menorrhagia, a restorative effort by excrementitious discharge in extra quantity, or to readjust the balance of the blood's composition?—The relation of human pathology to human physiology alluded to.

The final aspect of the Restorative Power—the readjusting power of Nature, during convalescence: illustrated by convalescence after natural labour—This process traced constitutionally, and, as regards the uterus, regaining its former size, locality, pelvic relations, and original structure—Lactation, and the safe subsidence of this temporary function, from time to time.

By the operation and resources of the innate Restorative Power, the balance of health is regained—This state of ease and comfort, the one ordained by Nature,—disease the exception—Frequency of self-restoration in the course of each individual existence—not confined to bodily restoration—Restorative Power of the mind, and recovery from its shocks, wounds, and tribulations, during this world's pilgrimage.

THE PRINCIPLES OF THERAPEUTICS,

MEDICAL AND OPERATIVE. (p. 827.)

The Guiding Principle of Therapeutics is established by considering all the preceding chapters on Prognosis, or foreknowledge of the natural course and tendency of Injuries and Diseases, individually, to, or towards, a favourable or an unfavourable issue.

Pathology alone determines the *earliest* occasion for, and the *least* amount, no less than the kind of interference and assistance—Medical, Operative, or both—necessary to aid and complete the operation and resources of the innate Restorative Power.

The eminently Conservative character of this Principle is obvious, and converts its Medical aspect into the Conservative Practice of Medicine, and its Operative into Conservative Operative Surgery.

CHAPTER XVI. (p. 827—840.)

PATHOLOGY THE GUIDE IN SURGICAL OPERATIONS AND MANIPULATIONS.

The twofold condition of 'diseased' or 'injured' and 'living' textures and parts is *alone* our Guide as to the *kind* and *least* amount of instrumental and manipulative proceedings in Surgical Operations.

This Principle is also our Guide in Surgical Manipulations, apart from Operations, and in the application of Surgical Apparatus.

The Conservative value of this general Principle illustrated by the chief Operations of Surgery.

CHAPTER XVII. (p. 841—860.)

THE PRINCIPLES OF SURGERY

(AND OF MEDICINE CONCURRENTLY),

ARRANGED SYNTHETICALLY.

The Practical value of Pathological Principles of Therapeutics should be tested and estimated by Statistical Results—of the Restorative Power, of Medicinal agents administered, and of Surgical Operations and Manipulations conducted in conformity therewith.

The Statistical Method of Investigation.

Results of Statistics and objects of Inquiry.

General Conclusion.

PRINCIPLES OF SURGERY.

INTRODUCTION.

Historical Review of Medicine and Surgery.—Origin of Pathological Surgery.—The distinctive foundation and object of this Work.—Principles are attained by the analysis of General Pathology, and are co-extensive with all Diagnosis.—Etiology, respecting internal causes, and their operation.—Prognosis and Therapeutics: such Principles are *earliest* and *most exact;* their Preventive and Conservative character; they extend to Medicine, and tend to elevate Medicine and Surgery unitedly to the rank of a *Science*, while by their character they are eminently Practical.

A CALM retrospective view of the past is the surest guide to the future, in all matters of experience; teaching what to avoid, and suggesting also what to pursue. This general truth is forcibly illustrated by the history of Surgery. The reflective mind may thus overtake the acquirements of very many years, and surpassing those of any individual experience, however prolonged, realize the character of the age, and anticipate the future.

The earliest system of Surgery, in conjunction with Medicine, was built on the observation of functional symptoms only, or chiefly,—accurately, indeed, observed and recorded. The art of Medicine being suggested by the bodily infirmities and sufferings of mankind, it was natural, in the first instance, to notice only the most obvious phenomena and conditions of pressing importance; such as pain, and other functional disturbances, of which the sufferer complained, and for which he sought relief. The various forms of injury to which the body was exposed, more especially in a primitive state of society, would, for this reason, claim immediate attention; and hence the origin and primary

character of the "healing art." Thence the system of that time-honoured school which Hippocrates founded some 2400 years ago, which was developed by Galen, and revived by Sydenham in the seventeenth century of our era. They taught, and their disciples believed, or blindly followed, the doctrine of *symptomatology,** *i.e.*, the nature of diseases and injuries as revealed at the bed-side by observation of functional disturbances alone, or chiefly.

This 'clinical' knowledge only, of disease, being coupled with a simultaneous observation of the remedial effects of medicinal agents and surgical operations, gave rise to a species of 'rational empiricism,' which prevailed during many centuries, and still holds its sway, although perhaps unacknowledged or even disavowed.

But this *purely* clinical knowledge never reached the essential cause, or morbid condition—the *fons et origo mala*, from whence all the evil symptoms flowed; and its credulous followers, therefore, ever experienced the insufficiency of a 'surface' system, which gave no firm foundation for the correct observation of remedial measures, and no surety to Therapeutics. Accordingly, each new science, in any way allied to Medicine, that turned up in the current of time, was pressed into the service, to explain morbid phenomena, and supply a more exact theory of disease.

Physiology was thus laid under contribution, and all its phases were successively extended, and applied to the interpretation of Pathology,—Functional disturbances, and thence to the practice of Medicine and Surgery inclusive. The *chemical* aspect of physiology was first invoked by Paracelsus (1658), followed by men of more reputable character: Sylvius de le Boe, of Flanders (1680); our countryman, Thomas Willis (1684); and many others of the *purely* chemical school,—even, shall I add, up to the present day. The *mechanical* element was applied, aided, moreover, by mathe-

---

* Genuine Works. Hippocrates. Trans. for Syd. Soc. by F. Adams, LL.D. 1849. Vol. i. Epidemics. "Fourteen Cases of Disease."

matics; but this system, or instrument, in the hands of Borelli, of Pisa,* Bellini,† a pupil and disciple, the learned Boerhaave, of Leyden, and the whole school of iatro-mathematicians, signally failed. Even the grand discovery of Harvey was distorted to the most absurd results, when the dynamics of the blood's circulation were submitted to mathematical investigation. Starting with the complex data, which physiological science—unlike those of physics, and, I may add, chemistry—ever presents, the force of the heart's action was variously calculated, being raised by some to the terrific power of a Nasmyth's hammer, 180,000 pounds and upwards; by others, reduced to the modest standard of eight ounces. A more subtle theory had arisen with Van Helmont (1648)—foreshadowed by Athenæus in the first century of the Christian era—who, as founder of the *Vitalists*, sought to explain the phenomena of health and disease respectively, by the agency of a something he named *Archæus*, which subsequently became the *anima* of Stahl,‡ and the *vis medicatrix naturæ* of William Cullen.§ But, although ranked with the Vitalists in the history of rational medicine, Cullen by no means looked with any confidence to that curative power of nature, with which his name, I am inclined to believe, is still associated by many. "I might," says he, "go further, and show how much the attention to the Autocrateia allowed of, in one shape or other, by every sect, has corrupted the practice among all physicians, from Hippocrates to Stahl. It must, however, be sufficiently obvious, and I shall conclude the subject with observing, that although the *vis medicatrix naturæ* must unavoidably be received as a fact, yet whenever it is admitted, it throws an obscurity upon our system; and it is only when the impotence of our art is very manifest and considerable, that we ought to admit of it in practice."‖

---

* De Motu Animalium. Add. Meditationes Mathematicæ de Motu Musculorum, Joh. Bernouilli. 1710.

† Opuscula Aliquot, ad A. Pitcarnium. 1696.

‡ Theoria Medica vera. 1708.

§ See Works, ed. by John Thomson. 1827.

‖ Ibid. vol. i. p. 406.

The creation of human anatomy by Vesalius,* in the sixteenth century, advanced by Eustachius† and Fallopius,‡ had long since laid the foundation of a more durable physiology, a physiology based upon the knowledge of structure.

Structure may indeed be known, and accurately, without its function or use being at all understood; witness the spleen, and the so-called ductless glands; but the function of any organ or tissue implies a prior *identification* of such part; and thus it is that the knowledge of physiology implies a previous accurate knowledge of anatomy. This obvious relationship is fully recognised and acknowledged now-a-days; and I allude to it only to mark the dawn of *true* physiology.

The more solid ground supplied by Anatomy was gladly sought by some inquirers after truth, who, having long wandered in the mists of the Archæus, and other Will-o'-the-wisp phases of an abstract physiology, had well nigh sunk in the quicksands of speculation and conjecture. Albert Haller was the father of this new physiology; and the "Elementa Physiologiæ Corporis Humani" (1765) is an everlasting monument of his industry, judgment, and originality. The extension of Anatomy to the structure of animals, and even to that of plants, suggested to John Hunter the proportions of a colossal Physiology, and corresponding Physiological Anatomy; and the Museum of the Royal College of Surgeons of England, interpreted by the penetrating analysis of Owen, Paget, and our lamented Quekett, is the gift of genius to a grateful profession and to an ungrateful country. "His body is buried in peace, but his name liveth for evermore!"

The Philosophy, or first principles, of anatomy and physiology yet remained to be discovered. Xavier Bichat arose to thus illumine the pathway of rational medicine. The history of inductive science records no generalizations more grand or fertile,

---

* De Humani Corporis. Basil. 1542.

† Opuscula Anatomica. Venet. 1563.

‡ Om. Op. "Institutiones Anatomicæ," p. 482. Francof. 1584.

indirectly, to medicine, than are to be found in the first eight sections of the "General Anatomy, applied to Physiology and to the Practice of Medicine."

Bichat was the great original analyst of physiology, who sought, and successfully, to reduce all the phenomena of life to a few primary vital properties of the organism; who showed how all the manifestations of life flowed from these few elementary properties, how they entered by various associations into compound functions—*i. e.*, circulation, respiration, &c.; how these are reciprocally related to each other, and the organic or vegetative to those of animal life—*i. e.*, the phenomena of the nervous system, &c.

Bichat, again, was the great original analyst who sought, and successfully, to reduce the structure of all organs and parts of the body to a few comparatively simple and extensively distributed component textures; who showed how, by their various combinations, the several organs and parts of the entire organism are constructed—an analogous process of analysis and synthesis, by which (compound) functions are viewed philosophically, as consisting of simple functions, and these again as co-operating in one organism. Bichat thus created *general* Physiology, and *general*, or textural and systemic, Anatomy. Moreover, like Hunter in his organic inquiries, Bichat extended these generalizations to the lower animals, and even to plants.

The consummation of the labours alike of Haller, Hunter, and Bichat, with many others of the anatomico-physiological school, has been to 'humanize' this knowledge, by showing its relation to the 'rational' practice of medicine. Thus has been presented a standard,—the model man, structurally and functionally in health, subject to individual peculiarities, wherewith to contrast the aberrations and abnormities of disease. As compared with this standard, all purely Pathological phenomena and conditions are found to be departures,—by increase, diminution, or perversion respectively, of organic or textural structure, with compound or simple functional disturbances.

Deviations from the standard of health, whether structural

or functional, can themselves only be learnt by *independent* observation; but the disposition, or indeed the avowed creed, of many masters of anatomy and physiology, has been to teach pathology too inferentially; as if an accurate knowledge of the standard of health supplied an *à priori* knowledge of disease: just as in the more exact science of astronomy, given the law of a planet's orbit, its aberrations can be found. Hunter was thus too inferential—read his "Surgical Lectures." Bichat was too inferential. "The history," says he, "of phenomena in which vital powers have their natural type, conducts us by a natural *consequence* to those phenomena in which the same powers are changed."* Marshall Hall was too inferential. Thus, for example, "Ages before 'the principle of life' is detected, we may trace its phenomena, both by observation and experiment, and *deduce* from them important inferences both in regard to the nature, the prevention, and the cure of diseases." †

This inferential extension of physiology is the same error as that of the older physiologists (before anatomy had been cultivated), for the old physiology itself was but an extension and application of physics and chemistry, or else the mere dream of transcendental metaphysics.

The *indirect* relationship only of physiology to rational medicine, is a concession so important to the development of the latter, and the bearings of physiology thereon are so lucidly set forth in a lecture ‡ by Professor Walshe, introductory to the principles and practice of medicine, that I shall transcribe a paragraph and an illustration.

"The prominent characteristic of the scheme for the foundation of Pathology I here set forth and espouse, is its independence. It recognises facts of its own kind—not only kindred, but identical in

---

* General Anatomy, p. xxi.

† Gulstonian Lectures on the Mutual Relations between Anat., Physiol., Pathol., and Therapeutics. 1848. p. 42.

‡ On the Logical Applications of Physiology to Pathology. Oct. 1, 1849.

quality—as its sole basis. It makes no provision for the qualification of its laws by, much less for the formation of these laws out of, facts or generalizations belonging to other sciences, be the consanguinity of these sciences ever so close, and *ex natura rerum*, direct. It holds pathology to be a code of doctrines founded upon distinct, proper, and special elements—elements whose ultimate adaptation to that code takes place utterly irrespective of *à priori* notions pressed forward by cognate branches of knowledge. It gives pathology a place as purely self-dependent and distinct, in regard of the means of its *actual establishment as a science* (but of this only), as is held by chemistry, for example, in the sciences of matter. Or, more clearly to particularize, this scheme refuses by implication to recognise as the *true formative material* of the science of pathology (that is, the sum of classified laws of diseased actions) inferences deducible prior to experience, from current notions (whether possible, probable, likely, very likely, or certain,) held concerning the natural texture and healthy actions of the frame. This scheme denies that physiology (vital, chemical, or physical,) is the basis of pathology, in the sense that acquaintance with the one secures, by involution, acquaintance with the other. This scheme denies that physiology is the basis of pathology in the sense that, given the recognised healthy life of an organ, the consequences of the derangements of that life can by any forms of reasoning, inductive, deductive, analogical, or other, be positively predicated prior to actual experience of their character and habitudes. It affirms, on the contrary, that from observation, interpretation, numerical comparison, and classification of those derangements themselves (collated, of course, with healthy conditions), are their natures and laws alone to be established.

"Take the instance of an aged person, the neck of whose femur suddenly snaps: physiology might certainly justify us in the positive affirmation, that inability to use the limb for progression would follow this accident; it might, by diligent consideration of the precise lines of action of the various connected muscles,

(admitting this knowledge to be perfect in its way), make a correct assertion, prior to experience, as to the unnatural direction the limb would assume. But all this is merely mechanical; it is the sort of inferential power which would indeed prove physiology to be the formative basis of pathology, were man a mere machine. But he is not simply a machine; his femur cannot be broken without vital action being disordered; and the sum total of physiological knowledge could never have established, prior to actual experience, the vital consequences, local and general, of that simple injury. No; scarcely, I affirm, could it have supplied even a solitary link of the great chain of impressions which, originating in the disruption of a few minute nerves, vessels, and bone lamellæ, may eventually make themselves felt in every fibre and every function of the frame." In this instance, the independence of pathology is exemplified by a simple mechanical function; but the same author is rich in illustrations of this position, and among others he alludes, very appropriately, to the 'synocha' or pure *idiopathic* inflammatory fever of Cullen, as being a quasi disease, which that physiological pathologist acknowledges he had never seen; neither had Dr. James Gregory, during thirty years' experience, nor Dr. T. Bateman; and yet this creature of physiological inference still exists in systematic works, as an observed and observable reality!

But, among 'clinical' observers, there has ever been a growing conviction that Pathology is a science of *independent* observation; and *à fortiori*, therefore, that the 'causes' also, of functional disturbances, cannot be deduced by any *à priori* inferential mode of discovery;—in fact, that Pathology must have its analogue, just as true Physiology implies Anatomy. Gradually, therefore, there have become more and more clearly visible the outlines of another science, which lay between the man in health and the symptomatology of the Hippocratic school—the point from whence we started in tracing the historical development of rational medicine.

Pathological Anatomy—the first lines of which had long since been drawn, indelibly, by John Baptiste Morgagni, in his

ever-memorable work, "On the Seats and Causes of Diseases"*—has thus opened before clinical medicine and surgery.

---

* In explanation of the circumstances which led him to write this memorable work, Morgagni makes the following statement, which is worth reading, to understand the historical relation of that work to the "Sepulchretum" by Bonetus, as well as to appreciate the general purpose of Morgagni's labours, his matter-of-fact mind, his modest, candid, and confiding disposition:—"The anatomical writings of Valsalva being already published, and my epistles upon them, it accidentally happened that, being retired from Padua, as in those early years I was wont frequently to do in the summer-time, I fell into company with a young gentleman of strict morals and an excellent disposition, who was much given to the study of the sciences, and particularly to that of medicine. This young gentleman having read those writings and those letters likewise, every now and then engaged me in discourse, than which nothing could be more agreeable to me; I mean, a discourse in respect to my preceptors, and in particular Valsalva and Albertini, whose methods in the art of healing, even the most trifling, he was desirous to know; and he even sometimes inquired after my own observations and thoughts as well as theirs. And having among other things, as frequently happens in conversations, opened my thoughts in regard to the 'Sepulchretum,' he never ceased to entreat me, by every kind of solicitation, that I would apply to this subject in particular; and as I had promised, in my little Memoir upon the Life of Valsalva, to endeavour that a great number of his observations, which were made with the same view, should be brought to public light, he begged that I would join mine together with them, and would show in both his and mine, by example as it were, what I should think wanting to complete a new edition of the 'Sepulchretum,' which he, perhaps, if he could engage his friends to assist him, would, at some time or other, undertake. He also desired that I would write in as familiar a manner as I would wish, and by this means throw in at any time what I had said in conversation or medical conferences, or anything of that kind, which, though ever so minute, would always be very grateful to him.

"You ask me what was the effect of his entreaties. I suffered myself to be prevailed on. For, you see, what he required of me was partly what I had promised in that Memoir, and partly what I hoped would be of use, if it should turn out agreeably to my design; as, by being afterwards revised and published, it might some time or other excite persons far more capable than myself to undertake the same kind of labour.

"With this view, then, I began, upon returning to Padua, to make a trial of that nature, by sending some letters to my friend; and that he was pleased with them appears from two circumstances—the first, that he was continually soliciting me to send him more and more after that, till he drew me on so far as to the seventeenth; the second, that when I begged them of him, in order to revise their contents, he did not return them till he had made me solemnly promise that I would not abridge any part thereof."—Trans. by B. Alexander, 3 vols. 1769. Preface, pp. xx, xxi.

The revival of the *purely* clinical system by Sydenham,* in the previous century—the seventeenth—continued to mislead many from this fertile land of promise. Not a few, indeed, who had become wearied with the uncertain results of therapeutic treatment, conducted on principles professedly 'rational,' but not really founded on a due pathological knowledge as to the nature, origin, course, and tendency of disease, deserted from the ranks of the clinical corps, and proclaimed themselves 'pure empirics.' In the course of time many more deserted—first in France, then in Germany, and last of all in this country. These allied themselves, not again, as in times past, with pure anatomists and physiologists, but with pathological anatomists, who ever since the days of Morgagni have volunteered in the cause of medicine.

Apart, indeed, from its relation to medicine and surgery, the abstract pursuit of Pathological Anatomy has few, if any, intrinsic qualities of value or interest. Compared with other branches of natural science, this has not the beauty of botany; its truths are not so marvellous as those of chemistry; it lacks the grandeur and the poetry of geology; and does not come home to us apparently with the every-day utility of mechanical science. By the side of anatomy and physiology, we can scarcely concede thereto that perfection of construction, and adaptation of means to ends, which invest with wonder the study of these sister sciences. On the contrary, pathological anatomy might aptly be termed the science of disorganization, for it records the destructive changes, and their results, which organized beings undergo; it is familiar with all that is most distressing during life,—from the dawn of existence, through childhood, adolescence, and decrepitude; and with all that is most revolting after death. It remains, therefore, to be discovered how far this science has the redeeming virtue of guiding to the removal and prevention of

---

* Works trans. for Syd. Soc. by R. G. Latham, 1848. See vol. i. "Med. Obs. concerning the History and the Cure of Acute Diseases." Also, the Purely Clin. Method Exemplified, Dysentery, sec. iv. chap. iii. p. 169, parag. 8; Pleurisy, sec. vi. chap. iii. p. 247, parag. 5.

human suffering more especially, and leading to the restoration of health.

Every form of injury or disease presents certain pathological objects for investigation, by the knowledge of which its therapeutic treatment, medical and operative, *should* be conducted. I allude to the kind of disease or injury, with its situation and extent, as made known by *diagnosis;* its cause or causes, and the operation of the disease or injury itself, as an 'internal cause' of other morbid conditions—its relationship, in these respects, being indicated by *etiological* investigation; the foreknowledge of its probable course and tendency, to this or that issue, by *prognosis:* and it would appear that a large assortment of well-ascertained facts having accumulated, the period has now arrived when the attempt may be made to discover guiding Principles* in each of these preparatory 'departments' of Surgery, themselves suggestive of guiding principles of *therapeutic* treatment, and *together* forming rational Principles of Surgery, which might ever serve to direct the practitioner aright in each particular case, and regulate the course of further investigation.

With the view of ascertaining whether I had been anticipated in this inquiry, I perused the various systematic works, entitled "Principles and Practice of Surgery," "Systems of Surgery," and works of similar import, which have issued from Heister as the earliest author, Benjamin Bell, John Hunter, John Bell, and other accredited authorities, down to those of the present day.

I must here confess that my researches were fruitless, for what I found in the books referred to are not Principles of 'Surgery,' but General Surgical Pathology, with Therapeutics combined. That such knowledge has ever been hitherto denominated "Principles of Surgery," will at once be seen on examining the Table of Contents prefixed to each of the various works in question. But, not to make any particular selection, I shall refer

---

* Principle—fundamental truth; first position, from which others are deduced; source.—JOHNSON.

to the most recent one of this kind—the "System of Surgery," by Professor Gross, of Philadelphia; and to this work I may point without hesitation, since, in the "Preliminary Observations" thereto, we are not left in any doubt as to what is really meant by 'Principles of Surgery,' as usually understood. "If," says Professor Gross, "there is any one part of the present treatise upon the study of which I would insist more than upon that of any other, it is that which treats of the great principles of surgery, as comprised under the head of 'inflammation, wounds, morbid deposits, new growths, and morbid poisons.'" Similar principles are inculcated in all other systematic works on surgery, and in those on medicine also. For example, in the "Principles of Medicine," by Dr. C. J. B. Williams, they are specifically defined to be "those leading and general facts and doctrines regarding disease and its treatment, which are applicable, not to individual cases only, but to groups or classes of diseases." Yet this is General Pathology—interspersed, it may be, with an elementary view of remedial measures. Such were the Principles I found more or less fully and correctly expounded—but not those of 'Surgery,' as developed in its several departments—*Diagnosis, Etiology, Prognosis,* and *Therapeutics;*—nor, again, could I find the Principles of *Operative* Surgery.

The kind of Principles which I had in view, obviously contrast with the doctrines of General Pathology; and a word of further explanation will suffice to indicate their character. Take any one department of Surgery,—for example, *Diagnosis.* The object I proposed to myself was, *not* to discover the general features of resemblance and difference by which the various elementary forms of injury and disease are allied and distinguished into classes, known as wounds, fractures, morbid deposits, new growths, &c., with their respective varieties; but, given this preliminary knowledge, to *analyze* it, in order to discover the *grounds* of diagnosis, and by virtue of *General* Pathology,* having

* When not otherwise specified, Pathology signifies Pathological Anatomy, as well as Functional Disturbances.

been subjected to analysis, the elementary truths thus evolved would be those which should guide *all* diagnosis. Such are the Principles I had in view; and a similar character distinguishes those which should guide *all* etiological investigation, and *all* prognosis; the whole leading to the cardinal principle which should guide *all* therapeutics, medical and operative.

The aggregate of these Principles, therefore, would issue from a systematic analysis of General Pathology.

Failing to acquire these guiding truths, the short-comings of Surgery—clinical, medical, and operative—became the subject of my daily observation and experience.

At the bed-side, when about to undertake a case, and amply provided with general pathology, I was unable to decide such preliminary essentials as the following:—in respect of diagnosis, how far I might trust the Functional symptoms presented—how far Pathological Anatomy would avail me;—whether "physical signs" were more trustworthy than "structural characters," as those of textures and products, when discoverable under the microscope; for example, by thus examining the substance of tumours procured by puncture: and the value of "chemical conditions" as signs of the disease. In respect of etiology, its guiding principles with regard to "internal causes" and their operation, were ill-determined; while respecting the grounds of prognosis, or the 'essential conditions and laws' which regulate the course and tendency of the injury or disease, little had been determined. Nay, general pathology itself, as preparatory to this department of investigation, had been insufficiently cultivated, for the *natural* course and tendency of the injury or disease (apart from surgical or medical interference) was in most instances unknown.

To crown all my difficulties, I was therefore unable to predicate how far *Nature* might be trusted to conduct the case to a favourable issue; and when interference became necessary, I could not determine the kind of assistance indicated, and still less the least amount or measure of such assistance. I there-

fore was unable to select the simplest and least meddlesome, as well as most efficacious kind of therapeutic treatment. Lastly, during surgical operations, anatomy did not prove an infallible guide. The hernia of the dissecting-room, and the operation during life, presented widely different appearances, and I gradually discovered that in this and other operations, diseased and living conditions, combined, altogether changed the scene with which the mere anatomist is familiar.

I again referred to the books of Principles, and there I found that anatomy alone was the acknowledged guide during surgical operations, and that the doctrines of General Pathology were the accredited "Principles of Surgery." These so-called Principles represent, as already stated, the general facts and doctrines which are true of *all* Wounds, *all* Fractures, *all* Dislocations, *all* Aneurisms, *all* morbid Deposits, new Growths, and so forth; but they do not, and cannot possibly, represent the guiding truths of *all* Diagnosis, *all* Etiology, *all* Prognosis, and *all* Therapeutics. The *Science* of Surgery could not have been discovered.

Taking, therefore, the *subject*-matter of systematic works—strengthened by that of many valuable monographs, and extended, as far as possible, by the results of my own observations—as the *starting*-point of inquiry, I sought by an 'original and systematic analysis' of General Pathology, as already explained, to evolve the elementary truths which guide in each of the four departments of Surgery. These—the Principles of Surgery, properly so named—are one continued exhibition of the guidance, chiefly of Pathological anatomy, at the bed-side and in operations. This science is the ultimate source of all guidance in Surgery—clinical, medical, and operative; and thus applied, I would name it — Clinical Pathological Anatomy. It tends to impart the *earliest* and *most exact* character to each department of Surgery, which is thereby proportionately raised, from an *empirical* Art, to the rank and dignity of a Science.

In the year 1857, I initiated a critical investigation in this direction, by a new Inquiry entitled, "What has Pathological Ana-

tomy *done* for Medicine and Surgery?" * and the present work is but an extension, although a considerable one, of the Principles I then endeavoured to establish on the basis of Pathological Anatomy. From this source, the practice of Surgery, and of Medicine concurrently, was also shown to derive a twofold character. Both become Preventive and Conservative. To the origin and signification of this latter attribute I would now more particularly invite attention.

The surgical tendency of the age is to Conservative *operations*, by virtue of which, parts of the body are preserved that would otherwise formerly have been removed by the surgeon. Limbs that would have been amputated are now preserved, by the excision of a diseased joint. This is Preservative Surgery; and so far, so good. But, to supersede any operative assistance; is Conservative Surgery, equivalent only to the "Medical Treatment of Surgical Disease,"—a definition I have somewhere read?

I would take my stand on higher ground than that of either view, and elevate Conservative Surgery on a broader and deeper foundation. The full import and extended significance of this (conservative) attribute, in general Therapeutics, have hitherto been very incompletely recognised.

As the measure of our judicious reliance on the curative provisions and resources of Nature in the treatment of disease, and in conducting the repair of injury; and, therefore, thus guiding and regulating therapeutics, respecting the kind and *least* amount of assistance, medical and operative; determining also the *earliest occasion* for such assistance; and, moreover, determining whether *any* interference whatever be necessary,—Conservatism, thus expanded, most adequately represents that Therapeutic Principle to which, by long-continued observation and reflection, I have been led.

But the practice of Surgery and of Medicine, concurrently, will become Conservative, in this sense, only proportionately to the *earliness* and *exactitude* of our prognosis or fore-knowledge

---

* Serial Essays, Lancet, 1857.

respecting the natural course of diseases and injuries individually, to or towards a favourable or unfavourable issue; a similar etiological knowledge of internal causes, and their operation; and previous diagnosis, of a similar standard, so as to discover the particular kind of disease or injury, its situation and extent also, in the first instance. Such knowledge of these preliminary departments of surgery (and medicine) is our only sure guide to the appropriate and due employment of remedial measures, medical and operative; and whereby to anticipate and circumvent any impending complications which would or might supervene, and which, denoting the declining efficacy of the Restorative Power of Nature, would over-balance our Conservative measures.

Therefore, this character of practical Surgery, and of general Therapeutics, takes its root and origin far in the depths of all those preliminary Principles of diagnosis, etiology respecting internal causes, and more immediately of prognosis, which alike issue from Pathology. This, the parent; that, the offspring.

The character of this work is, therefore, eminently Conservative. To this end, guided by Pathology, I shall endeavour to establish the Principles of that Diagnosis, whereby the nature, situation, and extent of injuries and diseases may be detected during life at the *earliest* period, and *most exactly* discriminated. The leading questions of diagnosis, as regards each particular case that may arise, will, I trust, be definitely answered;—the diagnostic value of functional symptoms apportioned; the superior value of evidence supplied, during life, by pathological anatomy, elucidated. The value of 'physical diagnosis' will therefore be determined; the comparative value of 'minute structural characters,' and the aid of the microscope in diagnosis, will be fully explained; and the potential value of 'chemical pathology' as fully attested.

The Principles of Etiology will follow. I shall hope to show the guidance of pathological anatomy in the *earliest* detection during life of the *exact* local origin (exact situation and extent) of diseases, and of those injuries which are themselves preceded by disease of the part injured; as, for example, fracture of the

femur, induced by cancerous softening. I shall also trace the etiological relation and application of Pathology in referring local diseases themselves to their constitutional origin; it may be in the blood or nervous system.

We may thus anticipate, and perhaps *prevent*, the consequences of various constitutional and local diseases, and those injuries also which have themselves originated in diseased conditions.

The contributions which I shall endeavour to gather from Pathology on behalf of Prognosis will be of analogous import, as guiding to the earliest and most exact anticipation of the particular course and tendency which diseases and injuries manifest to or towards a favourable or unfavourable issue.

These—the Principles of each preliminary 'department' of Surgery—will conduct to the leading Principle of Therapeutics, medical and operative—the earliest occasion for assistance, and the most exact knowledge of the kind, and least amount, thereof—or, a due reliance on the curative operation and resources of Nature, in the practice of Medicine and Surgery. Therapeutics will thus acquire a Conservative character, of which the accessory Principles in question are its rudimentary phases.

Such then is the distinctive foundation—the object—and the character—of this work. Pathological essentially, and analytic; for the attainment of Principles—respecting diagnosis, etiological knowledge of internal causes, their operation, and prognosis: Conservative, in respect of Therapeutics—by virtue of the earliness and exactitude of all these Principles.

General Pathology is so arranged as best to establish (by analysis), and illustrate, the series of Principles advanced. I have almost invariably selected those forms of injury and disease which may occur in any part of the body; obviously because morbid conditions, unrestricted in their locality, are best qualified to establish *general* Principles. But I have drawn chiefly from that division of General Pathology, which by usage is denominated Surgical. Yet the extension of the same Principles to Medicine will be readily suggested. If therefore it be urged, in objection

to the plan pursued, that the particular exemplifications adduced are not a sufficient basis on which to establish guiding Principles of general application, further reflection will, I believe, not fail to complete their generalization.

To what extent I have succeeded in this undertaking can alone be determined by the impartial judgment of others.

In working out my design, errors may or must have crept in; for the pursuit of facts has necessarily been extensive and laborious. If then I have incautiously admitted an error, here or there, I shall feel obliged by correction; and if, while intent on the accuracy of my data, I have omitted the acknowledgment due to this or that observer, such omission has been quite unintentional. I have, in most instances, *paged* my references; and this will also meet the convenience of all those who, in seeking original sources of information on Pathology and Surgery, may consult this work.

My own pathological and clinical observations, scattered throughout, were noted down at the time of observation.

Assuming my data to be correct, the Principles advanced will, I believe, remain unshaken. However, in the temperate language of Bacon, I trust that they who read will "read, not to contradict and refute, nor to believe and take for granted, nor to find talk and discourse, but to weigh and consider."

ELEMENTS OF PATHOLOGICAL ANATOMY.

Assuming that Pathological Anatomy is the foundation of Surgery, it is necessary to clearly understand at least the elements of this science, and particularly in its surgical aspect—Surgical Pathology.

"In the early days of Surgery, every book was regularly prefaced with a system of the anatomy of the human body; and if this *preludium* be omitted now, it is because Anatomy has become, in itself, an important study."*

Without overlooking the preliminary importance of this study, I would here advance *Pathological* Anatomy, as being more nearly related to Surgery; and therefore, to its elements, I would now invite special attention.

What then is the purport and province of Pathology? When we clearly understand the object and limits of this science, then, and then only, shall we be in a position to determine its true relation to Surgery. Now, the word Pathology undoubtedly signifies the knowledge of *disease*. But what is disease?

By comparison with Anatomy and Physiology, as the standard of health, disease may be defined to be a deviation from this normal standard—in respect of one or more of the solids and fluids which constitute the organs and textures of the body, and in respect of the function or functions of such part or parts. Disease is therefore not an entity—like a parasite, introduced, from without, into the body; but the organism, or at least the part affected, in various conditions of deviation or alteration from the

---

* Principles of Surgery. John Bell.

normal state. Disease is not anything *in* the body, but the body or a part in such state.

Guided by the terms of this definition, there are two aspects of disease; namely, structural and functional. Pathology represents both kinds of aberration from the healthy standard; but Pathology *proper* is more usually restricted to alterations of function; while alterations of structure constitute the province of Pathological Anatomy. These distinctions, therefore, are the analogues of Anatomy and Physiology.

Confining our attention exclusively to Pathological Anatomy; alterations of structure are accompanied with alterations of physical properties, and chemical composition, of the organ or texture affected; or these changes may be considered apart from any perceptible change of structure.

It matters not which aspect of Pathological Anatomy is considered first; but it is more reasonable and convenient to begin with the simplest deviations from the healthy standard.

Therefore, firstly: Alterations of Physical Properties—including mechanical relations to surrounding parts.

Changes of *situation* are frequently witnessed in the shifting of loose, and perchance weighty, internal organs. Elevation above the normal level,—as when the heart is displaced upwards by a distended stomach; or, when the gravid uterus rises from the pelvis into the abdomen: and by descent below the normal level, as exemplified by prolapsus (not inversion) of the uterus.

'Protrusions' of internal organs may occur—as of the eye from a tumour within the orbit—the tongue in cases of glossitis, of which an illustration is given by Liston:* or again, internal organs may protrude through wounds of the cavities in which they are lodged. Thus, the brain may protrude through a fracture of the skull,—the heart or lungs may project through a wound of the thorax—and one or other of the abdominal viscera may partially pass through a wound of the abdomen. 'Herniæ' of the viscera readily occur, by one or other of them finding its

* Practical Surgery.

way through various natural openings in the walls of its containing cavity—the organ being covered by those integuments which have successively impeded its progress and escape. The presence or absence of the natural integuments, as an investing covering, respectively distinguishes herniæ from protrusions. Herniæ of the intestine and omentum may occur through either the inguinal or femoral apertures, or at the umbilicus, and occasionally in other situations; or, by the escape of portions of other abdominal organs, —as of the bladder, uterus, ovaries; the stomach, spleen, liver, —through the diaphragm into the thorax.

But displacements are not confined to loosely connected and pendulous internal organs. The different portions of the osseous system—the bones—although more firmly held together by ligaments, are liable to displacements. Such are dislocations of their articular extremities. Some bones, however, are more prone than others to these casualties; and the circumstance of ligamentous union being naturally loose, as in the shoulder-joint, coupled with the laxity of articulation induced by disease, will chiefly favour the occurrence of dislocation. The conditions known as congenital, complete and incomplete, simple and compound, are subordinate varieties.

The fluids may escape from those vessels or receptacles which naturally contain them. Of such alterations of situation, I may mention hemorrhage, and extravasation of urine.

Alterations of *number* are illustrated occasionally by double organs—the congenital absence of one kidney for example, or the superfluous addition of one or more mammæ.

Changes of *position* are favoured by the same anatomical, or rather mechanical, circumstances which predispose to alterations of situation. Either chiefly happens to pendulous internal organs. The gravid uterus may fall forwards (anteversion)—backwards (retroversion)—or to one side (lateroversion) : so again, the heart —its apex more especially—is easily displaced.

By *configuration,* I mean form, resulting from the relationship of distinct parts. Deviations in this respect from the natural

condition may be either congenital malformations or acquired deformities.

'Internal sacculations' of hollow organs are seen in the pharynx, stomach, intestines, bladder, and uterus. In such conditions these organs bulge outwards. But 'inflexions' of these cavities occur more frequently, and are exemplified by inversions of the uterus, vagina, and intestines (invagination).

External alterations of contour are illustrated by flexions of the uterus, and by spinal curvature; while the general and uniform expansion of large cavities—as from hydrocephalus or hydrothorax; their equally general retraction occasionally, with local bulging or depression—say of the thorax—respectively from emphysema and tuberculosis, are further illustrations of altered configuration.

So also are *solutions of continuity*, whether congenital or acquired. Such are 'fractures' and 'wounds.' The former term is restricted to sudden divisions of bone, the latter to those of the soft parts. Many varieties of either lesion are of great practical interest. Thus, the simple fracture is limited to the incomplete or complete division of bone; and this lesion may be either transverse, oblique, longitudinal, comminuted, or depressed. The compound fracture implies, moreover, a wound of the soft parts, either by protrusion of the bone itself, or by an external wound extending down to the seat of fracture. A complicated fracture implies the extension of injury to adjacent parts—as to the cerebro-spinal axis, or to the contents of the thoracic or abdominal cavities. Thus the lungs, and even the heart, may be lacerated by fracture of the ribs or sternum, and the bladder is frequently ruptured by fracture of the pelvis. Again, large vessels and nerves of the extremities may share the injury of adjacent bones. Wounds (of soft parts) also admit of many important practical distinctions. Contrast the simple division of tissues by an incised wound, with the bruise of a punctured, and still more so of a contused (*e.g.* gun-shot) wound—and again, the local effects of such injuries with the more fatal, because constitutional, consequences of poisoned wounds. The clean division of tissues by simple incision is the

wound of many surgical operations, and although a necessary evil, yet its consequences are far less serious than those of other wounds.

The physical properties of *colour, consistence, elasticity, size,* and *weight,* peculiar to the several organs and tissues, may respectively be increased or diminished, as compared with normal conditions.

Such changes, excepting those of colour, are expressed by the appropriate terms—induration and softening—rarefaction and condensation—hypertrophy and atrophy: the two latter representing alterations of weight, or quantity of matter, by increase or decrease respectively; while rarefaction and condensation express merely the enlargement or diminution of size (volume)—for example, the expansion of an emphysematous lung, and the condensation of that organ in cases of hydrothorax.

Alterations of size may affect only the capacity of hollow organs: thus, the diminution or constriction of any canal—*e.g.* the œsophagus, rectum, vagina and urethra—is known as 'stricture': on the other hand, enlargement of cavities may occur. The varieties of true aneurism, and of varicose veins, are to be referred to this head. These lesions, together with certain of those already mentioned, which fall within the province of Surgical Pathology, will be more fully considered in future pages of this work.

Passing on from Physical alterations, the Chemical constituents of solids and fluids may undergo similar simple variations of excess and deficiency; or again, the natural Structure of organs and tissues being retained, it may exhibit modifications of quantity.

Alterations of structural quantity are adequately represented by hypertrophy and atrophy, in respect of the solids; by plethora and anæmia in respect to the quantity of blood,—the excess or deficiency of which may be in the whole vascular system, or limited locally to a particular part. Lastly, local plethora includes the various conditions of venous congestion, determination of arterial blood, and inflammation.

Now, all the foregoing alterations—physical, chemical, and

structural—agree in one particular; they are departures, it is true, of the solids and fluids from their healthy anatomical conditions; but merely so as deviations in respect of *degree*, and therefore imply that the normal materials of the body remain otherwise healthy and unchanged. I use the expression 'normal *materials*' to include the unorganized fluids (secretions), as well as those possessing structure—blood, lymph, chyle, and the solid tissues and organs. Thus far, Pathological Anatomy is the counterpart of Anatomy, and being due to the instability of organization, its elements are therefore alterations of only the normal anatomical conditions.

But the elements of Pathological Anatomy are not limited to these more simple forms of disease; they comprise alterations also, by the addition to the normal materials of Morbid Products of nutrition, and by Degenerative substitutions of texture; and, lastly—as the most extreme degree of adventitiousness—the introduction of 'foreign bodies,' including 'parasites,' into the body.

Various classifications have been proposed—each, perhaps, offering its own advantages.

Morbid Products severally possess physical properties, most obvious; chemical composition, less definite; and in most instances, structure, of more constant character. Therefore, like other natural objects, they admit of arrangement by virtue of their resemblances and differences in respect of any of these marks of identity. Thus, the classification of tumours proposed by Abernethy recognised chiefly the physical characters of outline, colour, consistence, &c. The grouping of morbid growths suggested by Müller —that of gelatinous, fatty, and protein composition—was founded on consideration of their chemical nature. I would select the characters, together with the vital endowments, of their *structure*, as the bond of alliance, and guided by this feature of relationship, new products—morbid so-called—are variously allied to healthy conditions.

Their primary division into Plastic and Aplastic points to the parallel distinction of the normal materials of the body, into those

possessing the definite arrangement of solids (and fluids) termed organization; and the unorganized fluids, or secretions.

The division—Plastic Products—presents a gradual transition from the more simple varieties of apparently alienated structure, to those products whose structure is apparently more dissimilar, and furthest removed from the normal conditions of healthy structure. I say *apparently* alienated and dissimilar; for the departure, *structurally* speaking, will prove after all to be more apparent than real and essential.

This brings us to the threshold of a new Pathological Law,—that of "Structural Retrogression," as I have termed it, which I sought to establish in aid of Diagnosis, in the course of an original Analytical Inquiry, already quoted.* This Pathological Law represents Plastic Products as merely *persistent rudimentary* conditions of the structural elements of their analogous *healthy* tissues, by *arrested* development of those elements.

I shall first *describe* these Products, and according to the classification of them commonly recognised by Pathologists.

Plastic Products have their local origin in the transudation from the capillary vessels of 'blastema' which may be regarded as modified *liquor-sanguinis*. This nutritive material undergoes various chemico-plastic changes, which terminate in equally various forms of structure, apparently unlike healthy conditions. Hence, while all such products are plastic or organized, the *kind* of their adult organization is supposed to represent departures, more or less remote, from healthy structures. They differ also more or less in their mode of *reproduction*—some being *directly* reproduced from blastema, others *reproducing themselves*, provided the parent organized product be duly supplied with blood.

Agreeably to this twofold distinction of structure and reproduction, the following classification of plastic products is commonly acknowledged, and the following distinctions recognised,

---

* "What has Pathological Anatomy done for Medicine and Surgery?" Serial Essays. Lancet, 1857.

as to their nature and apparent gradation of affinities to healthy tissues.

False tissues—so named from their imperfect structural resemblance to healthy tissues. Such tissues are supplemental, either by supplying the place of those which have been destroyed by injury or disease, or as super-additions to the normal tissues.

Fibro-cellular tissue (fig. 1),* approaching in structure to that

FIG. 1.

of the normal tissue, so named, is the first product of reparative blastema. This texture supplies the basis of union wherever parts have been recently divided, and it is therefore appropriately named "connective tissue" (Paget). At this stage the process of development may terminate, or the tissue may be afterwards fashioned off, so as to resemble the particular tissue, whether bone, tendon, skin, mucous membrane, &c., of which it is destined to form the bond of union.

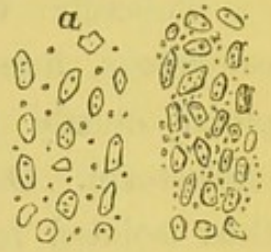
Deposits result from the supplemental overflow of nutritive material, which may be deposited in various structures. This material then undergoes more or less organization; hence tubercle, and the products of inflammation.

---

* Microscopic elements of fibro-cellular tissue; from a tumour of this kind showing cells in various stages of elongation and attenuation into filaments. Magnified about 450 times. (*Paget.*)

Tubercle—literally signifying a small tumour—is found in small masses, each varying from a pin's head to a hen's egg, of an opaque yellowish colour and friable consistence. These masses are formed of molecules and granules, which abound more as, in the course of its decay, the tubercle softens. Certain cells, tubercle-corpuscles, are also seen under the microscope, of a round, oval, or triangular shape, and varying from $\frac{1}{2000}$ to $\frac{1}{1200}$ of an inch in diameter. They contain each, from one to seven granules (Bennett); they are also not changed by water, but become very transparent by the action of acetic acid (fig. 2).*

FIG. 2.



Another kind of tubercle is described as the "miliary," or "grey semi-transparent granulations." They are little bodies, from the size of a millet-seed to that of a pea, of an angular shape, greyish, transparent, shining, and approaching to a cartilaginous consistence.

Some authorities maintain that these bodies are tubercles in the first stage of their development; other observers affirm that they are not any phase of those which I have just described. Carswell alleges that grey by no means necessarily precedes yellow tubercle, and, moreover, that it does not occur in many structures, as the lymphatic glands, where yellow tubercle is often found.

But true 'tubercle' undergoes certain alterations as its life advances, and which are accompanied with textural disintegration of those structures in which it is deposited. Thus, for example, cavities or *vomicæ* are formed in the lungs. I pass over these textural changes, as being details of special pathology.

Tubercle itself tends to change in two ways—diverse, although not opposed.

It may soften and resemble thick pus, or a mixed fluid,

---

* Tubercle-corpuscles, from firm tubercular exudation into the lung. *a.* after the action of acetic acid. (*Bennett.*)

partly transparent and colourless, and partly a caseous substance. These physical alterations are attended with structural disintegration of the tubercle-cells, which were in the first instance only an abortive effort of cell-formative power, and never became perfect. Molecular and granular matter, and broken cells, eventually constitute *softened* tubercle.

Tubercle may harden, so as to become *cretaceous* or *calcareous*, being certainly in this state structureless, and composed chiefly of phosphate of lime, with some crystals of cholesterin. Occasionally tubercle becomes enclosed in a cyst, and thus isolated from the surrounding texture in which it has been deposited, may then remain quiescent for many years. Encysted tubercle occurs occasionally in bone, but rarely in pulmonary texture. The *process* by which tubercle is eliminated from the body is not within the province of Pathological Anatomy to describe.

The chemical composition of tubercle varies with its condition, as grey semi-transparent, yellow, cretaceous or calcareous.

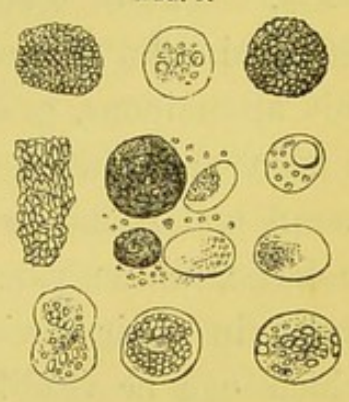
In its primary state, the standard qualitative composition of pulmonary tubercle is, according to the analysis of M. Felix Bondet, stated by Becquerel and Rodier, as follows: gelatine, albumen, casein, or a substance analogous to it, and fibrine, oleic and margaric acids, olein and margarin, lactic acid, lactate of soda, extractive matters, cerebric acid, and cholesterine, which alone constitutes about $\frac{1}{10}$ part of dried tubercular matter. Chloride of sodium, phosphate of lime, carbonate of lime in small quantity, sulphate and carbonate of soda, silica, oxide of iron, and lactic acid, are obtained by incineration. Calcareous tubercle consists of soluble salts, chloride of sodium, phosphate and sulphate of soda, and the residuum of phosphate of lime chiefly; with carbonate of lime, silica, and oxide of iron, in small quantity.

The products of inflammation are also deposits. These products are—serum, fatty and albuminoid granules, exudation or compound granular corpuscles, pus-corpuscles, and coagulated lymph, perhaps organized. They are usually found associated, in whatever situation the inflammatory liquor-sanguinis, from which

they are evolved, may have been deposited: but, one or other structural element will so far predominate in certain situations as to give its own structural character to the deposit. Thus, while in all cases the liquor-sanguinis effused separates into serum, and a solidifying organizing substance, this latter is represented in parenchymatous organs and cellular textures *chiefly* by granules and granular corpuscles; in respect of mucous membranes, *chiefly* by pus; and on serous membranes, *chiefly* by coagulating lymph. The products of inflammation have therefore been studied by the examination of samples taken from these several parts.

The following results are partly those obtained by the observations of Dr. J. H. Bennett, who has paid considerable attention to this subject.

Fig. 3.



The granules I have mentioned are found aggregating around and between, the capillary vessels, in, for example, pneumonia. Their diameter varies from $\frac{1}{12000}$ to $\frac{1}{6000}$ of an inch. Exudation-corpuscles (fig. 3)* are formed by these granules coalescing into little round masses, which then sometimes acquire an enclosing cell-membrane, and appear under the microscope like minute raspberries or mulberries, about $\frac{1}{1000}$ to $\frac{1}{750}$ of an inch in diameter. Within each of these corpuscles is seen a round transparent nucleus, the size of which is from $\frac{1}{5000}$ to $\frac{1}{3000}$ of an inch across. The granules are fatty. Water and acetic acid do not affect the cells or masses; but they dissolve immediately in either, and disintegrate under the influence of potash or ammonia.

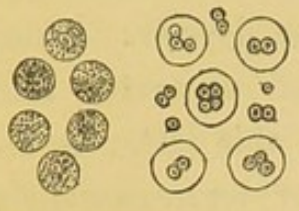
Pus is a further product of inflammation. Its physical characters are well known,—a greenish yellow fluid, of creamy consistence, having a faint odour, and an average specific gravity of 1030, but varying considerably, *i.e.* from 1020 to 1040.

---

* Granular cells and masses from cerebral softening. (*Bennett.*)

This fluid consists of a serum in which are suspended an innumerable multitude of corpuscles (fig. 4.),* which much resemble those of mucus, chyle, and the pale corpuscles of blood. That is to say, a pus-corpuscle is a spherical, somewhat granular-looking cell, from the $\frac{1}{200}$ to $\frac{1}{300}$ of a line in diameter; and in the interior of this cell is laid a nucleus, which adheres to the cell-wall, and is therefore *eccentric*. It varies in size from $\frac{1}{800}$ to $\frac{1}{400}$ of a line in diameter, and consists of two, three, or four granules aggregated together. This can be shown best by the addition of water (to the pus under microscopic examination), which distends the corpuscle; or by acetic acid, which dissolves the cell-wall, and breaks up the nucleus into its component portions. These secondary nuclei are round, oval, sometimes elliptical. An albuminous solution or molecular matter surrounds this albuminous, granular nucleus. The granular cell-wall itself is also albuminous, and being adhesive, the corpuscles skirt round and round in the field of the microscope; and yet being inelastic, unlike pale blood-corpuscles, they scarcely change their shape in their devious course. Owing to the family likeness, which can be readily recognised, between the corpuscles I have mentioned, Henle has proposed the generic name of *cytoid* (cell-like) corpuscles to include them all. This resemblance extends even to their chemical composition.

Fig. 4.



Pus has been analyzed by Güterbock, Valentin, Golding Bird, Wood, Von Bibra, and Wright, with unsatisfactory results.† I take the following particulars from the most recent work‡ of authority on this subject.

Pus-serum is a clear, colourless, or very faintly yellow fluid, having a weak alkaline reaction, and coagulating by heat into a

* Pus-corpuscles, as seen in healthy pus; the five transparent cells are as seen after the action of acetic acid. (*Bennett.*)

† See Pathological Chemistry. 1853. By Becquerel and Rodier. Translated by S. T. Speer, M.D. 1857. p. 529.

‡ Chemistry in its Relations to Physiology and Medicine. G. E. Day, M.D. 1860. p. 221.

dense white mass. Albumen is its chief constituent, in proportions from 1·2 to 3·7%. Fatty matter also, extracted by ether, and consisting of olein, margarin, oleic and margaric acids, and cholesterin. These fatty matters vary from 2 to 6% of the whole fluid pus, of which proportion, cholesterin alone often reaches to 1%. Mucin and pyin are occasionally found in pus-serum; also, casein, chondrin, glutin and leucine.

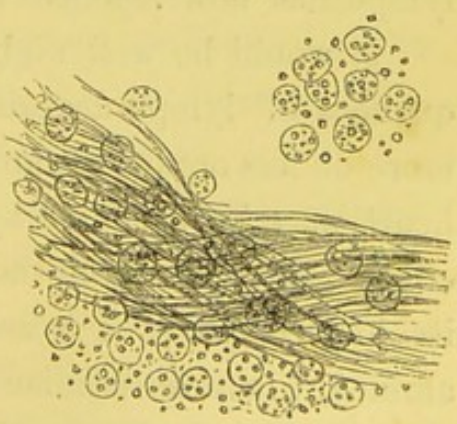
The solid constituents of this serum range from 14 to 16%, of which from 5 to 6% are mineral, and the soluble salts are to the insoluble as 8 to 1. Of the former, chloride of sodium is most abundant, being three times more so than in the serum of blood. The soluble phosphates in the ash range from 3 to 10%. The insoluble salts are phosphates of lime and magnesia, with a little sulphate of lime and peroxide of iron.

Certain incidental matters may be found in pus, as in all other exudations—namely, bile-pigment, the resinous acids of the bile, urea and sugar.

Pus is seldom found pure, but associated with various ingredients, the debris of surrounding textures, and mixed with secretions. Healthy or laudable pus may thus become serous, mucous, sanguineous, strumous, cancerous, &c. Or, pus may be specific, such as the syphilitic, the vaccine; pus of porrigo, of glanders, &c. These varieties either speak for themselves, or will be known best by personal observation.

Coagulated lymph (fig. 5)* is more or less tenacious, solid, and of an opaquish, yellowish colour. It consists of very fine filaments, from $\frac{1}{14000}$ to $\frac{1}{10000}$ of an inch across, and each is formed apparently by the linear union of molecules. The filaments are closely interwoven in various directions, and present a clot or coagulum, as that of buffed and sizy blood.

FIG. 5.



* Molecular fibres and plastic corpuscles, in "simple" exudation on a serous surface. Above, are corpuscles after the action of acetic acid. 250 *diam.* (*Bennett.*)

Certain corpuscles are interspersed throughout this coagulum; and these contain each from three to eight granules, the diameter of which equals that of the filaments above mentioned. The cells themselves range from $\frac{1}{1300}$ to $\frac{1}{1000}$ of an inch. They have been named *plastic* by Dr. Bennett, because occurring so frequently in plastic lymph; and also named *pyoid* by Lebert, from their general resemblance to pus-corpuscles. Neither water nor acetic acid, however, much affect these plastic cells.

Coagulable lymph appears to be deposited from the vessels in the form of minute villi. This disposition is seen on those free surfaces which are least subject to motion or pressure, and therefore we find villous lymph *between* convolutions of the intestines in peritonitis; *between* the lobes of the lungs in pleurisy, the villous appearance being less marked than in the former instance; and about the *base* of the heart in pericarditis. But wherever the serous surfaces are in contact, and play upon each other, there the ductile lymph is drawn out into threads or plastered into films, such as are found in free portions of the pericardium and pleura, and between the parietal aspect of the intestines and peritoneum.

In the course of time blood-vessels are developed in coagulated lymph, which protrude into its villous projections, and serve the purpose of absorbing the serum effused within the cavity. As the containing cavity collapses, and its opposed surfaces meet, they grow together, and form dense chronic adhesions. Inflammatory lymph has now reached its highest degree of organization.

It would be well nigh impossible to enumerate all the consequences of lymph-deposit in various structures, as it becomes more or less organized, and more or less pliant and yielding. The heart may be shackled by two or three tags of lymph, which, continually restraining the motions of this organ, at length induces its hypertrophy. I possess the drawing of a heart that I examined, where the cardiac reflexion of the pericardium had become thickened by the interstitial deposition of inflammatory lymph in it, to such a degree as to have caused atrophy of the enclosed muscular

substance, by continued compression, and eventually 'disintegration,' not fatty degeneration, of the fibrillæ.

Another and a well-known consequence of inflammatory lymph-deposit is the formation of tough and unyielding adhesions. Most students in pathology will have had frequent opportunities of peeling the lungs off the ribs, to which they were bound down by tight pleuritic adhesion. In making *post-mortem* examinations of cows that have had the "lung-disease," I have repeatedly torn away the lungs with a blunt hook; these organs being so solid as not to break up when thus removed. Peritonitis may agglutinate the intestines and abdominal viscera into one mass, of which there is a remarkable drawing by Carswell in the museum of University College, London.

Thus, the chief viscera become fettered by adhesions.

Within the substance of parenchymatous organs, an inferior kind of granular matter, with some contractile lymph, may be deposited, by which they become stuffed up and consolidated. Such are hepatized lungs, cirrhosis, and granular kidneys. Strictures may form around canals, as the œsophagus, rectum, and urethra. Eventually, displacements of moveable viscera are produced by slow contraction of lymph-deposit, resisting the struggle of function to recover the right adjustment of parts.

This fettered condition of organs—their consolidation, their obstruction, and their displacements—are some of the evil results of lymph-deposit, and its changes in respect of structure and physical properties. Analogous mischief may happen to the mechanism of the limbs: thus, joints become stiffened by firm fibrous anchylosis, the tendons refuse to play in their sheaths, and the fasciæ agglutinate, in chronic rheumatism.

In other and perhaps more numerous instances, lymph-deposit fulfils some good purpose by its organization. It is either reparative, to reinstate an old function; or constructive, to fulfil a new and useful one. This aspect of the subject will be fully illustrated in a subsequent chapter, on the Restorative Power of Nature.

But although in its highest degree of development, that of false membrane, the organization of lymph-deposit surpasses all other deposits, and approaches certain false tissues, yet all these products alike agree in the mode of their *reproduction* (no less than in that of their production); namely—*directly*, from blastema. Pseudo-tissues and deposits do not possess the power of reproducing themselves.

Growths also result from the supplemental overflow of a nutritive material, which gradually assumes various forms of organization, at least as highly developed as any of those already described; and yet, in point of kind, these products are said to be more unlike normal (healthy) conditions of structure. I shall enter on this question presently. (See Law of Structural Retrogression.)

But however nearly, in respect of minute structure, morbid growths may resemble the various healthy tissues (more especially those normal tissues with which they are associated), yet a 'growth' presents a well-defined *boundary*, by which outline of demarcation from adjoining tissues it can be readily distinguished. By virtue of their homology, or structural resemblance to healthy tissues, growths may perhaps be regarded as *hypertrophies*, but *discontinuous* from the surrounding tissue, and by this peculiarity distinguished from hypertrophies, or outgrowths, which are continuous—as seen, for example, occasionally in outgrowing portions of the thyroid and prostate glands.

Growths are, moreover, to be recognised by an additional peculiarity; not an anatomical feature, but a physiological characteristic. They possess the inherent power of reproducing their own structural elements, when adequately supplied with blood. Growths are *germal* plastic products. They increase and multiply; but, I should add, apparently fulfil no useful purpose in the animal economy. "It is not (says Mr. Paget) in the likeness or in the unlikeness to the natural tissues that we can express the true nature of tumours: it is not enough to consider their anatomy; their physiology also must be studied: as dead

masses, or as growths achieved, they may be called like, or unlike, the rest of a part; but as things growing, they are all unlike it. It is therefore not enough to think of them as hypertrophies or overgrowths: they must be considered as parts overgrowing, and as overgrowing with appearance of inherent power, irrespective of the growing or maintenance of the rest of the body, discordant from its normal type, and with no seeming purpose."

Such are the general structural characters, and such the general vital endowments of growths, or tumours. But the vital changes of certain growths contrast remarkably with the progress of others. Some appear to exercise a merely local and mechanical influence, and their pathological import is therefore limited to surrounding parts, which are variously pressed, obstructed, and possibly obliterated by absorption. The healthy mechanism only of the body becomes impaired by these growths. Other growths are not localised in their operation, but gradually pervade surrounding tissues, and affect neighbouring lymphatic vessels and glands; they are also prone to ulceration, and by the extension of this process of destruction, involve adjacent parts. They propagate, moreover, in different and distant regions of the body, and grow simultaneously. Such growths are therefore aptly denominated *infiltrating* (Walshe) or malignant—a less significant term; while others, being distinguished by the negation of this attribute, are non-infiltrating, or innocent — comparatively speaking.

Non-infiltrating growths include—

The fibro-cellular tumour. This is distinguished from polypus, mucous or cutaneous, chiefly by its relation to the adjoining texture. Both are overgrowths; but, while polypus is merely an *out*-growth of fibro-cellular tissue, the same structure, as a tumour, is distinctly isolated by a capsular investment.

The mass, thus detached, is roundish, and of tolerably regular outline, occasionally lobed, and may grow to great size and weight.

Its chief physical character is a remarkably elastic tension,

which is due to the structural resemblance of this tumour to dropsical cellular tissue, circumscribed. Section shows a yellow surface, marked with white lines, which have an undulatory direction across the tumour, and may divide its substance into distinct lobes.

Both the yellow substance, the white bands, and the capsule, alike consist of fibro-cellular tissue—more condensed in the latter portions; but the whole is remarkably succulent, being infiltrated with a serous fluid, which exudes plentifully, and continues to exude from the cut surface. The tissue, of which (together with the serous fluid) this tumour consists, represents an immature state of the normal fibro-cellular tissue (fig. 1). With many well-developed filaments are more abundant nuclei, and cells-forming fibres (fibro-cellular) in various stages of development. Portions of cartilage, sometimes partially ossified, are occasionally found in or over the tumour (Paget). Its texture may also degenerate.

Analogous as the texture of this tumour is to ordinary fibro-cellular tissue, it is nevertheless of rare occurrence, compared with other tumours—fatty or cartilaginous—whose component tissues respectively are reproduced far less frequently than this tissue.

Painful subcutaneous tumour, or tubercle—so named by Mr. W. Wood,* who first described it—is a peculiar variety of fibro-cellular tumour, to which and to fibrous tumours it is structurally allied; but peculiar, if only as distinguished by the pain, intense and paroxysmal, which commonly occurs, and which is not to be accounted for by the structure of the tumour, itself perhaps destitute of any nerve-filaments, nor by any special relation to adjacent nerves.

This painful tumour, situated, as its name implies, in the subcutaneous cellular tissue, is barely visible; for it is beneath the skin, and scarcely projects. It is also of small size, rarely exceeding half an inch in diameter; but it can be readily felt, as a

---

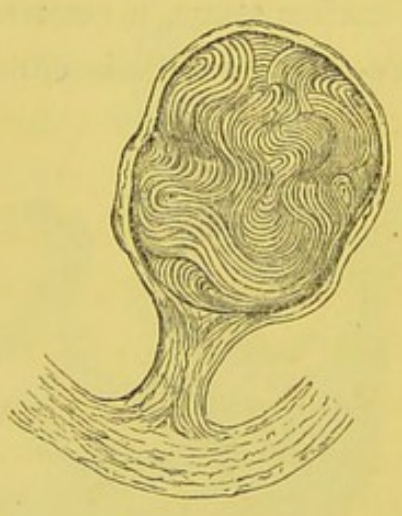
* Edinburgh Medical and Surgical Journal, viii. 1812.

roundish body, very firm and elastic. Isolated by a capsule, this hard body is free in the subcutaneous tissue, and is therefore so far moveable; but it may be intimately adherent to the skin, and move with it when examined with the finger. The superimposed skin, if adherent, has the general appearance of a cicatrix; it is slightly puckered, stretched, glistening and white, unless during a paroxysm of pain, when it may become congested and swollen, and the surrounding blood-vessels turgid.

The surface and section of this tumour are alike in colour, variable; commonly yellowish; occasionally only, greyish or pure white. Its substance consists of fibro-cellular or fibrous tissue, with an abundance of nuclei intermixed; and the whole represents apparently an immature state, or rather various immature states, of either tissue. Nerve-filaments have not been discovered. This tumour is usually solitary, as well as subcutaneous.

The fibrous tumour (fig. 6)* is recognised by its great firmness and elasticity, and by its lobulated, spheroidal shape, when uninfluenced by the pressure of surrounding parts. The tumour sometimes attains a large size. Its chemical basis is gelatin—but the other constituents are unknown. On section, it presents a greyish colour, variously intersected by white opaque lines. The fibrous tumour consists of the white and yellow (elastic) fibres of ordinary fibrous tissue, and, like it, is but scantily supplied with blood-vessels which pass within the substance of the tumour from its fibro-cellular investment; this investment being more apparent around those tumours which are imbedded in solid organs.

FIG. 6.



Earthy matter or cysts may be found within the substance of a

---

* Fibrous tumour diagram—section of an uterine fibrous tumour, like a polypus, but *discontinuous* with the substance of the uterus. (*Paget.*)

fibrous tumour; and hence the terms, fibro-calcareous and fibro-cystic, as denominating these varieties. Other and more important varieties are, the fibro-nucleated of Bennett, and the recurring fibroid of Paget. The former present under the microscope the white fibres of fibrous tissue, mixed with numerous oval nuclei, and represent a developmental stage of healthy fibrous tissue. Fibro-nucleated tumours do not affect the lymphatics in their neighbourhood, nor do they contaminate more distant parts; but, when extirpated, they have a tendency to return, *in situ*. Yet in external appearance and general characters a fibro-nucleated tumour singularly resembles an ordinary fibrous tumour, for which it might be readily mistaken. The recurring-fibroid variety possesses also the same general character and non-malignant tendency; but they recur, as their name implies, it may be again and again, when extirpated, and successively assume the malignant character, and also the soft, bloody, fungoid appearance of encephaloid cancer. Not so in structure, for whether examined when malignant or in an earlier stage, a recurring-fibroid tumour consists essentially of fibre-cells; *i.e.* cells elongated, and developing into white fibres,

FIG. 7.

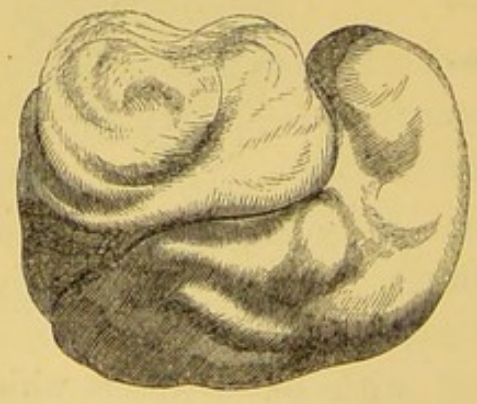
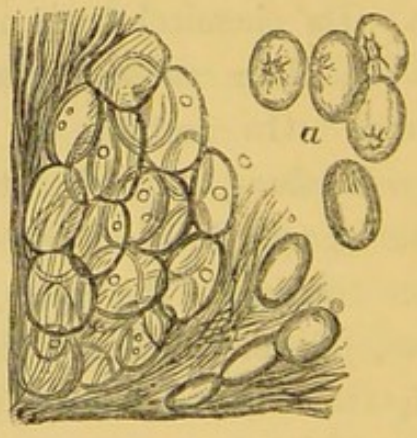


FIG. 8.



mixed with nuclei: in fact, this tumour represents another developmental condition of healthy fibrous tissue.

Fatty tumours (fig. 7)* are soft and lobulated, and often very large. They have the composition and general structure of ordi-

* Fatty tumour, removed from under the tongue; half the natural size. (*Liston.*)

nary fat—that is to say, fat-cells (fig. 8)* are collected together and imbedded in a fibrous mesh-work, more or less dense, and variously disposed; and this is continuous with a thin fibro-cellular capsule enclosing the tumour, and by which it is loosely connected to surrounding parts; so that a fatty tumour is loose and moveable, a circumstance which favours its easy removal by the knife. Blood-vessels, collected mostly at one point of the tumour, pass into its substance from the cellular capsule. When removed, this tumour never returns. Occasionally, cysts are developed within the substance of a fatty growth, or indurated knots of the fibro-cellular stroma are felt, which may eventually become bony; or the whole mass may be, or become, so fibrous as scarcely to be distinguished from an ordinary fibrous tumour. In due time I shall have occasion to recur to the fact of this transitional condition, and to show its relation to an exact diagnosis.

Cartilaginous tumours—the Enchondroma of Müller — are growths which present the appearance, the chemical composition, and structure of masses of fœtal cartilage. Each is enclosed in a tough fibrous capsule, which conducts a few blood-vessels. These tumours are seldom solitary, but aggregated together, and grow from or within the bones and joints; more especially are they seen attached to the phalanges of the fingers and toes (fig 9).†

Fig. 9.

The surface of a cartilaginous tumour is more or less irregular and lobulated, the fibrous capsule passing in between and separating the lobes. Its substance is pulpy or more consistent; it may be hard, but elastic, crisp when cut, and a section is bluish-white,

---

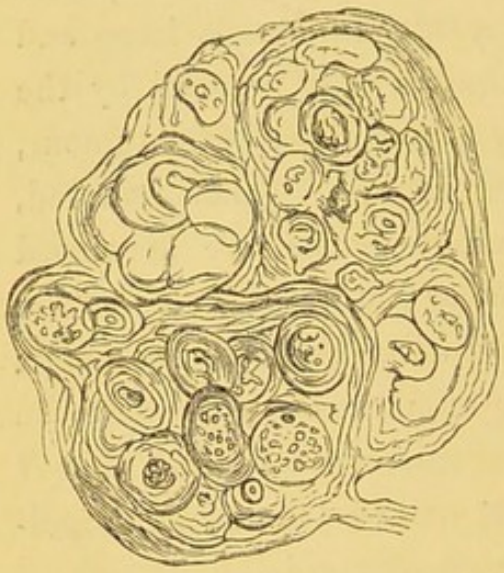
* Structure of a fibro-fatty tumour. *a*, isolated cells, showing stellate crystals of margaric acid. 250 *diam.* (*Bennett.*)

† Enchondroma of the hand. (*Druitt.*) The tumours are drawn too regularly round, and have not the nodulated character of the cartilaginous tumour. (F. J. G.)

like London milk, and translucent. These appearances vary with modifications of structure.

Two structural elements (fig. 10)* are found the same as in ordinary cartilage;—cells, and an inter-cellular substance, the latter being semi-transparent or molecular and dim, but more commonly fibrous. The cells also may be scattered or aggregated, multiform, and they resemble either mature or rudimentary cartilage-cells. Their envelope is therefore more or less defined, encloses one or more nuclei, within each of which is seen a nucleolus or two. Occasionally the nucleus may appear radiated, and not unlike the lacunæ and surrounding canaliculi seen in osseous tissue. We can therefore trace an intimate structural homology between cartilaginous tumours and healthy cartilage, more or less developed; and this resemblance extends also to the myeloid variety of enchondroma. Myeloid tumours are allied to the fibrous by virtue of one structural element—the fibre-cell; and also to ordinary cartilaginous tumours, by their more abundant *many-nucleated* corpuscles. These corpuscles and imperfect fibres appear to be identical with those of healthy rudimentary bone; the marrow-like "myeloid," so called by Paget, have therefore been named "fibro-plastic" tumours by Lebert, from their structural elements resembling those of granulations; and, indeed, the substance of such tumours resembles in appearance the granulations of healthy bone, in conjunction with which tissue and carti-

Fig. 10.



* Cells and inter-cellular substance, or stroma, of a cartilaginous tumour, from the phalanx of a finger. Many of the cells are only drawn in outline; some of them present double or triple contour lines; most of the nuclei are large and granular. The groups of cells are intersected by bands of tough fibrous tissue. Magnified about 400 times. (*Paget.*)

lage, cartilaginous tumours more commonly originate than in other parts of the body.

A myeloid tumour is liable, if not prone, to return when removed by the knife, and differs therefore in this respect, as well as also somewhat in its minute structure, from an ordinary cartilaginous growth. The general appearance and characters of the myeloid variety are also tolerably significant. Its surface is more or less lobulated. If enclosed within bone, its shape is uniform and spheroidal; if seated on the surface of a bone, its outline is irregular, as seen in epulis. The fleshy, inelastic firmness of a myeloid growth is remarkable, although its consistence varies; and on making a section of such a tumour, we recognise the greyish-white basis colour, but daubed with irregular blotches, which are of a bright red, livid, or brownish tint. The cut surface is also succulent, and exudes a yellowish fluid.

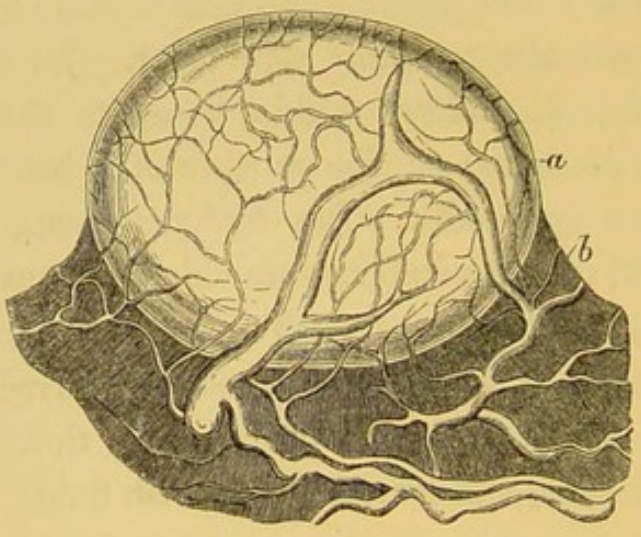
The consistence of an *ordinary* cartilaginous tumour may, however, undergo remarkable changes. A species of ossification may take place, beginning either on the surface or within the substance of the tumour. Again, the whole tumour may soften and feel like a fatty growth, or a mass of colloid cancer; or ossific nodules may soften and feel like a group of cysts. I shall refer hereafter to these important points of apparent resemblance between cartilaginous tumours and other growths.

Cysts and cystic tumours are very varied in their nature and characters. It would therefore be impossible within the narrow limits, and incompatible with the design, of this elementary view of Pathological Anatomy, to more than sketch a general outline of the cystic form of growth and its leading varieties. But here, as in other instances, the omission of some details will not prove of much consequence, because much of the minute anatomy of cysts does not at present admit of any practical application, and that which is applicable to the purposes of surgery will be more profitably considered in subsequent chapters.

The essential characters of a cystic growth are happily described by Mr. Paget in a few words. Only or barely these

characters are found in common: that each growth is essentially "a cyst, sac, or bag, filled with some substance which may be regarded as entirely or for the most part its product, whether as a secretion or as an endogenous growth."* This cyst (fig. 11),†

Fig. 11.



sac, or bag, is either solitary or frequently aggregated with others; and each may be free and moveable, or imbedded in the material of some other growth, so as to form 'a cystic tumour.' But the contents of the cyst or cysts are the chief features of distinction. Some contain *fluid* unorganized secretions, and are spoken of as *simple* or, more correctly, 'barren' cysts. Others contain organized (endogenous) growths, and these are denominated *compound* or, more appropriately, 'proliferous' cysts (fig. 12).‡ But the simple sac is the type, from which the proliferous cyst may be regarded as a departure to a more complex condition; and between the former in its simplest condition of development, and the latter in its most anomalous condition, each intermediate variety may be

---

* Surgical Pathology, vol. ii. p. 26.

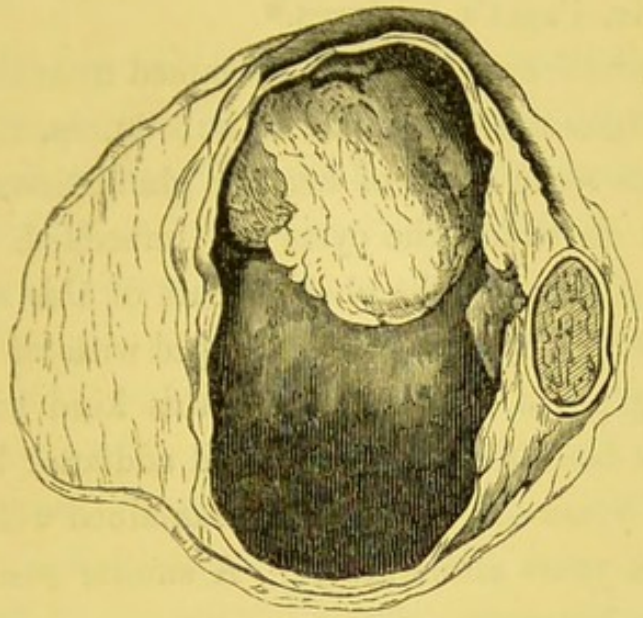
† A simple cyst in the broad ligament of the uterus, with very vascular walls. *a*, new vessels; *b*, broad ligament. 30 *diam.* (*Wedl.*)

‡ Proliferous cyst in a mammary gland. A vascular growth is seen attached to part of the inner surface of the cyst. Below is a smaller cyst, nearly filled with a similar growth. Museum, St. Bartholomew's Hospital. Three-fourths the natural size. (*Paget.*)

distinguished by the contents of the cysts, and the whole arranged in a tolerably even series of progressive organization.

The simplest cyst is formed of fibro-cellular tissue, but without an epithelial lining. This is present in the more finished cysts,

Fig. 12.



and is usually the tesselated variety of epithelium. A more perfect secreting surface is thus prepared, and the varieties of simple cysts take their names from the nature of their secretions—their contents. Thus we recognise the serous, sanguineous, synovial.

These barren cysts may be found in almost any part of the body—and to this subject I shall have occasion again to refer in connexion with my general theory of the distribution of growths; but the seminal cyst has, so far as I am aware, been found exclusively attached to the spermatic cord, and by virtue of the spermatozoa which it contains, may be regarded as on the verge of that higher organization which characterizes the proliferous cyst.

The organized growths found within a proliferous cyst are sometimes simple cells, detached, or pedunculated and attached to the interior of the cyst from whence they have sprung. Of such cysts are formed the common ovarian tumours. Occasionally the sub-cysts are found imbedded in the walls of the parent cyst,

or even projecting from its external surface, so as almost to appear of exogenous formation. This mode of cyst-formation is, I think, illustrated by inference from Dr. Mettenheimer's observations on the structure of the common hydatid mole, or cystic disease of the chorion; but for the details of this supposed process the student is referred to Mr. Paget's Lectures.*

Glandular proliferous cysts are so named from their containing some kind of organized substance or substances, the structure of which resembles some kind of healthy gland-tissue, and for the most part that in which the cysts are imbedded. The thyroid and mammary glands are the chosen seats of this species of cyst. But a glanduliferous cyst may be developed without any connexion with a secreting gland. A tumour of this kind was removed by Mr. Paget from beneath the gracilis and adductor longus muscles of a woman 25 years old. The patient remained well at the end of more than three years afterwards.† A similar case and operation occurred to Mr. Lawrence.

Cutaneous proliferous cysts are so called from their containing skin or its remains, with fat, hair, and other forms of epidermic tissue. These cysts are not necessarily confined to the skin, being more commonly found in ovarian tumours. Teeth may also be discovered within capsules in abnormal situations, and such capsules have received the name of 'dentigerous' proliferous cysts.

Concerning the origin of cysts, whether barren or proliferous, three modes of production are tolerably well established; but no accurate classification of these growths can be made upon this ground of distinction. Cyst-formation is as follows :—

Firstly. By the dilatation and coalescence of the spaces within an areolar tissue, a rude cyst is formed, and afterwards finished off on its internal surface, which becomes smooth, and perhaps lined with epithelium. Thus are formed certain adventitious bursæ,

---

* Ibid., cit., 1853, vol. ii.

† Op. cit. vol. ii. p. 74.

e.g., the little sac which Hunter first pointed out underneath the skin of an old corn.

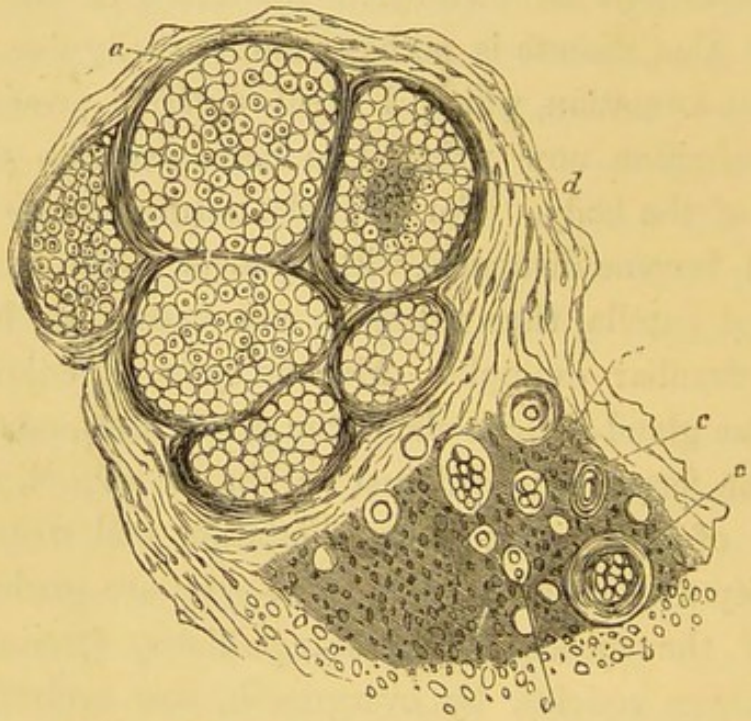
The simple cyst fashioned from areolar tissue may acquire a proliferous power, as witnessed occasionally in adventitious bursæ, from the inner surface of which pendulous little polypi sometimes grow.

Secondly. By the dilatation and distension of certain natural cavities. Of this kind of cysts are those sacculated enlargements of the lactiferous tubes, filled with milk or serum, which Sir B. Brodie first described as 'sero-cystic' conditions of the mammary gland. But this disease is perhaps more usually due to another mode of cyst-formation, which I shall describe presently. The mode of production now referred to may give rise to cysts in many parts of the body. For example, natural bursæ sometimes enlarge, and become distended with synovia. The bursa betwixt the skin and patella thus enlarged, and known as 'housemaid's knee,' is a familiar example. Mucous cysts, by enlargement of the Nabothian gland-follicles about the cervix uteri, or of Cowper's glands in the female situated just within the vagina, are further illustrations of cyst-formation by dilatation and distension of a natural cavity with its own fluid. Fat-cysts are produced in this way, out of the sebaceous and hair follicles, forming common wens. Graafian vesicles, by overgrowth, are evolved into ovarian tumours.

I may here notice certain rare kinds of sanguineous cysts, which, from a case related by Mr. Paget, appear, as he says, to be "dilated portions of blood-vessels shut off from the main streams." Of sanguineous cysts thus formed, one was removed by Mr. Lloyd some years since from a man's thigh. It lay in the course of the saphena vein; but neither that nor any other considerable vein was divided in the operation, or could be traced into the cyst. This cyst was of spherical form, about an inch and a half in diameter, and completely closed; its walls were tough and polished on their inner surface; it was full of dark fluid blood; and its venous character was manifested by two valves, like

those of veins, placed on its inner surface. On one of these a soft lobed mass, like an intra-cystic growth, is seated. The preparation is in the museum of Bartholomew's Hospital. This specimen teaches an important lesson—that a simple cyst, formed by the expansion of a natural cavity, may become proliferous, and this truth is confirmed by the proliferous power of the lactiferous tubes when enlarged in certain cases of sero-cystic disease of the breast, and the prolific growth of cells in the parent cysts of an ovarian cystic tumour.

Fig. 13.



Thirdly. Besides these two modes of cyst-formation, another mode of origin has been discovered, chiefly by the observations of Rokitansky,* Frerichs,† and Mr. Simon,‡ respecting cysts of the kidney. It would appear that certain cells expand and develope themselves into larger cells, which aggregate together in 'nests,' each nest becoming enveloped with a thin capsule of fibro-cellular filaments, which thus forms a cyst containing cells. (fig. 13).§

---

* Ueber die Cyste. Wien, 1850.

† Ueber Gallert, oder Colloidgeschwülote.

‡ On Subacute Inflammation of the Kidney. Med.-Chir. Trans. vol. iii.

§ Proliferous cyst-formations from the cortical substance of the kidney, as a

But the erring cells themselves may each acquire sufficient size to merit the name of a cyst. The cells which naturally inhabit the villi of the chorion, according to Dr. Mettenheimer,* occasionally enlarge into cysts, and form the hydatid mole. From erring cells are sometimes produced serous cysts in the neck—in the thyroid body, in the gums, in the mammary gland, and a cystic condition of the choroid plexus. By this mode of origin, also, certain sanguineous cysts—*e.g.* in the neck; certain adventitious synodial bursæ—*e.g.* ganglions formed in connexion with the sheaths of tendons; and certain seminal cysts. (Paget.)

A surprising proliferous power is frequently manifested by cysts derived from erring cells, of which some instances of cystic disease of the breast and other glands are probably illustrations. Associated with this power of growth and development, is the well-known fact that proliferous cysts frequently recur after, as it would appear, the complete extirpation of the original cyst. Mr. Paget relates a remarkable case of this kind, recorded by M. Lesauvages.† The patient was 63 years old. The first tumour of the breast, of great size, was extirpated in February, 1832; a second appeared, and was removed before the healing of the first wound; a third in May; a fourth in September of the same year; a fifth sprang up, and was removed in February, 1833; a sixth in the ensuing May; by a seventh operation, in June of the same year, three tumours were again excised; but from the same spot two more arose, which grew rapidly, and the patient died.

Infiltrating growths comprise, perhaps, only one genus—

---

sequel to Bright's disease. *a*, the fibrous sheath in progress of development out of *d*, the elongated and caudate nuclei coursing around the parent cyst, or aggregation of parent cysts. They eventually break up into the requisite fibres. *e* is to represent the point-molecule, within an amorphous blastema, out of which the nuclei (*b*) form. They are at first spherical, afterwards elongated, and ultimately broken into fibrillation. *This* constitutes the "alveolar type or arrangement." 90 *diam.* (*Rokitansky.*)

* Müller's Archiv, 1850, h. v. p. 417.

† Archiv. Gén. de Médecine. Février, 1844. p. 186.

cancer, its typical species being encephaloid, scirrhus, and colloid; with many sub-varieties, distinguished chiefly by shades of difference in their general characters of colour, consistence, &c.

But all species of cancer present, under the microscope, the same cell (fig. 14).* This, at first colourless, pellucid, and consisting of a delicate envelope, contains a large clear nucleus or two, sometimes more, never less—within each of which is imbedded one or two nucleoli, also large and clear. Such is the 'cancer-cell' (Bennett). But this cell is not peculiar to and characteristic of cancer. It assumes various shapes, being either

Fig. 14.

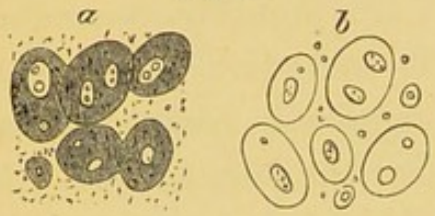
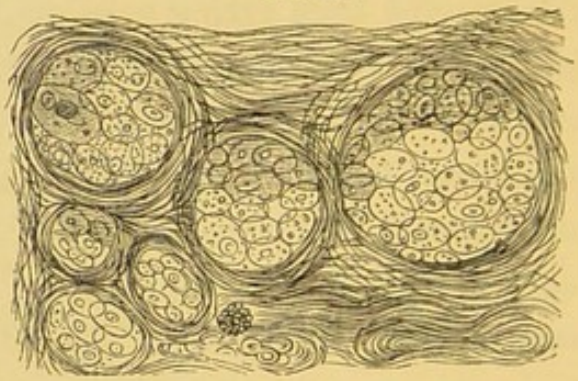


Fig. 15.



round, more usually either caudate or spindle-shaped, and presenting other forms by outgrowths in one or more directions. These cells are deposited in a filamentous stroma or meshwork, which has a variable locular arrangement and closeness of texture (fig. 15).† This intercellular stroma is probably, in most cases, nothing more than the fibrous tissue of the textures, amid which the cancer-cells are infiltrated. But another intercellular substance — gelatinous, translucent, and amber-coloured — may be present in more or less abundance, and *this* is probably peculiar to cancer.

The leading species of cancer are further allied by possessing a similar chemical basis: namely—chiefly albumen, associated with

---

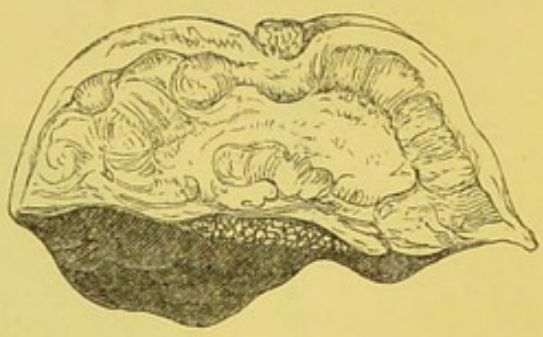
* Cancer-cells *a*, from scirrhus of the mamma. Transparent cells *b*, seen after the action of acetic acid. 250 *diam.* (*Bennett.*)

† Section showing the arrangement of cells and fibrous stroma in scirrhus of the mamma. (*Bennett.*)

fibrin, gelatin, osmazone, fat, certain salts—such as the phosphates and carbonates of lime, with the carbonates of soda and magnesia, the oxide of iron, and water. But the results of chemical analyses hitherto made are not very reliable.

As the proportion of cells, or of either intercellular matter, prevails, so do we recognise 'encephaloid,' or cancer *par excellence*, abounding with *cells*, and therefore soft, opaque, and of a dead white or fawn colour: hence the terms cerebriform or medullary, applied to this form of the disease; and—the cellular, or special element of cancer, predominating, encephaloid yields on pressure an abundant quantity of 'cancer-juice'—which resembles milk or cream. 'Scirrhus,' (fig. 16)* on the other hand, is far more fibrous, and therefore hard and craggy; semi-transparent in a thin section, and of a bluish white or fawn colour; comparatively little 'cancer-juice' is exuded on pressing the cut surface of the fibrous stroma, and this little rather resembles thick gruel than cream, or it may be a small quantity of thin yellow serous fluid; but the fibrous stroma itself contracts, so that the cut surface speedily assumes a concave aspect, unlike the section of any other tumour, which remains level, or becomes slightly convex at its margins.

FIG. 16.



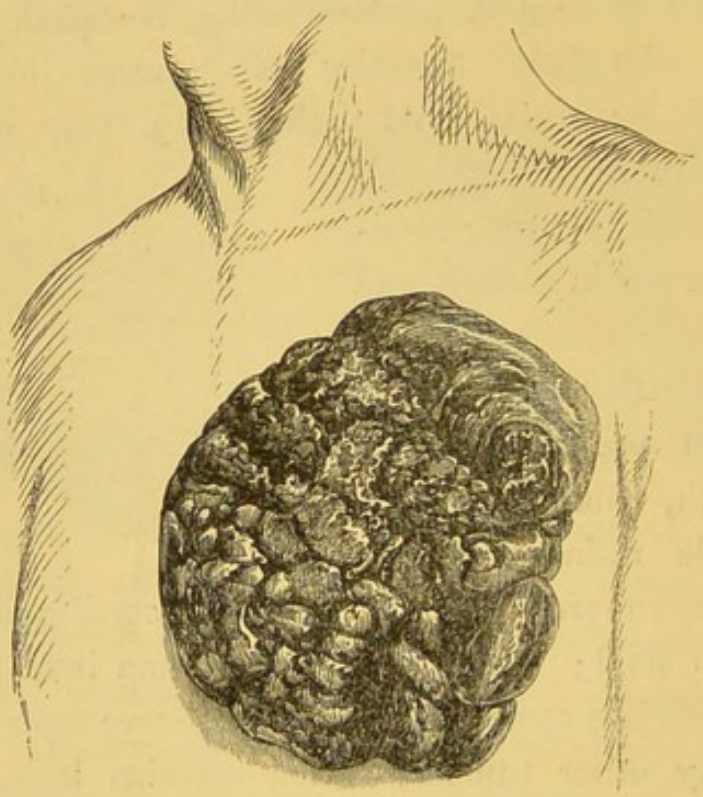
'Colloid'—in contrast with both the foregoing—is gelatinous, owing to the predominance of the gelatinous intercellular matter, and in which the cells are suspended; the whole being infiltrated through a delicate fibrous stroma: it appears, therefore, either as a trembling mass, or a glairy fluid—dimly transparent, and of a greenish yellow colour.

Cancer is essentially an *infiltrating* growth; but while this peculiarity almost constantly prevails in scirrhus and colloid cancers,

* Section of scirrhus cancer of the breast. Early stage. Marked retraction of the nipple. (*Liston.*)

encephaloid becomes encysted, about as frequently as it remains free. A very thin, yet distinct fibro-cellular capsule may invest this typical form of cancer, and from which thin partitions pass into the tumour thus formed, intersecting its substance, or investing its several lobes. Generally speaking, this capsule is not adherent to the surrounding textures; it furnishes a *matrix* in which numerous and tortuous blood-vessels ramify, previous to supplying the mass itself with vessels. Encephaloid is indeed

FIG. 17.



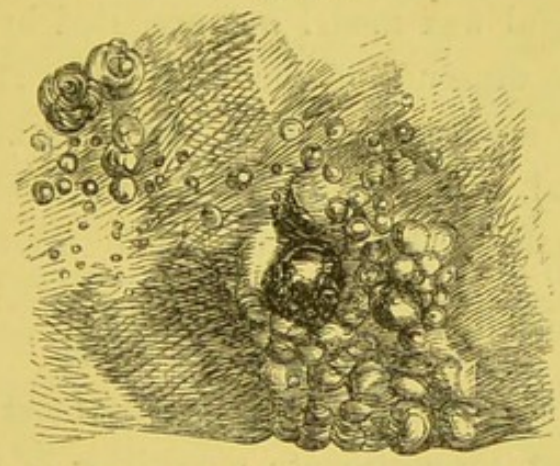
always abundantly vascular, as compared with scirrhus and colloid, both of which are relatively destitute of blood-vessels.

The investing capsule, when present, gives a definitiveness of outline to an encephaloid tumour—round, oval, spheroidal or lobed,—which contrasts with the irregular and unbounded outline of scirrhus and colloid, the infiltrating course of which is very rarely circumscribed. The boundary of either of these species of cancer is perceived rather by their degrees of consistence as compared with that of the textures surrounding the seat of infiltration.

The greater vascularity of encephaloid, coupled with its capsular

and therefore more isolated condition in many cases, are circumstances favourable to a corresponding rapidity of growth, and evolution as a distinct tumour; consequently this, *the* typical species of cancer, frequently attains an enormous size and protrudes (fig. 17) :* but scirrhus, being differently circumstanced in these respects, remains smaller, rarely acquiring a larger size than an orange, and this rendered indistinct by infiltration of the surrounding textures. It shrinks yet smaller by condensation and absorption of texture, as its abundant fibrous stroma, continuous with surrounding parts, draws texture after texture within the claws, as it were, of the infiltrating mass. Infiltration penetrates onward, while contraction pulls backward (fig. 18) ;† so that there is a double action at work—like the pulling on of a glove·

FIG. 18.



Colloid cancer, although ill provided with blood-vessels for rapid growth, is scarcely restrained by a contracting fibrous stroma, and in this particular resembling encephaloid,—spreads to an indefinite size,‡ but not as a distinct tumour.

The circumscribed, or non-circumscribed, condition of cancer, much affects its mobility as a distinct tumour. Encephaloid is often distinctly moveable in the organ or textures in which it is imbedded ; while scirrhus and colloid can be moved about, only

* Advanced carcinoma of the mamma, exhibiting a prominent, fungoid, and bleeding mass. (*Cruvelhier.*)

† Returning scirrhus in the breast, after operation ; presenting a series of nodules in and around the cicatrix. One in the centre has ulcerated. (*Cruvelhier.*)

‡ See Med. Chir. Trans. vol. xxxi. (*Ballard.*)

as a diffused mass in connexion with those textures to which the cancer has contracted adhesions by infiltration.

Such are the chief peculiarities respecting the shape, size, and mobility of the three typical species of cancer—in addition to their individual properties of consistence and colour; and by the concurrence of which characters they can be readily distinguished.

These three species—encephaloid, scirrhus, and colloid—may possibly co-exist and be combined in the same growth. Of this association I once saw a remarkable example in the *post-mortem* examination of a patient who had been under the care of Dr. O'Connor, at the Royal Free Hospital. The abdominal viscera were literally agglomerated into an enormous mass of cancer, which consisted of the three cardinal varieties above mentioned. A beautiful wax model was executed by Mr. Tuson, and is preserved in the museum of the hospital. Then, again, these species of cancer may succeed each other, as well as co-exist, in the same individual. Yet, with all this fraternization, they never lose their individuality; they never become transformed. On the contrary, each species preserves its own general characters, and this, too, through many subordinate varieties. These varieties, like the typical forms themselves, are, in most instances, occasioned by different proportionate quantities of their constituent elements—the cancer-cells and fibrous stroma. Some of these deviations, however, are occasioned by morbid changes of structure,—degenerations, and the consequences of inflammation.

Many varieties have been described; of which some are worthy of notice, not because they possess any essential importance, but owing to the characteristic appearances they present.

The most characteristic of these deviations are fully described by Walshe.* They are distinguished and recognised by their physical appearances, which have suggested appropriate names. The varieties of encephaloid are,—'Mastoid' cancer, so

---

* Nature and Treatment of Cancer. 1846.

named from its resembling, on section, the boiled udder of the cow. This term was originally proposed by Abernethy. 'Solanoid' cancer resembles, on section, a sliced raw potato. It is hard, almost homogeneous, pale yellow, and crisp; hence this name, which was first suggested by Recamier. The substance of such a mass yields milk-like fluid in abundance under pressure, and eventually softens. The 'milt-like' tumour, so named from its general resemblance to the milt of certain fishes—(Monro Tertius). 'Nephroid' cancer exhibits, on section, a peculiar arrangement of the fibrous stroma, and a semi-transparent watery glossiness, with other characters not unlike those which the section of a kidney presents; whence its name—(Recamier). 'Fasciculate' cancer is another variety suggested by the peculiar appearance of its fibrous structure—(Müller). The co-existence of softness, and linear or fibrillar arrangement, constitutes its most readily ascertainable peculiarity—(Walshe). 'Hæmatoid' cancer is a variety of encephaloid, in which the brain-like character is associated with an unusual amount of vascularity, the vessels sometimes interlacing so as to constitute a dense and somewhat spongy network, without, however, the peculiar structure of erectile tissue. 'Fungus hæmatodes' is rather an advanced stage of encephaloid, than a variety of this species of cancer. It represents the occurrence of interstitial hæmorrhage, which either infiltrates the whole mass, or forms irregular accumulations of blood in its substance; and ulceration of the integuments having taken place, a fungoid bleeding growth protrudes. 'Villous' cancer is a term somewhat expressing the appearance presented by this variety of encephaloid. It is very vascular, and apt to bleed copiously. This and its other characters are well marked when the disease occurs on the mucous membrane of the urinary bladder. It is described by Rokitansky as 'dendritic vegetation;' an excrescence consisting, in its stem, of a fibroid membranous structure, on which the branches and villous flocculi are borne, as larger and smaller pouch-like and flask-shaped buddings, or sproutings, of a structureless hollow tissue. 'Melanotic,' or melanoid cancer, is, with very rare

exceptions, medullary cancer modified by the presence of black pigment in its elemental structures—(Paget). This is a species of degeneration; besides which, encephaloid is subject to fatty and calcareous degeneration. Other morbid changes are suppuration and sloughing, which it is very liable to undergo.

The varieties of scirrhus are: so-called 'chondroid' cancer, which is, however, only an early stage of scirrhus; dense and crisp; exhibiting, on section, an unusually shining aspect, and bluish white colour—(Recamier.) 'Lardaceous' cancer is occasioned by the infiltration of scirrhus through the substance of an organ, which then frequently appears not unlike the boiled rind of bacon. 'Napiform' cancer is so named from the peculiar arrangement of its fibrous stroma, which on section has some similarity to a cut turnip—(Recamier). 'Apinoid' cancer derives its name from the striking resemblance presented, on section, to the cut surface of an unripe pear. This similitude arises from the dissemination of comparatively opaque and almost buff-coloured spots through a translucent ground of very pale yellowish lilac tint. The quantity of more opaque substance gradually increases, and eventually predominates, so as to alter the appearance of the surface completely—(Walshe). Allied, is the 'reticular' cancer of Müller. 'Hæmatoid' scirrhus is a condition of rare occurrence; but when it does happen, its peculiarities are of the same kind as those of the hæmatoid variety of encephaloid; differing only in being less fully developed—(Walshe). 'Osteoid' cancer, or ossifying fungus-growth (Müller), occurs as a tumour, consisting chiefly of bone, but having on its surface, and in the interstices of its osseous parts, an unossified fibrous constituent, as firm as fibrous cartilage: after a time, similar growths ensue in parts distant from the seat of the first-formed, and not on bones alone, but also in the cellular tissue, serous membranes, the lungs, lymphatics, &c. It would appear to be the calcareous or osseous degeneration of scirrhus, or of medullary cancer, with which it not unfrequently coexists. Uninterrupted gradations may be traced between the osteoid variety and these typical forms of cancer—(Paget). Scirrhus is subject to other species of degeneration, and to morbid

changes in common with encephaloid. Ulceration is more commonly observed in the course of scirrhus.

Colloid cancer is singularly exceptional—in not presenting varieties, properly so called. Nevertheless, the quantity of fibrous stroma, or of colloid matter, may respectively predominate. If the former, then this species assumes the appearance of a very tough, white, fascia-like mass, in which are small separate cysts or cavities, filled with the colloid substance. In the opposite extreme, large masses of colloid matter appear only intersected by fibrous white cords or thin membranes, arranged as in areolar tissue, or in a wide meshed network—(Paget). Another variety refers to the *quality* of the colloid matter, rather than its quantity. It may be, or become, white and pearly, or opaque. I once met with a remarkable specimen of colloid cancer, in a female patient at the Royal Free Hospital (1862), and which I carefully examined. Most of the abdominal and pelvic viscera were affected with this disease; namely—the stomach; the intestinal canal, which was beset externally, here and there, with pedunculated masses of colloid, somewhat resembling plums on their stalks; two colloid masses were imbedded in the spleen; the pancreas was wholly converted into the same gelatinous substance, enclosed in loculi; the bladder was distended with a trembling mass, which rolled out like a jelly, leaving the mucous membrane pulpy, ragged, and bloody. The substance of the uterus was infiltrated with drops of colloid, together forming a mass which seem to be incorporated with a similar condition of the rectum. But the gelatinous matter, thus extensively diffused, was in some parts white, and of brilliant pearly transparency, looking like colourless and clarified jelly throughout the pancreas, white and opaque in the spleen and uterus; while it presented its usual colour and degree of transparency in the other organs I have specified. This *opacity* of the colloid matter was probably due to molecular disintegration.

These are the principal varieties of each of the three typical species of cancer. Some of them are, as I have said, the result of certain morbid changes, to which cancer itself is liable;—they are

illustrations of a pathological law which prevails extensively—that morbid products themselves undergo morbid changes—the diseases of diseases. Hence their almost numberless, and sometimes perplexing, complications.

As a further illustration of this law, I have yet to notice 'cyst'-formation, in relation to cancer. This relation, if not one of accidental association, represents an actual substitution of cyst-formation for cancer-growth,—and this at the expense of its structural elements. Cysts are either formed with cancer, or from, and out of, the cancer-structures, by their erring development and growth.

Mr. Paget's work contains the best summary of this subject, of which the following is an abstract:—

Respecting cysts accidentally associated with cancer, but formed independently. Scirrhus of the mammary gland may occupy a portion of it only, in the rest of which many cysts are formed, that are in no sense cancerous; or, the chief lactiferous tubes may be dilated into pouches or cysts, contiguous to, but quite independent of, the neighbouring cancer-growth. Such a cancer may nevertheless in its course enclose these cysts, and they remain for a time imbedded in its substance. The ovary may be the seat of cysts, and become also the seat of cancer; the two growths thus accidentally associated, will probably become connected, although of independent origin. Further than this, cancers may grow from the walls of common cysts—*i.e.*, of cysts which have not originated in cancer-structures. Medullary cancer, especially the villous form, sometimes grows from the walls of cysts which have themselves no cancerous appearance.

Cysts derived from cancer structures—by their erring development and growth—constitute a series parallel with that of the cysts simple and proliferous, which form in innocent tumours or in the natural textures. Cysts having this origin are therefore either simple or proliferous.

Of the former species are:—Cysts filled with serous fluid, variously tinted. Serous cysts are often born in cancers, especially

in those of the medullary type, which grow quickly, or to a great size. One or many such cysts may be present on the surface or in the semblance of a cancer. Sometimes a single cyst of this kind enlarges so as to surpass the bulk of the cancer, exceedingly perplexing the diagnosis. Sometimes many cysts are present, as if the tumour were entirely composed of them, with cancerous structure only in their interstices. Sanguineous cysts are born as often as serous, in medullary and other cancers. The imprisoned blood undergoes changes in respect of colour and consistence, thereby diversifying considerably the appearances presented by cancers containing these cysts. Colloid cysts, *i.e.* cysts containing a glairy jelly—not cancerous—may likewise be developed in cancer-growths, by conversion of their structural elements.

The proliferous cysts which originate in cancers, bear on their inner surface cancerous growths—thus corresponding with the glandular growths which spring from the interior of cysts in the mammary and thyroid glands. These endogenous growths are often found in the alveoli of colloid cancer. Clusters of clavate, or flask-shaped villous processes, resembling those formed in the early stages of 'dendritic vegetation' of villous cancer, spring from the wall of the alveolus.

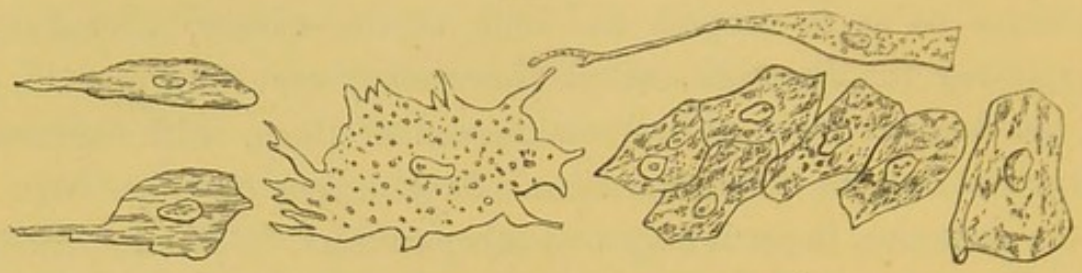
The origin and modes of development of these cysts—simple and proliferous—have been traced by Rokitansky, and shown to correspond with those of all other cysts: the only difference being the *source* of the cyst-formation—here a cancerous element, while in respect of all other cysts it is an element of some natural tissue.

One variety of cancer I have reserved for special description; because, although in many points allied to encephaloid, and somewhat to scirrhus cancer, it nevertheless presents very characteristic appearances, and possesses much surgical interest. I allude to 'epithelial' cancer.

This variety consists essentially of cells or scales, resembling those of scaly epithelium, infiltrated among the component textures of the skin or mucous membrane, and sometimes among the textures of internal organs. Whatever portion of the skin

or mucous membrane may be thus affected, the epithelial cancer-cell or scale has very much the same characters (fig. 19.)* It is

FIG. 19.



flattened, of an irregular outline, with usually a prolonged diverticulum at some point of its margin, and of a variable size. It contains pale molecular matter, converging towards a central nucleus, which is clear, bright, and well-defined, round or oval, and very small in comparison with the cell; more uniform also in shape and size than the cell. This nucleus is usually single. It may contain two or more minute granules, but rarely a bright distinct nucleolus.

Associated with these cells are what Paget calls 'brood-cells,' or endogenous cells. They present many varieties of appearance, which may be regarded as the results of one or more nuclei, enclosed within cells, assuming, or tending to assume, the characters of nucleated cells.

The 'laminated capsules' of Paget—'globes epidermiques' (Lebert), are the most singular and characteristic structures of epithelial cancer, but not peculiar to this disease; nor are they, apparently, special structures, for they consist of epithelial scales. These capsules are very large and spherical cysts, containing granular matter, nuclei, or cells, obscurely seen within them; and they may be clustered, so as to almost appear as if fused together; but each capsule consists of epithelial scales—superimposed in successive layers, thus forming a laminated capsule.

Such, then, are the structural elements of epithelial cancer. They are found infiltrated—principally in the substance of the

---

* Epithelial cancer-cells or scales, in various forms. Magnified 350 times. (*Paget.*)

skin or mucous membrane—but not uniformly diffused throughout the component textures of the part affected.

The cancer-cells may predominate in the 'corium'—forming a swelling very slightly elevated above, or imbedded below, the proper level of the integument, and the depth or thickness of which is much less than its dimensions laterally; or these cells may predominate in the 'papillæ'—presenting a prominent warty or exuberant outgrowth; or the 'sub-integumental texture' may be their chief seat—forming a deeper-seated, flat or rounded mass.

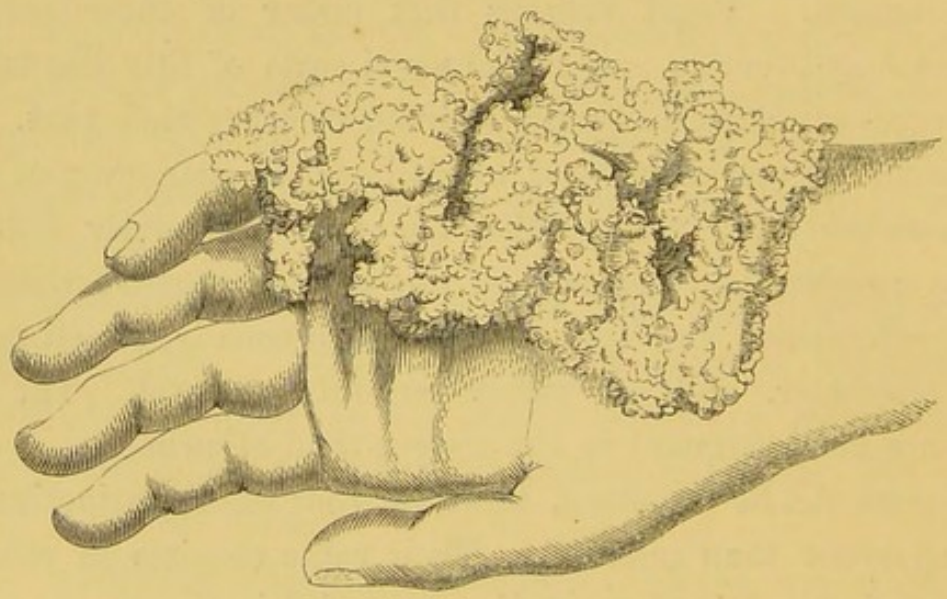
Of these varieties, the first two may be named the superficial or outgrowing; while the third is the deep-seated form of epithelial cancer. Paget believes that either of these principal varieties may occur in any of the usual seats of this disease, but that they are not both equally common in every such part. The superficial, and especially those which have the characters of warty and cauliflower-like outgrowths, are most frequently found on mucous membranes, especially of the genital organs; those also on the extremities and the scrotum have usually a well-marked warty character, and are rarely sub-integumental. The deep-seated are more frequent in the tongue than elsewhere.

It must not be forgotten, however, that these distinctions are more apparent than essential. Their value consists in reference to the earliest and most exact diagnosis of this disease—in whichever form it may chance to make its first appearance. For subsequently—as Paget himself remarks—and especially when ulceration has commenced, an epithelial cancer which was superficial or exuberant, is prone to extend into deep-seated parts; and one at first deeply seated may grow out exuberantly. Moreover, when ulceration is progressing, a greater uniformity of external appearance is found; because, in general, while all that was superficial or exuberant is being destroyed, the base of the cancer is constantly extending, both widely and deeply, into the sub-integumental tissues.

Respecting, then, the earliest appearances of epithelial cancer, which are fully described by Paget, the following are most characteristic.

Of the superficial or outgrowing, prior to ulceration, they are these: — an outspread swelling arises—say on the lower lip, labium pudendi, or scrotum; and an unnatural firmness or hardness of the affected skin is perceptible; but the superficial dimensions much exceed the thickness of this swelling. Its outline is round, oval, or sinuous; and its surface, sometimes nearly smooth, is more often coarsely granulated—like that of a syphilitic condyloma,—deriving this character from the enlarged and closely clustered papillæ. Generally, the surface is moist with an ichorous discharge: it may, however, be covered with a scab, or

Fig. 20.



encrusted with a soft substance, consisting of detached epithelial scales. In most cases the part is unduly sensitive, and injected with blood. If the papillæ become infiltrated, they constitute the cauliflower-like mass so characteristic of the ordinary form of epithelial cancer (fig. 20).* This mass looks very vascular, is moist with ichor, and covered with pasty cakes of epithelial scales, which beset the interstices of the enlarged papillæ.

---

* Epithelial cancer of hand; showing the papillary character, from specimen in the museum of St. Bartholomew's (Ser. x. i. 6). The history of this case is in Potts' Works, by Earle, iii., 182. The patient was a gardener, who had been employed in strewing soot for several mornings. The disease was of five years' duration. (*Paget.*)

Occasionally, the shape of an outgrowing epithelial cancer is that of a sharply bordered circular or oval disk, upraised a little above the level of the adjoining skin or mucous membrane, and imbedded to about the same depth below it. The surface of this disk—usually flat or slightly concave—is granular, spongy, or irregularly cleft; and its margins are surrounded with healthy texture, which becomes raised and often slightly everted by their enlargement; *e.g.* many epithelial cancers of the tongue.

Sometimes epithelial cancer grows out in the form of a cone.

Lastly, the outgrowing form of this disease may be a narrow-stemmed, and possibly pendulous, growth from the skin. These and other shapes of superficial epithelial cancer resemble somewhat the appearance presented by warty and condylomatous growths; but they differ essentially in respect of their minute structure;—being infiltrations of the skin and papillæ with epithelial cancer-cells; whereas the structure of warty growths remains healthy, however strange may be the appearance they assume.

Deep-seated epithelial cancer is generally an advanced stage of the superficial; for by progressive infiltration, the subcutaneous or submucous tissues become invaded: but this variety of the disease may occur primarily, although comparatively rarely.

Thus "the first formation of epithelial cancer may be in masses of circumscribed infiltration of the tissues, beneath healthy skin or mucous membrane."

"This condition is more frequent in the epithelial cancers that form, as recurrences of the disease, near the seats of former operations, or as secondary deposits about the borders of primary superficial growths."*

The *ulcerative* stage of epithelial cancer is that in which the disease is usually seen; and the usual state of ulceration observed is that of progressive destruction of the central and superficial parts of the cancer, with more than coextensive growth of the

---

* Surgical Pathology. (*Paget.*)

marginal and deeper parts—thus presenting the type of the 'cancerous ulcer.' It is important, therefore, to be able to discern the first aspect of ulceration—both as regards the superficial and the deep forms of epithelial cancer respectively; and then the characters of the 'complete ulcer.'

Ulceration of superficial or outgrowing epithelial cancer, primarily appears as either a diffuse excoriation of the whole surface of the cancer, except its borders, or else a shallow ulcer, limited at first to some fissure where the disease commenced. The discharge from this excoriated or ulcerated surface usually dries into a thin scab, or a thicker and darker crust, which conceals for a while the ravages of ulceration, still slowly extending beneath, —downwards and outwards.

Ulceration of deep epithelial cancer begins in one of three ways.

In some cases the superimposed skin or mucous membrane having become adherent and thin, cracks; and this condition may remain stationary for a long while, in the form of a dry dark crevice; usually, however, ulceration commencing from this point extends into the mass of the cancer.

In other cases, the substance of the cancer having become inflamed, it softens, suppurates, and discharges its contents through an ulcerated opening, or a long rent; leaving a cavity which speedily assumes the characters of an ulcer, and extends peripherally.

In a third series of cases, and—Paget is disposed to believe —especially in secondary formations, and in those under the scars of old injuries, the cancer protrudes through a sharply bounded ulcer in the sound integument or scar, and grows exuberantly, with a soft shreddy surface, like a medullary cancer, or with a firmer, warty, or fungous mass of granulations.

Dissimilar as are the earliest aspects of ulceration in both forms of epithelial cancer, outgrowing and deep, they gradually assume an uniformity of appearance which is very characteristic.

An excavated sore, of a round or oval shape, presenting a roughly granular surface which has a brick-dust red colour, and oozes a stale-smelling discharge. This surface bleeds easily, al-

though not freely. The textures surrounding its base and borders become indurated and rigid, as they progressively become more and more infiltrated with cancer-cells; so that the ulcer acquires a remarkable degree of immobility, and its margin protrudes in the shape of a thick everted ridge, well defining the boundary of the ulcer; but, this bordering infiltration increasing, the marginal ridge crops out, and presents an irregular nodular belt, which overhangs the base of the ulcer, and gives an undermined appearance to its everted borders.

If the papillæ of the surrounding skin are more particularly the seat of cancerous infiltration, then a warty rather than a nodular belt springs up around the ulcer.

With all these unequivocal signs of progressive infiltration, followed by ulceration, let it not be supposed that the work of destruction is stayed below the base of this already excavated sore. It spreads deeper and deeper, sparing no texture, not even large arteries, which hold out against the invasion of most other ulceration, if indeed they do not eventually escape altogether. But arteries of the first magnitude yield to the unsparing ravages of epithelial cancer.

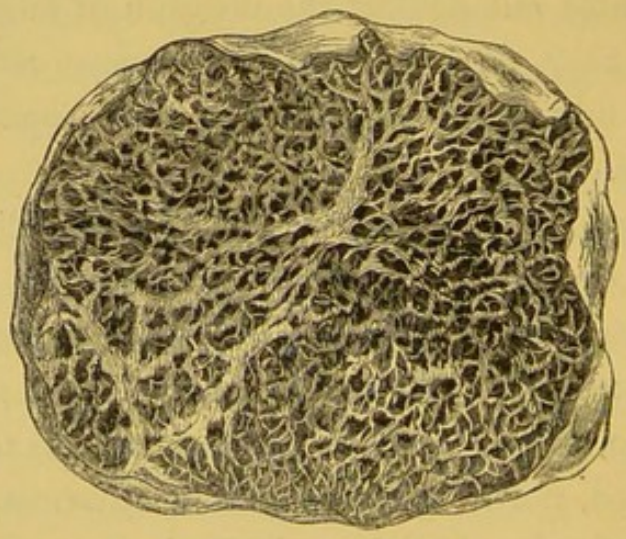
There are good and sufficient reasons for regarding this disease as a variety of 'cancer.' Its infiltrating character, as a growth, is the essential guarantee of identity; and its indiscriminate invasion of all textures, next the ulcerative tendency just mentioned, is a sign of affinity to cancer; and the obstinacy of this tendency should not be overlooked, nor *its* unsparing destructiveness. Then again, the lymphatic glands are liable, although not prone, to become affected by the same epithelial infiltration, which constitutes the primary growth; but these secondary formations are almost exclusively limited to those glands which are in connexion with the immediate vicinity of the primary disease. A similar epithelial infiltration is, however, liable to spring up indiscriminately in parts *distant* from the original disease; and coupled with this resemblance to the career of cancer, is the more significant fact of recurrence after removal by operation. Lastly, members of the same

family, in which either scirrhus or medullary cancer occurs, are peculiarly liable to the epithelial infiltration. What else then, duly weighing all these reasons, singly and collectively, can we name this disease but epithelial *cancer?* In this conclusion the most able pathologists concur—Rokitansky, Virchow, Paget, and others. Epithelial cancer must not be confounded with the 'epithelioma' of Hannover and Bennett, who include under that title many other growths besides this variety of cancer; and in like manner it differs from the 'cancroid' of Lebert.

There is yet another kind of growth, which I have reserved to the last, because in point of structure it is less a new and distinct species of growth, than an hypertrophy or overgrowth of healthy texture. I allude to the 'vascular,' erectile and pulsating tumour, otherwise than aneurism.

This tumour (fig. 21)* essentially consists of a conglome-

FIG. 21.



ration of blood-vessels, connected together, more or less intimately, by fibro-cellular connective tissue, which may form an investing capsule; the whole being thus circumscribed as a distinct tumour, but of irregular shape, and perhaps lobed: or the mass

* Section of an erectile tumour, showing the fibrous trabecular structure and investing capsule. Museum of the Royal College of Surgeons, England. Drawn one-third larger than the specimen. (*Paget.*)

not being distinctly isolated, is diffused, flattened, and shades off into the surrounding textures. The size or extent of such a mass, in either case, varies with its growth, and is subject to temporary alterations, under circumstances which will be noticed presently.

This species of tumour may be deep-seated in or among muscles, in bone, the stomach, spleen, liver, the orbit, the tongue, and indeed wherever capillary blood-vessels naturally exist and most abound. The diffused form is more frequently situated superficially, as beneath the skin, and probably involving it; constituting the common cutaneous nævus, as seen on the scalp or the face.

Three varieties of this tumour are well defined, and easily recognised when it is patent. The constituent blood-vessels are capillaries, with arterial and venous trunks proceeding respectively to and from the mass; but either order of vessels may predominate, and constitute nearly the *whole* mass, apart from the connective tissue. Thus there are the capillary, the venous, and the arterial varieties of vascular tumour.

The first variety consists almost entirely of capillaries—large, dilated, and convoluted. The second, almost entirely of veins or venous sinuses, forming a cavernous and erectile structure. This variety of vascular tumour was that which John Bell specially described,* and to which he gave the name of "aneurism by anastomosis," or "aneurism by the dilatation of anastomosing vessels."† He compared it to the erectile structure of the penis, the gills of a turkey-cock, or the substance of the spleen, placenta, or womb. The third variety consists almost entirely of arteries—large, tortuous, and perhaps convoluted. *This* is now called "aneurism by anastomosis."

These varieties of structural condition are accompanied with corresponding peculiarities of physical characters, by which their diagnosis, during life, may be determined.

---

* Principles of Surgery. Ed. C. Bell, 1826. Vol. iii. p. 328.

† Ibid., p. 386.

The vascular tumour is soft and compressible, but regains its former size when the congeries of vessels is left free to fill again. Its substance somewhat resembles a sponge; and if visible, as when subcutaneous or in the skin, the colour of this distended sponge plainly shows that it is distended with *blood*, arterial or venous. The capillary and venous varieties are alike characterized by their circumscribed shape and soft doughy consistence, which can be moulded under the fingers; but at the same time a trembling sensation or indistinct throbbing is felt, if the mass be chiefly venous, and have attained some size. After compression, the tumour slowly re-distends, and assumes increasing size and tension during exertion—especially if sudden and violent — as running, coughing, straining, struggling, and by any obstruction to the free flow of venous blood. A bluish tint is perceptible when the tumour is situated superficially. Lastly, if wounded, the mass collapses. The arterial variety is chiefly characterized by its strong pulsations, and threatening distension, increasing also under any occasional excitement of the heart's action, and with the flow of arterial blood.

The vascular tumour is not unfrequently a congenital growth; it may be a subsequent formation, but more frequently in childhood than in after years. The progress of growth is not generally steady, but by fits and starts; sometimes rapid, sometimes slow-growing. Very commonly—observes Paget—their increase is only proportionate to that of the rest of the body; and when full growth is attained, they also cease to grow. Not rarely they are stationary, or shrink, even while the rest of the body is growing; and this is especially likely to happen with those that are white and scar-like where the skin is involved.*

The same authority also notices certain structural alterations to which the vascular tumour is subject—by degeneration or by disease.

These morbid conditions disguise the original character of the

---

* A System of Surgery. 1860. Ed. by T. Holmes. Vol. i., Tumours.

tumour, and mislead diagnosis. Thus, a vascular tumour may have become converted "into a soft, but tough and dry, yellow ochre or brown substance, like that of a supra-renal capsule." Scar-like patches is another transformation which the substance or surface of this tumour is liable to undergo; and this change is accompanied with closure of the vessels in that portion. Acute inflammation may consolidate and cure; or leading to ulceration, form bleeding sores, ill-disposed to heal. Either blood-clots, or phleboliths, sometimes partially consolidate; or cysts, serous or sanguineous, give a more elastic feel to the mass. More rarely, this kind of growth forms in another growth, and usurping its structure, converts part, or the whole, of such growth into a vascular tumour, and acquires its characters. Lastly, "it is probable also that one form of vascular tumour may be converted into another: a capillary one becoming either arterial or venous, by the excessive growth of one or other set of vessels; or an arterial one, by opposite changes, becoming either capillary or venous."

Osseous and glandular growths are ranked by some authors in the class of tumours; but the former are, with very rare exceptions, outgrowing hypertrophies of bone (exostoses), and the latter, hypertrophies, not outgrowing, of glandular organs; neither are *discontinuous* overgrowths, and, this characteristic of all tumours being absent in respect of these growths, I shall pass them by.

Aplastic Products remain to be considered. They are essentially, as their name implies, unorganized. Thus negatively distinguished from the former division by the absence of structure, Aplastic products are, furthermore, chemical compounds, both inorganic and organic, which are adventitious, by not entering into the normal composition of the solids and fluids in which they are found. Their physical state varies, being more frequently solid, occasionally fluid, rarely gaseous. They are formed, for the most part, in diseased secretions, particularly in those from the mucous membranes; but also in the secretions of serous and synovial membranes, and of the skin. Their most characteristic forms are

represented by *calculi* and *concretions*, the latter being distinguished by their possessing, not indeed a distinct structure, but an ill-defined organized basis, with which the new chemical compounds are intermixed. This partial structure makes concretions the link, as it were, between plastic and aplastic products.

Of calculi are found many varieties, the chief of which consist of lithic acid (fig. 22), lithate of ammonia (fig. 23), oxalate of

FIG. 22.

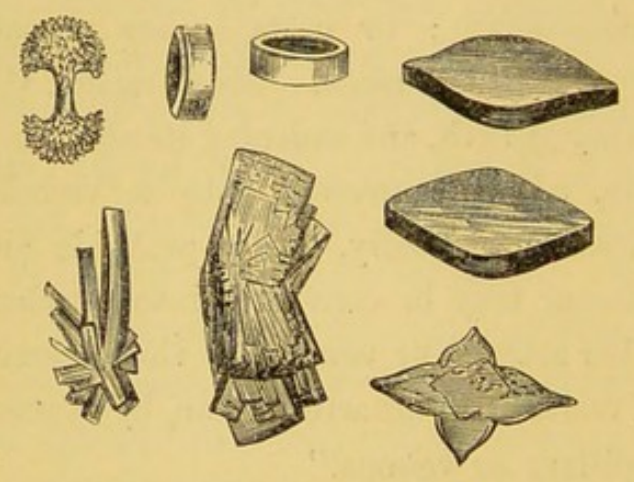
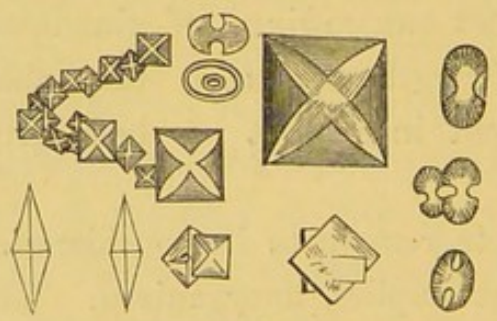


FIG. 23.

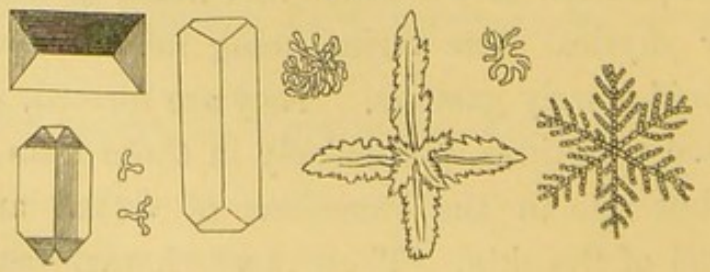
lime (fig. 24), ammonio-phosphate of magnesia with lime (triple phosphate) (fig. 25); amorphous phosphates and carbonates of

FIG. 24.



lime, and lastly, the cystic oxide calculus, which is very rare. Concretions are illustrated by certain bodies, occasionally found,

FIG. 25.



either free or attached, within arteries—arteroliths, more frequently within veins—phleboliths.

Besides the large class of 'supplemental' or additional Morbid Products, properly so called, and which are either aplastic or plastic, the latter comprising "false tissues," "deposits," and "new growths," Pathological Anatomy extends to Degeneration of textural structure, manifested in various ways.

Firstly, however, it is necessary to clearly understand the meaning of (this generic term) Degeneration, and this implies its comparison with healthy Nutrition, of which Degeneration is, strictly speaking, only a phase or modification.

The functions of the various organs and parts of the body are originally so adapted or adjusted, as to constitute an evenly balanced whole living organism. This original status is continued throughout life, by virtue of the vital powers, congenitally bestowed; but the condition of balance thus secured is itself regulated by the nutritive power of the body during the successive periods of its existence. After birth and an independent existence, during infancy, youth and adolescence, to maturity, the function of nutrition is yet more one of growth and development. During mature manhood or womanhood, this function is one simply of maintenance, to repair the molecular waste of the body consequent on the functional exercise of its various members and organs. Lastly, as age approaches, and during decrepitude, the nutritive power of the body proportionately declines. Waste is still repaired; but the textural structure reproduced is an imperfect representation only, not a copy, of either its original or mature condition; it is a 'retrogression' of textural structure, effected by a *degenerative* modification of Nutrition. Degeneration is therefore only the concluding stage or phase of the natural course of healthy nutrition, and as the concomitaut retrogression of textural structure extends more or less throughout the body, and certainly pervades vital organs, the whole organism retrogrades or reverts to the unorganized matter from whence it came. Earth returns to earth, ashes to ashes, dust to dust. To die by 'degenera-

tion' is therefore as natural as to live by 'growth' and 'development,' and by the subsequent 'maintenance' only, of Nutrition. This function is indeed the consummation and resultant of all the other organic functions; and when, in the order of Nature, they decline, nutrition declines also into degeneration, which it then represents. But this ultimate phase or modification of nutrition is sufficient as age advances, and all other functions also decline simultaneously and proportionately.

The *senile* retrogression of textural structure, thus effected, is not, therefore, in any sense a diseased condition, nor the result of any morbid process. Nor again, when degeneration, natural to advancing and advanced life, occurs *prematurely*, can it then be regarded as a morbid process of nutrition, issuing in a diseased condition of textural structure. It is only premature old age; but the individual overtaken by degeneration is the subject of *defective* nutrition. In this sense only, retrogression of textural structure, effected by degeneration, ranks, and may be classed, with *morbid* products of Nutrition. Degeneration—whether senile or premature — is a form of atrophy; but it is so by a deterioration of structural quality, not by a mere diminution of quantity. And be it observed, this (structural) deterioration is effected by the 'relapse' or falling back from a higher condition to a lower or more elementary grade of textural structure. It takes the place of the proper constituents of the original texture, which concurrently disappear. The resulting metamorphosis I would therefore designate 'substitute' texture, to distinguish it from mere transformation of texture, resulting from other and widely different causes, as 'disintegration.' Analogy and facts alike concur to render the conclusion highly probable that this substitution of texture by degeneration or a modification of nutrition is effected *primarily* by an appropriate 'blood-disease' in each case, of which it is the local and anatomical manifestation. It may, however, be opportune to remark, that Paget expressly points out the parallelism of premature degeneration and simple atrophy of quantity, in respect of their causes: such as diminished supply of

arterial blood, as by partial closure of the chief nutrient artery of the part; or again, abrogation or suspension of function. Furthermore, that both degeneration and atrophy occur commonly in old age, but possibly prematurely; that both may occur simultaneously in one and the same texture, under precisely the same conditions; and, that a cause of atrophy in one case may cause degeneration in another.* In relation to disease, the same author observes, both may concur, as in inflammation; or the disease, so-called, may be only degeneration, as in simple softening of the brain or spinal cord, and the liquefaction of inflammatory exudations during the suppurative process. The whole may be termed "liquefactive degeneration."†

Having made these references, in order to show the accepted and prevailing opinions respecting degeneration as a process, and its etiology, the general diagnostic characters of all degenerations are thus enumerated by this author:—

They are such changes as may be observed naturally occurring in one or more parts of the body at the approach of the natural termination of life, or if not then beginning, yet then regularly increasing.

They are changes in which the new material is of lower chemical composition; *i.e.* less remote from inorganic matter than that of which it takes the place. Thus, fat is lower than any nitrogenous organic compound, and gelatine lower than albumen, and earthy matter lower than all these.

In structure, the degenerate part is less developed than that of which it takes the place: it is either more like inorganic matter, or less advanced beyond the form of the mere granule, or the simplest cell. Thus, the approach to crystalline form in the earthy matter of bones, and the crystals in certain old vegetable cells, are characteristic of degeneration, and so are the granules of pigment and of many granular degenerations, and the globules of oil that may replace muscular fibres or the contents of gland-cells, and

---

* Surgical Pathology, vol. i. † Ibid.

the crystals of cholesterine that are often mingled with the fatty and earthy deposits.

In function, the part has less power in its degenerate than in its natural state.

In its nutrition, it is the seat of less frequent and less active change; and without capacity of growth, or of development.*

The various forms of degeneration are partly suggested by these general remarks; but pathologists differ as to their number and specific differences.

The classification of Paget is not that of Dr. J. Hughes Bennett; nor this again, that of Dr. C. J. B. Williams.

As this branch of Pathology stands at present, I would propose the following arrangement:—

(1) Fatty, and (2) Pigmentary degeneration. (3) Fibrous, including (4) Amyloid, waxy, or lardaceous degeneration. (5) Granular degeneration. (6) Calcareous, osseous, or mineral degeneration.

Each of these modes of degeneration merits a separate description; and, not to complicate the subject, I shall regard the process of degeneration as synonymous with its *result*—'retrogression of textural structure.'

'Fatty' degeneration presents certain general, yet distinctive characters—structural, physical, and, it may be, chemical. Examination with the microscope shows minute granules in the very substance of some structural element; be it a cell—in its contents, or in the place of its nucleus; if a primitive fibre—in its substance; if a simple membrane—in its substance: thus, in all cases substituting, at least in part, the original structural element, which has undergone this degenerative transformation. These granules are particles of fat or oil; for being highly refractive, they are bright and glistening; they are soluble in ether; and they tend to coalesce into distinct drops of oil.

Proportionately as the oil-granules usurp the place of an elemental structure, its proper substance disappears. For example,

---

* Op. cit., vol. i.

a cell having become full of oil-particles—a 'granule-cell'—its walls disappear, leaving only a 'granule-mass;' and these granules are apt to coalesce into drops of oil.

The interstitial tissue of the texture usually undergoes fatty degeneration, simultaneously. And the disposition of *these* oil-granules is peculiar and significant. They are arranged, as Virchow observed in the liver, in the course of the capillary blood-vessels, which become thickly studded with particles of oil, as if deposited from the blood. But I shall have occasion to return to this subject.

The general physical characters of a texture whose structural elements have undergone fatty degeneration are these:—The texture is softened, or if soft in its healthy condition, it has now assumed a doughy consistence, which retains any impression or mould given by the fingers, as if it were soft putty; but this fatty texture is friable, greasy, burns readily and with a bright flame, and yields, by analysis, an unusual quantity of fatty matter—(Paget). It has also acquired a dirty-brown colour, here and there mottled with a dead-leaf or wash-leather colour; these patches corresponding to the most advanced stage of fatty degeneration.

Muscular texture in the state of fatty degeneration well illustrates these physical characters; while the co-existing changes of minute structure, as revealed only by the microscope, are also well defined, and these are characteristic of the degeneration. I select the muscular texture of the heart; because while, on the one hand, its fibres being striated, resemble and represent those of voluntary muscle, the question of fatty degeneration of this organ is moreover one of cardinal importance in relation to Medicine and Surgery; and also, I may add, in relation to Hygiene, and Medico-legal Inquiry.

The history of our present exact knowledge respecting fatty degeneration of the heart proves beyond a doubt that this condition of its muscular texture was for a long time confounded with the mere *interstitial* deposit of fat in the muscular substance of this

organ. For the physical appearances of both these conditions are similar. They are those which I have just described, and they conspicuously contrast with the healthy characters of muscular texture.

Portions of the heart, commonly of the left ventricle, have acquired a mottled yellowish appearance, chiefly visible on the interior or the external surface of the ventricle, immediately underneath the endocardium or pericardium. When viewed more closely, these spots present wavy transverse lines. Each spot is not abruptly defined, but tinted off into the adjoining healthy texture. If degeneration be incipient, these spots are yellowish brown; if advanced, they have assumed a dead-leaf colour. The muscular texture is no longer fibrous, but friable; and if the degenerative transformation be extensive—involving a considerable portion of the whole substance—the heart will have acquired a doughy softness, which retains any impression given by the fingers. The organ is palpably greasy, and its texture burns readily, and with a bright flame. Chemical analysis is wanting to determine the alteration of composition which the texture has undergone.

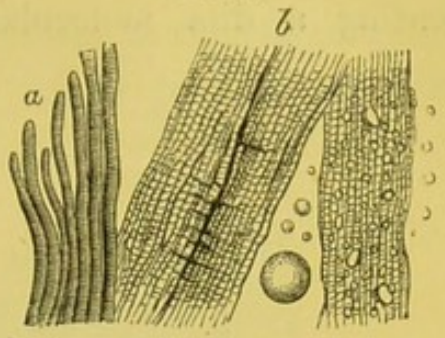
Both ventricles are more subject to this degeneration than the auricles; but, although rarely, the muscular substance of the whole heart may undergo the same transformation.

The foregoing physical appearances had long since been observed by Laennec, and previously by Dr. A. Duncan, in 1816, and would even seem to have been known to Lancisi and Morgagni; but many such observations referred to fat, merely accumulated, on the surface of the heart, and deposited interstitially amongst its fibres. It was reserved for the researches of Rokitansky, Drs. C. J. B. Williams and Peacock, and more especially for those of Mr. Paget, Drs. Ormerod and Quain, to fully demonstrate the changes of structure which the muscular fibres themselves undergo by this mode of degeneration.

These alterations of textural structure were at length revealed by examination with the microscope. The fibrillæ evince a disposition to disintegrate—as shown by their readily splitting—longi-

tudinally and transversely, (fig. 26, *a* and *b*, *Wedl*). The transverse striæ become less distinct, and oil-particles visible—*within* (the sarcolemma of) the fibres, and externally—besetting the fibres, a condition represented by the fibre to the right of *b*, (fig. 26); but this is not an essential condition. The former may be termed *intra* oil-particles, to distinguish them from the latter, between the fibres, or inter oil-particles.

Fig. 26.



The intra oil-particles appear,—firstly (figs. 27 and 28),* as minute granules, arranged transversely, as it were, across the direction of the fibres; thus corresponding to the sarcous elements

Fig. 27.

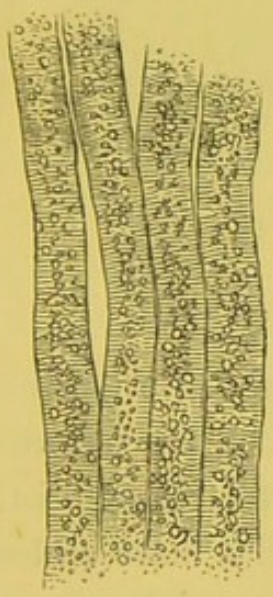
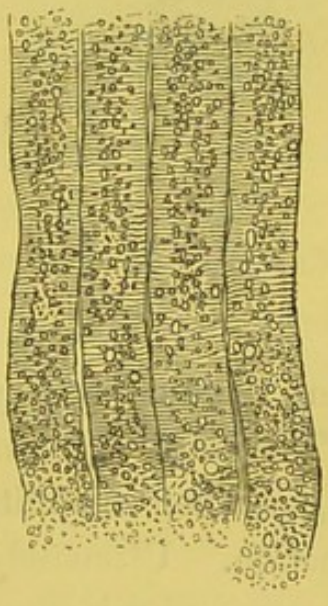


Fig. 28.



whose place they occupy, but from which they are readily distinguished by their bright glistening appearance, being highly refractive, and by their solubility in ether. Subsequently, in a more advanced stage (fig. 29, *Wedl*), fig. 30,† or simultaneously in other adjacent fibres (the transverse striæ have entirely disap-

---

* Fatty degeneration of muscular fibres of heart in cattle, exhibited by the Smithfield Club as prize specimens of breeding and feeding, 1857. (*The Author.*)

† Another prize specimen; the *best sheep*, of any long-woolled breed, one year old, showing more advanced fatty degeneration of muscular fibres of the heart. (*The Author.*)

peared); the intra oil-particles have no definite arrangement, each fibre consists of oil-particles confusedly aggregated together, presenting a dim molecular aspect, in this respect somewhat

FIG. 29.

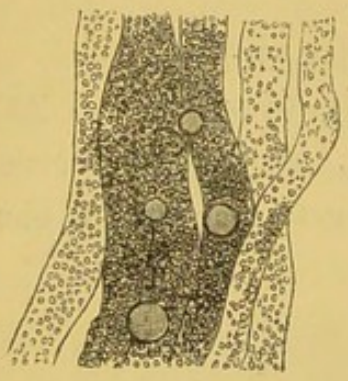


FIG. 30.

resembling the healthy appearance of unstriated organic muscular fibre. The bright glistening appearance of the aggregated oil-particles, and their solubility in ether, are still characteristic marks of distinction. Ultimately, the oil-particles have coalesced into distinct *drops* of oil (fig. 31),* some of which adhere to the (interior of the) sarcolemma, which still remains unbroken and enclosing them. The large oval nuclei, which are perhaps peculiar to the muscular fibres of the heart, have become obscure, or altogether disappeared. But no fat-particles or drops are commonly found between the fibres, and the absence of any interstitial fat is conspicuous, unless the sarcolemma has partially

---

* The *best* Devon ox, above three years old, showing most advanced fatty degeneration of muscular fibres of the heart. (*The Author.*)

disappeared in the course of degeneration, or has been ruptured in displaying the fibres with needles.

Fatty degeneration is regarded as a *chemical* transformation.

It is alleged that this conclusion might be inferred from

Fig. 31.

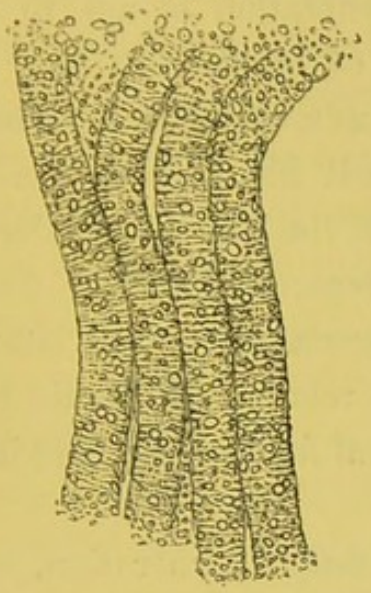
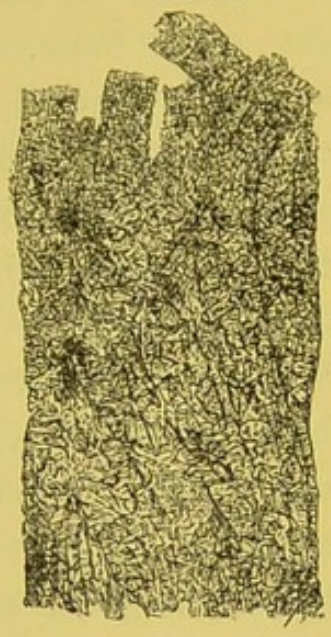


Fig. 32.*



the fact, that in fatty degeneration of striated muscular fibre the oil-particles are primarily arranged in the same manner as the proper structural constituents, or sarcous elements of the fibrils, whose place they occupy. It is urged as a more direct proof, by Dr. Quain,† supported by Dr. C. J. B. Williams,‡ Paget,§ Bennett,‖ and by many other distinguished pathologists, that fatty matters are produced during the spontaneous decomposition of nitrogenous substances, as the formation of adipocire in muscular tissue; instances of which are given by Virchow.¶ Then again, muscular tissue is converted into adipocire when kept in water for some time; and this artificial and extraneous

---

* Disintegration of muscular fibres (of heart), showing the entire absence of transverse striæ, unaccompanied by the substitution of oil-particles—thus contrasting with fatty degeneration. This transformation resulted from mal-nutrition, induced by dense thickening of the cardiac reflexion of the pericardium consequent on pericarditis. Sudden death while turning in bed. Royal Free Hospital.

† Med.-Chir. Trans., vol. xxxiii. p. 140, &c.

‡ Principles of Medicine, 1856, p. 449 *et seq.*

§ Surgical Pathology, 1853, vol. i.

‖ Principles and Practice of Medicine.

¶ Archiv, B. i. p. 167.

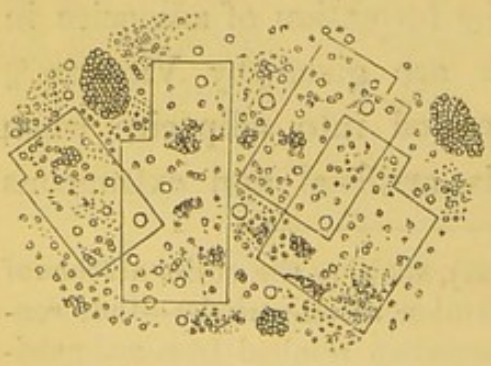
production of fatty matter in muscular tissue was originally adduced by Dr. Quain, and since by the above-named pathologists and others, in triumphant confirmation of the chemical theory of fatty degeneration, by which muscle and other textures are transformed into fatty matter within the living body.

On the other hand, fatty degeneration is apt to arise in connexion with a general increase of fat in the body; a fact adverted to by Dr. T. K. Chambers,* as a casual coincidence in the human subject; but I have establishedt his connexion in over-fed cattle ;† thereby exposing the fallacy of the Principle of the English system of fattening cattle, both as regards quality of *breed* and *nutritive* value of cattle for human consumption; this latter aspect of the subject representing its direct relation to Public Hygiene, coextensive with the question of animal-food supply, and its nutritive value.

The blood-vessels are prone to fatty deneneration, which is then known by the name of atheroma.

The larger arteries are not unfrequently beset with flattened and slightly elevated patches on their inner surface, but underneath the thin lining membrane. This atheromatous matter is yellowish, opaque, of cheesy consistence, yet friable; and it consists of oil-particles with crystals of cholesterine (fig. 33)‡ (Gulliver), which partially occupy the place of the middle-muscular coat. Eventually, a further deposit ensues, consisting of mineral salts — probably phosphate and carbonate of lime, by which the atheromatous matter becomes hard and brittle, and the patches assume the character of calcareous or bony plates, but with a very imperfect

FIG. 33.



* On Corpulence, 1850, p. 120-1.

† Evil Results of Over-feeding Cattle. 1857. A New Pathological Inquiry.

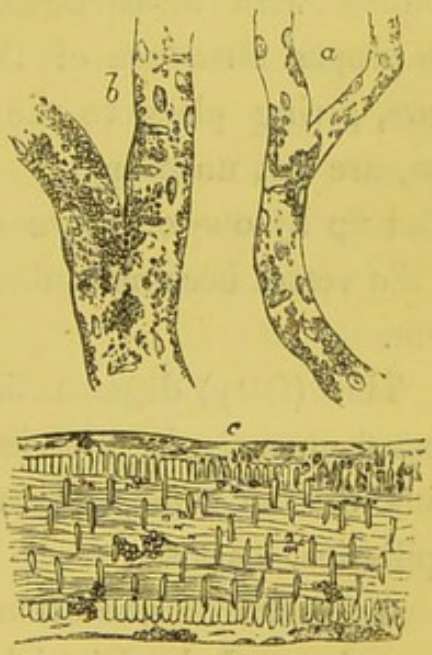
‡ Fatty particles, oil-drops, and granule-cells, with crystals of cholesterine; from broken down atheroma of an artery. (*Bennett.*)

imitation only of the structure of bone. The inner lining membrane may then give way, and the degenerate portion of artery having lost its elasticity, is disposed to yield to the pulsating throbs of the arterial wave-current, and expand into an aneurism. This series of degenerate transformations is especially apt to occur in some portion of the arch of the aorta; and in one remarkable case of aneurism of the transverse aorta, which I dissected for my colleague, Dr. Cockle,* a no less remarkable condition coexisted:—the whole thoracic aorta was converted into a continued series of bony plates, forming a bony tube, which condition terminated abruptly at the aortic aperture in the diaphragm.

The histological character of fatty and calcareous degeneration of the larger arteries was discovered by Mr. Gulliver;† but the smaller blood-vessels, arteries, veins, and capillaries are also liable to a certain transformation, of which two opposite views are held.

The smaller blood-vessels (fig. 34)‡ may become studded with fatty granules, at first thinly and irregularly scattered, subsequently more thick-set; or, the oil-particles may aggregate into patches, sometimes oval or round, generally of an irregular shape; and these aggregated granules are apt to coalesce into drops of oil.

Fig. 34.



Now the moot question is, are these granules outside the vessels, or in the substance of their walls? Patches of granules were described and figured by Dr. J. H. Bennett§ in 1842, and attributed to fatty

* Case read before Med. Soc. Lond., 1862.

† Med.-Chir. Trans., vol. xxvi. p. 86.

‡ Fatty degeneration of blood-vessels, illustrated by the cerebral vessels of an aged individual who died of apoplexy. *a*, ultimate capillary; *b*, larger vessel; *c*, small artery, with fatty particles scattered over its surface. (*Wedl.*)

§ Edin. Med. and Surg. Journal, vols. lviii. and lix.

degeneration of exudation-matter, thrown out from the vessel. In 1849, Mr. Paget* also described the same appearances, but attributed them to fatty degeneration of the vessels themselves. These latter observations referred to the smallest cerebral vessels, in connection with apoplexy, and softening of the brain, respectively. Paget describes the fatty granules as being situated "beneath the outer surface" of the vessels. That in small arteries of 1.500th of an inch in diameter, the granules are formed in the more or less developed muscular or transversely fibrous coat; in veins, the corresponding layer, immediately within their external fibro-cellular nucleated coat; in vessels, whether arteries or veins, whose walls consist of only a simple pellucid membrane, bearing nuclei, the substance of this membrane is the first seat of the fat deposit. As this degeneration proceeds, the portion of vessel thus affected undergoes changes of structure, the nuclei and fibres disappearing, until at length blood-vessels of 1.150th of an inch in diameter appear like tubes of homogeneous pellucid membrane, studded with oil-particles, in its substance. By this change, the proper structure of the vessels wastes, and eventually disappears, giving place to the fatty granules. Alterations of shape, also, are not uncommon. Usually, the outer layer of the wall is lifted up by one or more clusters of oil-particles, and the outline of the vessel becomes tuberous. Sometimes aneurismal dilatations form.

This (fatty) degeneration of the smaller blood-vessels occurs most frequently in arteries of 1.300th of an inch in diameter; veins of the same size, or smaller, are next in order liable; and capillaries, least so.

A similar degeneration of the blood-vessels of the lungs has been observed by Dittrich, and in branches of the pulmonary artery, accompanied with tuberculous disease of the lung, by Dr. R. Hall.† The placental blood-vessels may undergo a similar

---

* Medical Gazette, vol. xiv.

† Med.-Chir. Rev., October, 1855.

transformation (Paget); and those of the eye, in connexion with arcus senilis.

Fatty degeneration of the cerebro-spinal substance, and of nerve-fibres, is questionable. The brain or spinal cord may liquefy or soften, partially, or throughout its whole substance, with breaking up of the nerve-fibres, and the production of abundant granule-cells or masses, and free-floating granules; but these changes are collectively denominated by Paget "liquefactive degeneration:" and this arises from obstructed circulation through the substance of the organ, or from abrogation of its function. Atrophy of nerve-fibres is thus described by Waller:—At first transverse lines appear in the intratubular substance, indicating its loss of continuity; then it is apparently divided into round or oblong coagulated masses, as if its two component materials were mingled; then they are converted into black granules—resisting the action of acids and alkalies; and finally, these granules are slowly and imperfectly eliminated.

Besides fatty degeneration of textures, the healthy integrity of which is most essential to life, this transformation may occur in perhaps any other texture. In the air-vesicles of the lungs, and in the bronchi (Bennett);* in the cells of the liver—'hepatic cells'—constituting fatty degeneration of that organ; in the shut sacs of vascular glands, as those of the spleen (Bennett); in the pancreas (C. J. B. Williams);† in the kidneys, as first shown by Bowman; and generally in the ducts of all glands which are lined with epithelium. The stomach, intestine, and urinary bladder, are probably not exempt; their fatty degeneration respectively being a further illustration of this process, chiefly as regards organic muscular fibres. By a similar transformation of the uterine substance after parturition, this organ regains its former dimensions, or nearly so, and re-adjusts itself—a subject which I shall have occasion to notice more particularly, as illus-

* Op. cit., p. 234.

† Principles of Medicine, p. 448.

trative of the Restorative Power, in a subsequent chapter of this work. The placenta is liable to fatty degeneration—so named by Dr. Barnes, and originally figured by Dr. J. H. Bennett, in 1844,* who regards the fatty molecules and granule-cells, not as produced by a transformation of the placental tissue, but of exudation-matter, or by that of extravasated blood.

"The yellowish or fawn-coloured deposits may be infiltrated throughout the tissue of the placenta over a greater or less space, or they may occur in isolated spots forming nodules. They are generally somewhat indurated, and give rise to the idea that they are coagulated fibrin. I (Dr. Bennett) have frequently examined them, and traced all the changes intermediate between a coagulated exudation, or extravasation of blood, and the ultimate conversion of the foreign matter into a mass of molecules filling up the intervascular spaces. Similar observations have been more recently made by Drs. Handfield Jones† and Cowan.‡ In many cases the fatty material may be seen forming a layer separate from the vessel, and inside the limitary membrane of the villus."§

Bones are liable to fatty degeneration of their texture; a condition corresponding to the mollities ossium of most English authors. The characteristic properties of the osseous texture in this state are—its softness and brittleness, pale yellow colour, oily greasiness, and lightness; the texture being that of a spongy bone deprived of its earth, and soaked in soft fat, while the original size and shape of the bone remain unaltered. Hence the name—Eccentric Atrophy of Bone—proposed by Mr. Curling, as suggesting one of the most striking characters of this transformation. I have elsewhere denominated it "fatty disintegration,"‖ in describing the cancellated portion of an os calcis.

---

* Treatise on Inflammation, plate, fig. 10.
† British and Foreign Med.-Chir. Rev., vol. ii. p. 354.
‡ Edin. Med. and Surg. Journal, April, 1854.
§ Principles and Practice of Medicine, 1859, p. 238.
‖ Lancet, 1858, Nov. 20.

Oil, not fat-cells, fills the cancelli, and is even found in the lacunæ and canaliculi (Virchow). Combined with this oil, Bennett describes numerous cells, each of which varies in size from $\frac{1}{1200}$ to $\frac{1}{500}$ of an inch in diameter, and contains a round nucleus, also varying much in size, and occasionally showing various stages of division and of endogenous development. The production of this cell, as in most of the so-called fatty *degenerations* of texture, is attributed to an exudation from the blood-vessels, mingled with more or less of the coloured corpuscles, in which new cells are developed, combined with fatty transformations of the albuminous and fibrinous materials; thus differing from the structural condition of bone, known as "rachitis," which, although also accompanied with softening, is regarded as arrested development of bone, with increased growth of cartilage-cells (Kölliker).

Cartilage may undergo fatty degeneration; oil-particles, and by coalescence, oil-drops, substituting the contents of the cartilage-cells; during which transformation the nucleus vanishes. Oil-particles may likewise beset the intercellular hyaline basis; which, together with the cell-contents, acquire a marked opacity.

"Arcus senilis" consists of oil-particles in the substance of the cornea, near the iris, forming a marginal ring round it. This fatty degeneration was discovered and originally described by Mr. E. Canton.* Its significance extends far beyond the organ of vision, whose function remains unimpaired; for, unless consequent on inflammation of the eyeball, arcus senilis is the pathologico-anatomical indication of many other and most important coexisting fatty degenerations,—of the ophthalmic artery, of the muscles attached to the eyeball, of the heart; and in fact, a degenerative tendency throughout the body. Arcus senilis is the surest external and visible sign, during life, of internal and unseen fatty and other degenerate conditions of organs, discovered after death. Arcus senilis was present in about $\frac{9}{10}$ of the cases in which Dr. C. J. B. Williams had reason to infer the existence of fatty

* "Observations on Arcus Senilis," Lancet, 1850-51.

degeneration of the heart. Lastly, although commonly seen, as the term implies, only in advanced life, arcus senilis in earlier years is, apart from local disease of the eyeball, a sure indication of premature old age.

The lens may also be transformed into fat (Dalrymple, Lebert), constituting soft cataract.

New Products—*i.e.*, false tissues, deposits or exudations, and growths—are by no means exempt. A pleuritic false membrane, for example, may become converted into fatty matter. Of deposits, pus-cells are specially prone to fatty transformation of their contents, forming granule-cells; tubercle-corpuscles, in the same way, disintegrate, this change being denominated the 'softening' of tubercle. Among growths, cancer is apt to become studded throughout its substance with oil-particles, free, and *in* the cells, which, by this substitution of their contents, forming granule-cells, are no longer distinguishable as "cancer-cells." Eventually the cell-walls disappear, and the whole or portion of the growth which has undergone this degenerative transformation becomes a confused mass of fatty matter, with the remnants of cells. It is worthy of note, that the fatty matter may appear in yellow masses, not unlike softened or yellow tubercle, and that this resemblance explains some instances of the so-called coexisting admixture of tubercle with cancer.

'Pigmentary' is akin to fatty degeneration; the material deposited in either case being *essentially* carbonaceous; in the one, oil-particles or drops; in the other, pigment-particles or granule-masses of pigment: but the origin of this pigment-matter is different in different cases; and its pathological significance is therefore equally diverse.

Pigment-matter may represent the colouring matter of the blood, or of the bile; and these being distinct sources, refer to morbid conditions of origin, as distinct; but the particular colour of the pigment-deposit—commonly black, sometimes red, brown, green, or yellow, does not enable us to determine with precision the question of origin.

Carbonaceous matter introduced extraneously into the body, and thus deposited in the textures, is obviously not pigmentary degeneration. Of this kind is the black matter found in the lungs and bronchial glands of colliers and others who habitually inhale smoke. The colour of this matter is not discharged by hydrochloric acid or by chlorine; a point of distinction between it and the pigment of morbid origin, which disappears under the influence of these chemical agents.

Pigmentary degeneration occurs frequently in the mucous membrane of the stomach and intestines of old persons, and is recognised by slate-coloured discoloration of this texture. So also in cases of dysentery. Of new tissues, intestinal cicatrices not unfrequently present the same appearance. Deposits of tubercle eventually become surrounded with black pigment-matter. New growths may be infiltrated with this matter, as commonly occurs in encephaloid cancer, or pigment may be deposited by itself, and accumulate in the form of a distinct tumour, known as melanosis,—a new growth, falsely so called.

'Fibrous' degeneration follows next in order; for although, structurally and chemically, a minor degree of transformation than the fatty, yet it will, I am inclined to believe, prove to be an evolution of one structural constituent of the transformed texture —the fibrous element being evolved—at the expense of another constituent, which itself has undergone degeneration and disappeared; leaving its surviving companion more conspicuous, and the representative of the originally compound texture transformed as if entirely by degeneration.

Muscles are the chief seat of this fibrous transformation, and especially the voluntary muscular texture. In the course of that general and natural wasting of the body which denotes old age, the voluntary muscles especially become paler and more fibrous, as seen after death; though softer, because less contractile, during life. This senile atrophy is characterized by wasting of the muscular fibres, while the interstitial fibro-cellular tissue consequently becomes more apparent, forming a condensed sinewy

texture, instead of muscle. As felt during life, the tendons seem to have encroached upon and substituted the muscles. By this shrinking, and by that consequent on the disappearance of subcutaneous fat, the rounded outline of youth is lost in the shrunk shanks of the lean and slippered pantaloon.

Apart from this textural transformation incident to age, it is questionable whether "fibrous degeneration" occurs otherwise than by virtue of interstitial exudation-matter. The muscular texture is still a chosen seat. Of voluntary muscles, those of the limbs may acquire a fibrous condition, as the result of chronic fascial rheumatism; the intercostal muscles and diaphragm also, in chronic pleurisy; while fibrous degeneration of organic muscular texture is exemplified by that of the heart after endocarditis or pericarditis (C. J. B. Williams). Membranous textures likewise are peculiarly liable to this change—apparently by virtue of interstitial exudation-matter. Hence, probably, the opaque white thickenings of serous membranes which are seen as patches in the cardiac reflection of the pericardium, in the valves of the heart, in the pleura, in the peritoneum, especially over the liver, and in the arachnoid. Less obviously the result of interstitial exudation is that fibrillation of cartilage by which, according to the observations of Redfern, an ulcer of articular cartilage heals. Ulceration having destroyed a portion of any such cartilage, through softening of the intercellular substance and the release of the corpuscles, which also very often disintegrate and discharge their contents—thereby completing the ejection of all the elemental structures of cartilage from the ulcerating surface;—then the process of repair and cicatrization consists "in the transformation of the intercellular substance, and the nuclei of the cells, of the adjacent cartilage, respectively, into the white and yellow tissue of the fibrous cicatrix."* Thus, this observer concludes, "ulcers in articular cartilages heal by transformation of the surrounding

---

* Monthly Journal of Medical Science, 1851.

cartilage-tissue into fibre; but those occurring in other textures are cured by the formation of a cicatrix out of newly exuded blood-plasma."

Other accredited examples of "fibrous degeneration" are obviously indurations by interstitial fibrinous effusions, as in the (interstitial) cellular tissue of parenchymatous organs—the liver, kidneys, spleen or lungs—constituting cirrhosis; or in the submucous or subcutaneous cellular texture; the latter, so-called degeneration, being that induration of this tissue which is occasionally witnessed in a newly-born infant.

Fibrous degeneration, or induration of parenchymatous organs, takes place at the expense of their proper structure, which concurrently atrophies, and partially disappears.

'Amyloid,' waxy or lardaceous degeneration, is probably allied to, if not a variety of, "fibrous degeneration." The texture affected acquires considerable firmness and density, is somewhat transparent, and of a yellow or yellowish brown colour, which in many cases contrasts with the colour of the original texture. The cut surface is not unlike that of firm bacon. Thus, the liver, the kidney, or the spleen, having acquired these characters, is then said to have undergone waxy degeneration. The essential structure of the texture or organ atrophies;—the secreting cells of the liver or kidney, and its Malpighian bodies, wither; at the same time these cells present a remarkably pale and transparent appearance, as they become insignificant. The corpuscles of the spleen, both in its parenchyma and those in its malpighian bodies, shrivel, and have also a similar appearance.

The waxy character of amyloid degeneration is apparently due to interstitial exudation-matter which has undergone transformation. This material is unorganized, and belongs to the albuminoid group of organic chemical compounds; but its precise composition and chemical relations are unknown. It assumes a blue colour by the action of iodine; and this fact, first observed by Virchow,* led

* Archiv f. Path. Anat., Bd. vi. s. 135, 268 and 416.

him to conclude that the waxy material in question was similar to starch, and therefore hydro-carbonaceous. He accordingly named it "animal amyloid." Meckel regards the matter as fatty, and especially cholesterine; he therefore retains the name, lardaceous or cholesterine disease: but Friedreich and Kekulé* have ascertained that the composition of the purest amyloid matter obtained from the spleen closely resembles that of the albuminous principles; and this conclusion is confirmed by the independent analysis of Schmidt.† Yet the reaction of the waxy material being unlike that of albuminous matter, the material itself is so far peculiar.

Waxy and fatty degenerations are frequently combined, as in the liver and kidney respectively; but a series of analyses, chiefly by Dr. Drummond, and collected by Gairdner,‡ has elicited this important general result:—that the human liver in the state of waxy degeneration contains considerably less fat and water, and a greater amount of solid constituents, than in the healthy state.

'Granular' degeneration is manifested by an albuminous matter —granular, *i.e.* structureless, and, it is said, *deposited* interstitially; but superseding the proper structural elements of the texture, which atrophy and disappear. Thence the original texture becomes converted into a granular mass. With this degenerative transformation, the texture loses its cohesion, and also acquires an opaque yellowish colour. It is apparently identical with what Bennett denominates "molecular albumen," and a variety of "albuminous degeneration." I am inclined to regard this degeneration as being, more correctly speaking, a 'disintegration' of the structural elements of the original texture, without the deposition of any new granular or molecular albuminous matter. It chiefly affects the fibrous textures, or fibrous new formations; either of which by their disintegration would yield this kind of

---

* Archiv f. Path. Anat., Bd. xvi. s. 50.

† Annal. d. Chemie und Pharmacie, Bd. cx. s. 250.

‡ Monthly Journal of Medical Science, May, 1854.

matter, and present the physical characters of yellowish opacity and want of cohesion, already noticed.

Lastly, 'calcareous' degeneration—the lowest form of all—approximates organized textures to unorganized concretions. The new matter consists of phosphate of lime and magnesia, and carbonate of lime. This matter also is said to be *deposited* interstitially in the original texture, the structural elements of which, having atrophied proportionately, are gradually superseded; thus eventually completing that substitution of texture which constitutes the degenerative transformation. Rokitansky regards calcareous degeneration as a chemical precipitation of the apparently new mineral matters, from their natural combinations with the animal matters of the texture. This view is *analogous* to what I have denominated disintegration of organized matter; and still more so, to degeneration by absorption of one structural constituent of a compound texture, and thereby the evolution of another such constituent.

Calcareous degeneration takes place in tissues of simple organization and of low vitality in respect of blood-supply and nutrition. For example, in fibrous textures, in cartilage and fibro-cartilage, and in bone; the mineral transformations of these textures being, collectively, that natural petrifaction whereby, as age advances, the body is slowly yet surely reduced to its primitive earth. But this degeneration is not unfrequently secondary to some other; commonly consequent on the fatty transformation, and of the arteries, principally. Atheroma in the coats of these vessels, or in the valves of the heart, is followed by calcareous degeneration, so that both become associated in the same portion of texture. Calcareous degeneration of nervous texture is referred to by Bennett, but as a rare occurrence in man. Of deposits, tubercle is most liable to calcify, as in the pulmonary texture, and the tuberculous matter deposited in the bronchial and lymphatic glands. Bennett states,* that he possesses specimens

* Op. cit., p. 254.

of miliary as well as of infiltrated tubercle, arrested in all stages of their progress, by cretaceous transformation; in which case, on microscopic examination, it is seen to consist of mineral masses, associated with a few tubercle-corpuscles, débris of the tissue in which it occurs, and occasionally a few crystals of cholesterine. This change would seem to arise from absorption of the animal (albuminous) matter of the deposit, and precipitation thereby of the associated mineral constituents. The same transformation may occur in exudations on serous membranes, forming osseous-like or calcareous plates, as on and in the pericardium, and in parenchymatous organs, forming calcareous concretions. Morbid growths of all kinds are liable, and some are prone, to calcification; fatty tumour, sometimes fibrous, cystic, and cartilaginous tumour, very commonly; cancer, not unfrequently. And in all cases this degenerative transformation arrests the further progress of growth, and is a natural mode of cure.

Regarding Degeneration, of whatever kind, as a *process*—resulting in the retrogression of the structural elements of textures and organs; this process may be conducted in apparently *three* different ways, representing as many modes or varieties of degeneration.

Retrogression of textural structure may issue from the 'deposition' of new matter, which *directly* substitutes the proper structural elements of the texture, undergoing degeneration; they having concurrently disappeared: or, degeneration may proceed, apparently from 'chemical' transformation of the proper structural elements, or again, from similar transformation of exudation-matter, *interstitially* deposited. These modes of degeneration are illustrated by fatty degeneration. Pigmentary degeneration, properly so called, I have restricted to the deposition of pigment-matter from the blood or from the bile. Fibrous degeneration, in its typical form, I have regarded as the 'evolution' of fibrous tissue, by the concurrent atrophy and disappearance of another and more essential structural constituent of a compound texture; as in muscle, the muscular fibres disappearing, makes conspicuous

or evolves their interstitial connective tissue, which becoming condensed, readily assumes the character of fibrous tissue. The actual conversion of muscle into fibrous tissue, is at present the received view of fibrous degeneration. Obviously, however, this change cannot be effected by conversion of the muscular fibres,—for they have partially or entirely disappeared; but it may be by the more or less complete substitution of *interstitial* exudation-matter, which assumes the fibrous character. This is the acknowledged mode of amyloid degeneration—the new matter *interstitially* deposited being chemically allied to that of fibrous degeneration in having an albuminous nature, while structurally it differs in being granular or structureless. Granular degeneration is also regarded as another transformation by the deposition of new matter *interstitially*, this being another variety of albuminous matter, which gradually usurps the place and substitutes the structural elements of the original texture, they disappearing, and thus completing this degenerative transformation. I have regarded it as a process of Disintegration of the said structural elements, without the deposition of any new matter; and that the granular matter is formed chiefly by the disintegration of textures originally fibrous, or by that of fibrous new formations. Calcareous degeneration is an analogous process; being either a chemical precipitation of saline matters from their associated albuminous matters: or, possibly the deposition of new calcareous matter, *interstitially*; the concurrent absorption and disappearance of the albuminous matter completing this transformation.

These Transformations—degenerative and disintegrative—of textures—original and adventitious—are shown by the following table:—

RETROGRESSION OF THE STRUCTURAL ELEMENTS OF TEXTURES.

1. By the deposition of new matter—

   a. Directly substituting the original structural elements, which have themselves disappeared.

   Fatty and Pigmentary degenerations, respectively.

b. Interstitially deposited, and accompanied with the absorption and disappearance of these elements.
Fibrous, including Amyloid or waxy degeneration.
Granular degeneration.
Calcareous degeneration.

2. By chemical transformation—
a. Of the original structural elements.
Fatty degeneration of muscle. (Dr. R. Quain.)
b. Of interstitial exudation-matter.
Fatty degeneration, in many cases. (Dr. J. H. Bennett.)

3. By atrophy of one structural constituent of a compound texture, and the evolution thereby of another constituent. (The author.)
Fibrous degeneration of muscle. (The author.)

4. By disintegration— (The author.)
a. Of the original structural elements.
Granular degeneration, so named, of fibrous textures.
b. Of interstitial exudation-matter.
Granular degeneration, so named, of fibrous new formations.
By chemical precipitation, analogue of disintegration.
Calcareous degeneration, so named. (Rokitansky.)

Retrogression of textural structure, by Degeneration—morbid, only when it occurs prematurely—represents, as I have said, the relapse or falling back from a higher condition, to a lower or more elementary grade of textural structure; and which substitutes the proper constituents of the original texture. This *relapse* and *substitution* are the essential characteristics of Degeneration.

The Products of nutrition—morbid, so-called—are *supplemental:* either as supplying the place and purpose of the original textures—such are false tissues; or as being superadded to the original textures—such are deposits and new growths.

Now, the question is, are these false tissues, deposits, and growths—collectively speaking, these "plastic products"—morbid because they are supplemental or adventitious to the original and healthy tissues, with which they are associated; or are they, by virtue of their structural elements, merely rudimentary stages of analogous healthy tissues; and therefore that these *apparently new* products represent only *healthy* tissues, in various stages of *their* development? That, in fact, "false tissues," "deposits," and "new growths," representing only so many developmental conditions of analogous healthy tissues, are severally and collectively formed by as many *arrests* of *their* development; just as 'degenerative' retrogressions of the original tissues are formed by so many *relapses* to as many more simple, if not developmental, conditions of their structural elements.

This law would be a further and complete extension of Structural Retrogression—to interpret the (minute) structure of textures, morbid so called; and would thus far indicate the relation of Physiological to Pathological Anatomy.

Does then the Structural Retrogression of *healthy* tissues, by *arrest* of their development, explain the formation of so-called *new* plastic products, or the origin, locally speaking, of false tissues, deposits, and growths?

We must approach this question by first ascertaining the *essential* structural elements of Morbid Products and Healthy Tissues, respectively. This will show the similarity or difference of their structural elements.

On reviewing *False tissues*, we perceive that their representative type, connective tissue, consists of two structural elements—namely, certain *cells* and *imperfect fibres* produced therefrom, and resembling the young white fibres of ordinary cellular tissue. This fibro-cellular structure may be variously fashioned off and finished in resemblance of healthy tissue; hence the term false or analogous tissues, applied to this class of plastic products.

Other products are purely cellular, with granular matter

intermixed. *Deposits* of pus and tubercle—miliary and yellow tubercle—alike consist of cells and *granules*.

Other products, again—*Growths*—consist of certain cells and fibres; the latter being of two kinds—namely, the white fibre already mentioned, and the nuclear fibre (of Henle) which is also found in healthy cellular tissue. Thus, cells, like those of either young cartilage or of epithelium, are the essential structural element of cancer-growths. These cells are found infiltrated amidst the natural fibrous tissue of the part affected; but they abound most in encephaloid cancer, less in colloid, and least in the scirrhous variety. The cell-element also prevails in cartilaginous, cystic, and fatty growths, and in their respective varieties; whilst fibres predominate in other growths, the white fibre in the fibrous tumour, and its recurring fibroid variety; the same fibre, with free oval nuclei, in the fibro-nucleated variety.

It would therefore appear that all plastic morbid products may be reduced to three kinds of structural elements: *cells*, *fibre* of two kinds, and *granules*.

Passing on to Healthy Tissues, what structural elements do they present under the microscope? Certain *cells* are the essential element of blood (lymph and chyle) epithelium (and cuticle), also of cartilage and fat; while nerve and muscular fibres are tubules, formed most probably by the coalescence of a longitudinal series of cells, the partitions of which are then absorbed, and the nerve-matter or fibrillæ respectively developed in their place (Schwann). Two kinds of *fibre*, the white and the yellow, together form cellular tissue, the most abundant material in the body; the former fibre predominates also in the fibrous and fibro-cartilaginous tissues, and is, moreover, the essential structural element of the osseous lamellæ; the nuclear fibre is essential to elastic tissue; while both kinds of fibre are blended and intermixed in the tunics of the blood-vessels and lymphatics, in the serous, synovial, and mucous membranes, and in the skin.

Associated with these—the fibres of cellular tissue—is sometimes found a peculiar delicate *membrane*,—clear, colourless, and

apparently structureless. This primitive membrane may be seen in the blood-vessels ("fenestrated membrane" of Henle), also in the lymphatics, and constitutes the "basement membrane" beneath the epithelial layer of the above-mentioned membranous tissues. The same structureless membrane forms the envelope of cells and tubules in those tissues which consist essentially of these elements.

A few words will suffice, for our present purpose, to complete this brief survey of healthy tissues. A cell presents certain general features :—a delicate, thin, transparent, colourless, vesicular film, without structure, enclosing contents of various quality and quantity, and with or without one or more cell-germs amongst them. By peculiarities of size, shape, and the contents of cells, cell-tissues may be readily distinguished under the microscope; but I need not enter on details with which modern surgeons are familiar. The two kinds of fibre to which I have so often referred contrast remarkably, and are the well-known fibres of cellular tissue. Structureless membrane has been sufficiently noticed. Lastly, with these elements, more or less *granular* matter is associated, some of which consists of particles, having each a well-defined outline, varying in size from $\frac{1}{12000}$ to $\frac{1}{6000}$ of an inch in diameter, and are apparently minute vesicles of oil (elementary granules of Henle); whilst other particles are altogether shapeless (amorphous matter), and probably the *detritus* of waste tissues.

From structural elements thus simple—as GRANULES, HOMOGENEOUS MEMBRANE, CELLS, and FIBRE of two kinds—are fabricated the foregoing fifteen healthy tissues, and plastic morbid products also. Nature is, as ever, sparing in means, but fruitful in ends. So far we must admit the identity of morbid growths, deposits, false and healthy tissues, as to acknowledge their common origin. It remains to be seen whether, in passing through their successive stages to maturity, the cells and fibres of healthy tissues assume the characters of the aforesaid morbid products. If so, these products represent healthy tissues in various rudimentary stages of their development; and the law of structural retrogression is completely established.

Let a portion of cuticle be removed, and the subjacent surface of true skin gently sponged dry; a clear colourless fluid will soon be observed to again moisten the surface. Under the microscope this fluid will be found altogether structureless, but abounding more or less in granular matter. This fluid represents the "liquor sanguinis," and may be obtained from all other parts of the body —from other free surfaces besides the skin, and from the interstices of tissues, although then, of course, adulterated by the admixture of extraneous matter. Out of this fluid all the tissues are formed, and a few words will suffice to sketch an outline of the plastic process by which they are wrought. The results of this process will be seen to represent the various healthy tissues, and their several rudimentary stages of development will be observed to correspond with so-called *new* or morbid plastic products. While therefore we briefly trace the successive rudimentary stages of healthy tissues in the process of their development, we shall also perceive the structural correspondence of morbid products to the *transitional* conditions of their analogous healthy structures.

Thus, to begin: the elementary granules aggregate together and form compound particles of larger size—*nuclei* (Schwann and Schleiden), whose component granules are then named *nucleoli*. A nucleus having formed, may itself give rise to a cell; hence its name sometimes—*cell-germ* or *cytoblast;* while the clear fluid from which it was derived is appropriately named cytoblastema, or shortly, blastema.

This blastema undergoes further changes, which are properly designated plastic for they issue in that definite, although varied, arrangement of matter denominated organization. The formation of a nucleus by the aggregation or attraction of elementary granules is itself a primary plastic change; and by an analogous attraction of granules into a mass, which is then enclosed by a membranous envelope, a *cell* is produced.

Or a cell may result from the aggregation of matter in the form of a membrane being thrown around a previous cell, which encloses it as the nucleus of a secondary cell.

Again, instead of an attractive force, the repulsion of particles may come into operation. Indeed, the two opposing forces of molecular attraction and repulsion apparently co-operate in cell-formation; but as one or other force preponderates, so are cells produced in one way or the other. Thus—by the repulsion, as it were, of particles from the surface of a nucleus, together with their laminar cohesion, a cell may sprout therefrom, like a soap-bubble rises.

The nucleus, already within a cell, may loosen by repulsion of its elementary granules, each of which may then become surrounded by a membrane. If originally vesicular, they expand. In fact, minute spherules may thus enlarge by a kind of centrifugal expansion.

Lastly—to complete the series of cell-geneses—the force of repulsive distension is further manifested occasionally by the outgrowing or sprouting of cells.

These various modes of cell-formation will be readily recognised by those who are familiar with histological anatomy.

Cells may be generated in blastema by any one of these modes of origin; but some of the methods I have described are of more common occurrence. Granules, for example, may collect around a nucleus—itself consisting of nucleoli—and the whole mass then become enclosed within a vesicular membranous envelope.

By this process, apparently, the pale blood-corpuscles are evolved, and also the corpuscles of lymph and chyle, which are nearly identical. The red blood-corpuscles are probably derived from them by the loss of their nuclei, the cells becoming flattened, and their contents assuming a reddish tint.

Should, however, the pale corpuscles retain their *transitional* condition—that of aggregated particles, without presenting a distinct nucleus, and with or without an enclosing membrane,—then we recognise the granular exudation-cells of inflammatory deposits. Exudation-cells are liable to a partial liquefaction of their contents, by which a central molecular nucleus becomes apparent; and thus are formed the "many-nucleated corpuscles" of pus (Paget). Very

similar to exudation and pus-cells are those of tubercle, when seen in their highest stage of development, as in semi-transparent miliary tubercle. These cells may, however, undergo a retrogressive change of structure, and by their resolution into mere granular matter (mixed with the remains of cells), present the appearance of the soft opaque variety of tubercle. Lastly, the red blood-corpuscles are liable to a degenerative change, the exact nature of which has not yet been traced; and by this metamorphosis is produced the black matter of melanosis, either liquid or solid, and free or mixed with the materials of other growths—as, for example, in melanotic cancer. Morbid deposits are therefore, apparently, developmental conditions of the structural elements of analogous healthy tissues—by arrests of their development.

By another mode of cell-formation, the attraction of matter around a nucleated cell, which then itself officiates as a nucleus, may probably be explained the formation of "nerve-cells;" such as abound in the grey portion of the cerebro-spinal axis, and even more abundantly in nerve-ganglia;—a circumstance which suggested their more usual name—"ganglionic corpuscles." But I am not prepared at present to specify any forms of morbid cells as developmental conditions of these corpuscles.

The production of certain healthy cells—as epithelial and cartilage-cells—is in some way or other determined by the presence of nuclei; and although the precise nature of the process is unknown, yet it is certain that cancer-cells closely resemble the *rudimentary* cells of healthy cartilage. Both present a similar pellucid, colourless envelope, enclosing a large, clear bright nucleus or two at least, and sometimes more; with one or two nuclei, also large and clear, imbedded in the substance of each. Cancer-cells sometimes resemble certain epidermic cells. It is, I conceive, owing to this twofold relationship of the cancer-cell, that Dr. Hughes Bennett, in defining this cell, limits his definition to localities unconnected with either a mucous or epidermic surface, and nowhere surrounded by a hyaline or fibro-hyaline substance—the intercellular basis of cartilage.

The cells of cartilaginous tumours (enchondroma), with those also of their myeloid variety, resemble the cells just mentioned, and appear only retrogressions to various *rudimentary* conditions of healthy cartilage-cells by arrest of their development.

The cells of fatty tumours very nearly resemble those of ordinary adipose tissue; and the cells of cystic tumours are probably produced within parent cells, and this endogenous mode of origin corresponds with a well-known method of *healthy* cell-formation. Cystic growths are, therefore, so far related to healthy organization. But the recent progress of Pathology goes further, and points to cystic growths as another illustration of the law of structural retrogression. It is here that I would notice the tendency of other writers *towards* the recognition of this law.

Mr. Simon regards cysts of the kidney "as vesicular transformations of the ultimate structure of the gland;" and to this view—adds Mr. Paget, without adopting some ingenious suppositions which he has connected with it—I would adhere: "For, unless a cyst or a solid tumour (assuming this mode of their origin to be correct) were really a transformation of a nucleus, or a cell, of the *part in which it grows*, we could not understand the very general similarity that we find between the contents of certain cysts, and the secretions or structures of the glands in or near which they occur, nor yet the likeness which commonly exists between *the solid tumour and the tissue in which it is imbedded*. These things are as if the first beginning of the abnormal growth were in some *detached element of the natural tissue*, which element being perverted from its normal course, thenceforth multiplies and grows, conforming with the type in minute structure and composition, but more and more widely deviating from it in shape and size. Such are the facts, and such the speculations that we may entertain, respecting the origin, or at least the smallest visible beginning, of a cyst, or of an innocent solid tumour."*

* Surgical Pathology, vol. ii.

I have placed in *italics* those portions of this paragraph which pertain more especially to our present inquiry. The identity in point of structural elements, by the growth of which innocent solid tumours and their analogous healthy tissues are alike produced, is clearly recognised. But the law of structural retrogression by arrest of the development of these elements, and its application to explain the (apparently peculiar) structure of the tumours in question (not to mention other growths), is overlooked.

To further illustrate this law, I shall now briefly notice the formation of healthy *fibres*, and trace the relation of their development to the production of fibrous growths and false tissues. Fibrous tubules (hollow fibres) are formed by the coalescence of cells, whose sides are then absorbed. But the fibres referred to are not hollow, and result from the metamorphosis of *single* cells. A cell elongates and assumes a spindle-shaped appearance; at length becoming more and more elongated, its opposite extremities each splits into a brush of several fibres, and by a further extension of their longitudinal division, a bundle of separate fibres is produced. The *white* fibres of the cellular and analogous tissues are probably formed by this process (Schwann); and these fibres, in their more or less rudimentary conditions, constitute fibro-cellular tumours and painful subcutaneous tubercle. Again, the nuclei, which are free in blastema, may undergo other changes besides officiating as cell-germs. They elongate, and having broken up into more minute particles, disappear; or, retaining their elongated shape, they undergo further development. They coalesce in a linear series, and form dotted-looking fibres, this appearance being due to the remains of nuclei. Such are the characteristic '*nuclear* fibres' of the elastic and allied tissues, and which further contrast with white fibres in their size, shape, arrangement, &c. If these nuclear fibres retain more or less their above-mentioned rudimentary conditions, then we recognise the structural elements of

fibrous tumours, and those also of their recurring-fibroid and fibro-nucleated varieties.

Thus may the law of *structural* retrogression, and by arrest of development, be traced through the various forms of 'deposit' and 'growth,' with which the Pathologist is familiar; and this law can be extended to 'false (analogous) tissues.' Connective tissue, regarded as their type, consists of *cells* passing into *fibres*, but—not attaining the condition of mature or perfect fibres—they are retrogressions of structure by arrest of development.

This law is confirmed by the *structural* kind of healthy texture with which each kind of deposit, growth, and false tissue is connected, in the various parts and organs where they are severally most liable or prone to occur. I therefore omitted their *situation* and *distribution* in describing these various forms of morbid products, and reserved this aspect of their pathological anatomy until now, that I might here take advantage of it to confirm the law of structural retrogression which I have advocated.

Deposits represent structural retrogression of the blood-corpuscles by arrests of their development, and which therefore retain one or other of their developmental conditions. The situation and distribution of deposits correspond with this, their origin, for they are co-extensive with the distribution of blood throughout the body. Wherever blood permeates, or the liquor sanguinis permeates by imbibition, deposits may occur. Thence the unlimited range of exudation-corpuscles and fibrin; of pus-corpuscles,—tubercle, miliary and opaque; melanic cells and pigmentary granular matter; respectively. But the particular form of deposit is regulated, not to say determined, by the elective influence of texture. For example, exudation-corpuscles together with lymph, forming adhesions, are *mostly* deposited by serous membranes; granule-cells *mostly* by the brain and spinal cord; pus-corpuscles *mostly* by mucous membranes; while exudation-corpuscles, fibrin, and pus-corpuscles, are deposited indefinitely,—by the skin, in the various forms of skin-diseases; by the cellular texture; and by the substance of parenchymatous organs.

Tubercle-deposit probably ranges as extensively, but in certain recognised situations more especially. These are merely specified by pathologists of the highest repute. Viewed, however, in relation to my theory of structural retrogression by arrest of development, and as affected by 'texture,' the question of tubercle-distribution assumes a new and most interesting character; surpassing the barren facts of purely descriptive pathological anatomy. Rokitansky takes the matter-of-fact view of this question; for, says he, "a scale of the frequency of tubercle in the various textures and organs offers but limited points of interest."* According to his experience, it would present in adults something like the following series, namely :—

Lungs.
Intestinal canal.
Lymphatic glands, more particularly the abdominal and bronchial.
Larynx.
Serous membranes, especially the peritoneal and pleural.
Pia mater.
Brain.
Spleen.
Kidneys.
Liver.
Bones and periosteum.
Uterus and tubes.
Testicles, with prostate gland and seminal vesicles.
Spinal cord.
Striated muscles.

For children this scale does not answer completely. In them the lymphatic glands, together with the spleen, would take the lead, followed by the lungs with the bronchial mucous membrane, the brain, the serous membranes, &c.

---

* Pathological Anatomy. Translated for Syd. Soc. 1854. Vol. i. p. 320.

Then again, the foregoing scale represents the relative liabilities of different textures and organs to tubercle-deposit, secondary as well as primary; both being regarded indiscriminately. Respecting, however, the primary depôts of tubercle;—"the lungs and lymphatic glands, it is true, retain their uppermost rank, but are immediately followed by tuberculoses, which stand very low in the foregoing scale—namely, of the urinary system, of the female sexual mucous membrane, of the bones, of the testicles with the prostate gland and the seminal vesicles. Meanwhile tuberculoses of the intestine, of the larynx and trachea, of the serous membranes, of the spleen and liver, take a very subordinate position in this new scale, seeing that they seldom, if ever, become the primary seat of tubercle."

"Accordingly, certain tuberculoses which in the first scale occupy a high place, possess but a very subordinate nosological import. They are seldom, if ever, primitive, but almost always secondary, dependent upon other tuberculoses, often indeed only participant in general tuberculosis. The liver, spleen, kidneys, nay, in many cases the lymphatic glands, stand in this relation to tubercle."

"Tuberculosis almost invariably attacks several determinate organs concurrently at the outset, or at a very early period. Of this communion we have examples not only in the joint tuberculosis of lymphatic glands and of the implicated organs, but also in that of the brain and of the lymphatic glands; of the testis, prostate gland, seminal vesicles, and of the urinary organs; of the spleen and suprarenal glands, and of the lymphatic glands; of uterine and tubal, and of peritoneal; of pulmonary, and of intestinal or of laryngeal tubercle."

"Secondary tuberculoses have a sort of groundwork or starting-point in certain pre-existing tuberculoses. In other words, secondary tuberculoses accede to already existing ones according to a tolerably constant rule. Thus, tuberculosis of the lungs or lymphatic glands offers for all such secondary tuberculoses a general *point de départ;* whilst, on the other hand, it commonly associates

itself to most other tuberculoses. Tuberculosis of the serous membranes accompanies that of the implicated parenchymata; tuberculosis of the urinary system, that of the genital apparatus in the male."

"It is worthy of note that in every organ, tubercle, unless thrown out with much violence, has its almost invariable and readily demonstrable point of incipiency. In the lungs, it is at the apex, the upper third of the superior lobes; in the pia mater, at the part investing the base of the brain within the common groove, running from the chiasma to the pons Varolii and the medulla oblongata or about the fossæ Sylvii; in the brain itself, in and about the gray substance; in bones, in the spongy bones or parts of bones; in intestinal mucous membranes, in that of the inferior ileum; in the laryngeal mucous membrane, at the portion covering the transversus glottidis muscle; in the testicle, in the epididymis; in the female sexual apparatus, in the mucous membrane of the tubes and uterine fundus—that the deposition of tubercle first commences, and concentrates itself."

"Again, there are a few marked limitations set to the advance of spreading tubercle. For example, tubercle of the larynx never extends to the pharynx; uterine tubercle hardly ever passes beyond the internal orifice, so that the cervex uteri and the vagina remain exempt."

These relations of certain textures to tubercle indicate an inherent and equally special formative power—inducing the deposition of tubercle in them; so also does the frequency, and more especially, the primary character of this deposit, in some textures, rather than in others: but the different kinds of textural structure in which tubercle is deposited, and the large number of such textures, indicate some other and prevailing cause of tubercle-formation. I allude to the formation of tubercle-corpuscles by the arrested development of pale blood-corpuscles, as suggested by the relative structural characters of these two forms of cells. Thence the extensive and apparently indiscriminate deposition of tubercle in so many textures, and of different structure. But this explanation is only

a further confirmation of the special structural relations of textures to their deposits—like to like; tubercle-corpuscles forming wherever pale corpuscles of the blood have aggregated, by congestive or inflammatory hyperæmia, which precedes the deposit.

Melanic cells of pigment-matter, and as free granules, together constitute melanic pigment-deposit; and this, in its typical or cellular form, represents arrested developmental conditions of red blood-corpuscles. This deposit, also, may therefore occur in very many situations, but rarely, if ever, in the human subject, unmixed either with the normal textures, or with some deposit, growth, or false tissue; the interstitial and substituting deposit being commonly regarded as pigmentary degeneration, of which the more frequent examples were mentioned in illustration of this degenerative transformation. Melanotic tumours are formed by the infiltration of the various kinds of growths with melanic pigment; of which, medullary cancer infiltrated with this matter, forming melanotic cancer, is the most common. These tumours, therefore, are not new and distinct kinds of growth.

The normal situations and distribution of *Growths* most conspicuously coincide with the law of structural retrogression—by arrested development of the analogous healthy tissues, with which they are severally connected, *in situ*. Morbid growths are morbid only as being rudimental conditions of their analogous healthy tissues. Their normal situations and distribution can almost be predicated.

Cancer is the least conformable of all growths to the law in question. This species of growth, representing as it does a structural retrogression—by the arrested development of cells—commonly like those of cartilage, infiltrated through a fibrous stroma, and, in one leading variety, a gelatinous intercellular matter, is nevertheless not commonly associated with cartilage. One variety, however—*epithelial* cancer—consisting of epithelial cells, infiltrated, is as commonly seated in or immediately under some portion of skin or mucous membrane. But here the rule is not absolute.

The three leading species of cancer—encephaloid, scirrhous, and

colloid—do severally conform in one respect to the law which regulates situation and distribution; namely, their analogous cartilage-cells, *not* being *peculiar* to cancer-growth, these species of cancer are *not* associated with *any one* kind of normal tissue, nor with any particular group of normal tissues.

Guided by this reflection, we are prepared to find cancer in textures and organs, severally, whose structural elements are *not* analogous to cancer-cells.

The preference of cancer for different tissues and organs, ranges according to the following average scale (Rokitansky).

"First, the uterus, the female breast, the stomach, the large intestine, and especially the rectum; next, the lymphatic glands, especially as retroperitoneal cancer-accumulation in front of the vertebral column; hepatic, peritoneal cancer; bone-cancer; cancer of the skin and of the lips, of the brain, of the globe of the eye, of the testis, of the ovary, of the kidneys, of the tongue and the œsophagus, of the salivary glands and parotis;"* to which I may add, without pretending to determine their relative places in the scale of frequency, cancer of the urinary bladder, of the pancreas, and of the spleen; in each of which organs I have seen masses of colloid. In one remarkable case the bladder was fully and tensely distended with colloid cancer, feeling like a bag of jelly. The uterus, ovaries, and rectum were also similarly affected.

This scale does not specify the particular species of cancer, in each of the different tissues and organs referred to, yet their relative liabilities in this respect are a matter of great practical importance, considering the unequal degrees of "malignancy" evinced by the several species of cancer.

Encephaloid cancer—the most malignant—selects first, in order of frequency, the testicle; next, the bones, and particularly the femur—(Paget); the intermuscular cellular texture of the limbs; the eyeball or orbit; the breast, the walls of the chest, or abdomen; the lymphatics. Moreover, this species of cancer "occurs

---

* Pathological Anatomy. Translated for Syd. Soc. 1854, vol. i.

in organs in which no other cancer, least of all scirrhous, ever occurs,—as in the liver, the kidneys, the lungs, the testicle, the lymphatic glands "—(Rokitansky).

Encephaloid cancer is not usually solitary. It commonly coexists in many textures and organs.

Melanotic cancer—mostly a variety of encephaloid, by pigmentary degeneration (of its cells), and the most common of all melanotic tumours—is prone to grow first in or beneath pigmentary moles—(Paget); or selects first the liver—(Rokitansky); but it may occur "in the brain and about the nerves; at the eyeball, in the lungs, thyroid gland, liver, spleen, kidneys, bones, lymphatic glands, ovaries, in and beneath the intestinal mucous membrane, between the mesenteric layers, in the skin and subcutaneous areolar texture, upon serous membranes, in the dura mater, upon and within the heart."

This variety of cancer occurs as secondary formations, in very many textures and organs, simultaneously.

Villous cancer—another variety of encephaloid—is produced, according to Rokitansky, exclusively upon membranes; more especially upon mucous membranes, and most of all that of the male urinary bladder, near the opening of either ureter; next to this the mucous membrane of the stomach, and in particular the pyloric portion. It has been observed suspended by a pedicle from the internal membrane of the rectum, and even from that of the gall-bladder. Secondly, it is very apt to grow extensively from the internal wall of ovarian cysts—cysto-carcinoma—where it is recognised as villous cancer by its copious accompaniment of medullary sap. In these cases, it is often concurrent with cancerous infiltration of the lymphatic glands, about the lumbar vertebræ, and with peritoneal cancer, representing villous cancer upon a serous membrane. It has been observed also upon the dura mater; occasionally upon the general integument, and perhaps in bone. Lastly, it occurs in parenchymatous organs.

Epithelial cancer—another variety, allied especially to encephaloid, though somewhat to scirrhous—selects either the skin

or subcutaneous texture, or the mucous membrane: and of these textures the portions most liable are ranged by Paget in the following scale of frequency:—First, its chosen seat is the lower lip, at or near the junction of the skin and mucous membrane; then, the prepuce (glans, Rokitansky),* scrotum of chimney-sweeps; the nymphæ, the tongue; more rarely in very many parts, as at the anus, interior of the cheek, upper lip, mucous membrane of the palate, larynx (trachea, Rokitansky), pharynx and "cardia;" neck and orifice of the uterus (stomach, Rokitansky), rectum, and urinary bladder; skin of the perinæum, of the extremities; the face, head, and various parts of the trunk. In more rare instances, as a primary disease, in other than integumental parts, as in the inguinal lymphatic glands, in bones, in tissues forming the bases or walls of old ulcers. Rokitansky has met with epithelial cancer only once in a parenchyma—namely, in the liver, and then encysted in a capsule of fibro-cellular tissue. By extension from its original seat this growth may involve many deeper textures; fasciæ, muscles, bones; and as a secondary disease may, but very rarely, supervene in internal organs—the lungs, liver, and heart.

Epithelial cancer, as a primary disease, is usually solitary. Occasionally two or more coexist, and even in the same part, as on the prepuce and glans. Eventually, secondary epithelial cancer-growths may form in the tissues surrounding the primary and parent growth.

Scirrhous cancer selects first the breast in the proportion of 95 per cent. (Paget). Next in order of frequency, the stomach; perhaps still more frequently in this organ (Rokitansky); then, according to this last-named authority, the colon in the submucous cellular tissue; more rarely in the vaginal portion of the uterus; upon serous membranes, and in the subserous areolar

---

* This and the other parenthetic insertions here introduced are placed in the most convenient order of association; and do not necessarily represent the order of frequency, respecting epithelial cancer as a primary disease, in the parts mentioned.

tissue. Again, as an expansive degeneration of the omentum, and of the mesentery; in the salivary glands; in the fibrous tissue of the bronchi. In several of these, as well as in other structures—for example, the ovaries and the brain—there occur "cancerous growths of embryonic composition, and in all likelihood of fibro-cancerous (scirrhous) nature." To this list may be added cancer-growths secondary to scirrhus, and which, proportionately as they are consecutive, incline more and more to the condition of encephaloid; for example, of the lymphatic glands, the bones, muscles, skin.

Scirrhous cancer is not unusually solitary.

Colloid cancer selects the stomach and large intestine; the serous membranes, and particularly the peritoneum. In other textures and organs this species is mostly secondary; as in the lymphatic glands, the lungs, the ovaries, the bones, the breasts; "and in rare cases the kidney, uterus, and liver" (Rokitansky); to which I may add the pancreas, spleen, and urinary bladder.

Colloid cancer is usually solitary.

Cartilaginous growths are most frequently connected with the bones and joints; especially those of the hand—*i.e.*, phalanges and metacarpus; the corresponding bones of the foot, particularly the last phalanx of the great toe; the lower end of the femur, neighbouring end of the tibia; the humerus, sternum, ribs, ileum, and cranium. Apart from bone and cartilage, these growths may form in the parotid gland, testicle, mammary gland, lungs, and in the subcutaneous areolar tissue (Rokitansky).

This growth is not unfrequently solitary, excepting in connection with the bones of the hands or feet, where several may coexist. The myeloid variety selects bone more frequently than any other tissue. Paget has seen myeloid growth in the mammary gland, and probably in the neck, near the thyroid gland.

Cystic growths, originating from the erring development of cells or nuclei, may occur in any texture or organ, but more frequently in the kidney, thyroid gland, mammary gland, choroid plexuses, chorion; in the neck, gums, about the sheaths of

tendons at the wrist, forming ganglions; and about the epididymis—as seminal cysts—encysted hydroceles or hydroceles of the spermatic cord (Paget).

Cysts having this origin are single, or numerous, in the same organ or part.

Fatty tumour most commonly occurs in the subcutaneous adipose texture, epecially of those parts where fat normally abounds in the healthy state, and is liable to accumulate; as about the trunk, *e.g.* on the back, neck, and shoulders; also over the glutæi, and the thighs; between the peritoneum and abdominal walls, escaping from which by the abdominal rings, it forms "fatty hernia," so-called (Walshe);* in synovial sacs, more especially that of the knee-joint, the "lipoma arborescens" of Müller. Fatty tumour sometimes forms where fat is normally scanty, as beneath the hairy scalp; or where fat is normally absent, as in the submucous cellular tissue of the stomach, the intestine, the bronchi; in the subserous cellular tissue of the pleura or dura mater, and beneath the investing membrane of the ventricles. Also in the substance of organs, more especially in the lungs, liver, kidneys, and in bone "affected with osteoporosis and eccentrical atrophy" (Rokitansky). Between the corpora albicantia and optic nerve, in one case (Müller).† A fatty tumour, the size of a mushroom, was found between the arachnoid and dura mater, on the level of the fourth lumbar vertebra (Albers).‡ One as large as a walnut, in the walls of the vena portæ (Andral).§

Steatoma—a lard or suet-like variety of fatty tumour—may occur in whatever part of the body this growth makes its appearance; but steatoma has been found in some parts more especially —among them, in the mesentery, testicle, and mediastinum (Walshe).‖

Fatty tumour is usually a solitary growth; but in exceptional

---

* Cyclop. Anatomy and Physiology, Article "Adventitious Products."
† On Cancer, p. 153.
‡ Pathologie, b. ii. s. 189.
§ Anat. Path. ii. 412.
‖ Op. cit., "Adventitious Products."

cases, several may coexist, from two or three, up to as many as a hundred, or more.

Fibrous tumours are formed in connexion with the fibrous or fibro-cellular textures;—most commonly in the substance of the uterus, or in the fibro-muscular tissue of the ligamentous reflections of the peritoneum; in the ovaries, fallopian tubes, or vagina (Walshe); in the interstitial fibro-cellular tissue of nerves. Connected with bone—and like cartilaginous tumours—either in its substance, or between it and the periosteum. The jaws are most liable to this kind of growth. Connected with the dura mater, is another frequent situation. In the submucous cellular tissue, "more particularly of the intestine, stomach, and œsophagus; now and then in that of the larynx" (Rokitansky); in that of the pharynx, the nares, the frontal and sphenoidal sinuses; in the subperitoneal and subpleural tissue (Walshe); in the subcutaneous cellular tissue, as in the lobules of ears, after piercing for ear-rings (Paget); in the mammæ, testicle, thyroid gland, thymus gland; in the arterial tissue, and indeed wherever fibrous or fibro-cellular tissue is normally present or most prevalent.

Fibrous tumour is usually a solitary growth, excepting in the nerves or the uterus; in either of which several may coexist. But when in the uterus, this tumour rarely forms in any other part at the same time (Paget).

Painful subcutaneous tumour or tubercle occurs, as its name denotes, beneath the skin, and especially in the extremities, more particularly the lower limbs; very rarely on the trunk or face. This tumour is solitary in nearly every instance.

Fibro-cellular tumour occurs most frequently in the scrotum, labium, or tissues by the side of the vagina; or in the deep-seated intermuscular spaces in the thigh and arm.

As outgrowths, some proceed from, and are connected with, the mucous membranes—forming polypi, *e.g.* in the nasal passages, very rarely in the antrum; in the external auditory meatus; in the uterus, and urinary bladder (Paget). As cutaneous outgrowths, they appear on the scrotum, prepuce, nymphæ, clitoris and its

prepuce. In one instance,* a fibro-cellular outgrowth—which I examined with the microscope after its removal from the clitoris,—was the size and shape of a large cocoa-nut. It weighed thirty ounces.

Fibro-cellular *tumour* (not outgrowth) is usually solitary.

Lastly, *False tissues*—of which simple fibro-cellular, or connective tissue, is the type—are formed in connexion with the normal or healthy tissues, whose places they severally supply in the reproduction of injured and lost parts, and whose structure they permanently, yet imperfectly, represent—being, as they are, only rudimentary conditions of their analogous healthy tissues, by arrests of their development. Thus, the permanent callus of bone is an imperfect representative, because rudimentary condition of the healthy and original bone. In respect of some tissues, the original one is not at all reproduced, its representative being another and more simple tissue-structure. For example, fibro-cellular tissue only is produced, instead of muscular or nerve-fibres, to repair a breach of their continuity. But, in all cases, nevertheless, the situation and distribution of the new or false tissue is determined by the original tissue, to which it is supplementary.

Surveying Plastic Morbid Products as a whole, the relation which I have endeavoured to establish between them and the development of healthy tissues—their relation to the developmental anatomy of these tissues—is, I think, sufficiently established, and expressed by this law:—that such products represent only retrogressions of analogous healthy tissues to various rudimentary conditions of their structural elements—*i.e.* of certain cells, fibres, and granules; and that deposits, new growths, and false tissues are alike produced by the *arrested* development of these elements; while, I may add Degenerative retrogressions of structure are produced by the *relapse* of these healthy structural elements to more and more simple, though not developmental conditions, of structure.

---

* Lancet, August 22, 1857.

This law indicates an entirely new classification of the whole range of *Plastic* Morbid Products; and their relation severally to healthy tissues, also to Degeneration and Disintegration of Texture, will be seen more clearly by reference to the tabular view on following page—the basis of which first appeared in the "Lancet" for October 31st, 1857.

Such, then, are Morbid Products and Degenerations; but there is yet another or second and further degree of adventitiousness in Pathological Anatomy, as compared with the healthy state of the organism. This results, not from the production, but the introduction of new matters into the body, and which are therefore properly denominated Foreign bodies.

Here again subordinate degrees of adventitiousness may be recognised. Some Foreign bodies are themselves living beings, and which have introduced themselves into the organism, or been originally deposited by their parents, as ova, or seeds. These (invaders) are *Parasites*—either animal or vegetable; for in and upon the organism they invade they live and reproduce themselves, at the expense of that organism. All other foreign bodies are *dead*—albeit organized or unorganized—are therefore introduced accidentally (or designedly), and then and there act only as local irritants, or, becoming encysted with inflammatory lymph, remain dormant, but never undergo any physiological history as separate and independent beings.

Taking first in order, *Parasites*, it is obvious that these animal or vegetable living beings must find in that body which they invade an appropriate food or soil for the completion of their development and growth, and for the germination of their offspring. Herein lies the essential difference between true and false parasites, *i.e.* animals or plants, which, having been introduced accidentally by impurities of food, drink, or air, find not the appropriate conditions to sustain their life for any continued existence, are therefore soon subjected to the laws of organic decomposition, and die without reproducing their species.

NEW ARRANGEMENT OF PLASTIC MORBID PRODUCTS,

Showing their relation to the Structural Elements of Healthy Tissues; also, to Degeneration and Disintegration of Texture.

Retrogressions of the structural elements of Healthy Tissues
By arrests of their development, Supplemental tissues, are :—

Rudimentary Cells.—Pale corpuscles of the blood.....are	Exudation corpuscles. Pus corpuscles. Tubercle corpuscles, miliary and opaque.	Deposits.
Red corpuscles of the blood .....are	Melanic cells—pigment.	
Cartilage cells.....are	Cartilaginous tumour. Myeloid variety.	Growths.
Epithelial cells .....are	Cancer cells—Encephaloid. Scirrhus. Colloid. Epithelial.	
Cells—mode of origin uncertain ...are	Cystic tumour. Fatty tumour.	
Rudimentary Fibres.—White (and Nuclear) with nuclei ...are	Fibrous tumour.	
with nuclei oval are	Fibro-nucleated variety. Recurring fibroid variety.	
White fibre-cells .....are	Painful sub-cutaneous tubercle. Fibro-cellular tumour.	
Connective tissue, etc.		False tissues.

By relapses to more simple conditions of structure, Substitute tissues, are—

Fatty..... Pigmentary ... Fibrous..... Amyloid ..... Granular ..... Calcareous ... Débris of Textures	non-substitution.....	Degeneration. Disintegration.

Human parasites are those animals or vegetables which find their appropriate physiological conditions in the human body. Many such parasites visit indifferently man and other mammalia, and even invade vegetables; others select man as their source of nourishment, and for that of their offspring.

But those conditions of the body which are appropriate for, and conducive to, parasitic organization, life, and reproduction, are *morbid* states in relation to the organization and life of *that* body. Morbid states of the human body predispose to, although they do not engender, parasites, as was formerly supposed. The precise nature of these predisposing conditions, in relation to the different species of parasites, has not been hitherto determined; doubtless they are *peculiar* morbid conditions, for different species of parasites invade different animals, and those which are proper to this or that animal find an appropriate home unfrequently, compared with their extensive dissemination, and they abandon their tenements apparently as capriciously as they took possession.

Human parasites are not apparently subject to the attacks of secondary parasites. But, as they imply previous morbid states of the body, so in their turn they engender other and new morbid conditions, and may also propagate these or similar conditions of disease from one individual to another.

According to the habitation of parasites, in or upon the body, they are known as epi, or ento-zoa, in respect of Animal species; and epi, or ento-phyta, in respect of Vegetable parasites; but these differences are not always obvious, and I shall not pursue them as distinctions in describing the different species of human parasites.

The *Animal* species, all excepting parasitic infusoria, which are akin to the vegetable species, belong to that division of the animal kingdom which Cuvier designated the Articulata. Of these again, in the scale of organization, rank firstly, Insect parasites; secondly, parasitic Arachnida; and thirdly, parasitic Annelida (Annulata). Below all parasitic Articulata come lastly, the aforesaid Infusoria.

Küchenmeister* arranges all parasites in two primary divisions—parasites whose muscles exhibit no transverse striæ, which comprise parasitic infusoria, and annulata,—helmintha, or worms ; and those whose muscles have distinctly-striated fibres, which comprise parasitic arachnida and insecta.

Full particulars of the anatomy and natural history of all these creatures, as well as respecting their therapeutical management, are to be found in various works, especially in that of Küchenmeister. The following brief description—taken chiefly from Rokitansky's able summary,† and from Küchenmeister's standard work—will suffice in this elementary view of Pathological Anatomy :—

Parasitic Insects, infesting the human body, are commonly illustrated by—(1) flies, which infest putrid ulcers with their ova and maggots; (2) fleas—the common flea (Pulex irritans)—the sand-flea (Pulex penetrans), common in the West Indies and in South America; the impregnated female burrows into the skin, especially beneath the toe-nails, where the brood gives rise to malignant sores ; (3) lice—the head-louse (Pediculus capitis)—the crab-louse (Pediculus pubis), infesting, the scalp excepted, every hairy part, and penetrating the skin with its head—the clothes-louse (Pediculus vestimenti), infesting parts of the body devoid of hair, and uncleanly vestments—the louse of wasting disease (Pediculus tabescentium), occurring in great multitudes ; (4) bugs —the ordinary bed-bug (Cimex lectularius).

Parasitic Arachnida are commonly exemplified by (1) the itch-mite (Acarus scabiei, sarcoptes hominis), punctiform, from a quarter to half a millimetre long, ovoid, garnished with transverse, band-like, dorsal striæ, and with central acuminate warts; anteriorly a bristled proboscis, prolonged inferiorly to a band upon

---

* Animal and Vegetable Parasites of the Human Body. Translated for Sydenham Society. 1857. From Second German Edition. By Edwin Lankester, M.D.

† Pathological Anatomy. Syd. Soc., 1854, vol. i.

the thorax; four bristly fore-feet terminating in a disc-plate, whilst the four hind-feet taper into lengthy bristles. It burrows in the epidermis, often boring beneath it a canal several lines long, at the termination of which the acarus is, on close inspection, seen as a minute whitish speck, marked with a brown point. When the said canals penetrate to the cutis, they engender the itch-vesicles and pustules. (2) The follicle-mite (Acarus commedonum, sive folliculorum), an elongated acarus, from one-fifth to one-third of a millimetre long, and about one-twentieth broad, the head having two lateral antennæ and an intermediate proboscis. The head passes immediately into the anterior part of the body, which occupies about one-fourth of the entire mite. From it project four pair of very short, thick, conoid, three-pointed feet, each furnished with three toes. The anterior body passes without break into the posterior, which gradually tapers, but is rounded off at the extremity, is transversely striated, and contains a finely-granular, brownish mass. It inhabits singly or numerously the hair-sacs and sebaceous follicles on various parts of the person. Amongst other anomalies, it occasionally displays only six feet, which no doubt implies an earlier state of its development. Its presence is probably often of little moment. Occasionally, however, it may, by stimulating the secretion, engender commedones, or excite inflammation and thus give rise to the acne-pustule.

To these two parasitic arachnida may be probably added gnats and mosquitoes; scorpions, house-spiders, and hunting-spiders; bees, wasps, and hornets (Küchenmeister).

Parasitic Annulata are equivalent to the helminthoid entozoa, or worms, in a mature or immature state.

Cestoid or tape-worms are characterized by their enduring growth and by the great length to which they attain. They consist of a succession of linked joints, of which the fully-developed sexually-mature, hindmost ones, become cast off in greater or lesser series; whilst at the neck fresh joints are continually being reproduced. As in these again, a brood is rarely seen associated with the old individuals, whilst the separated sexually-mature

joints so frequently become ejected, it is probable that the embryones are developed externally to the animal they infest, to re-immigrate subsequently.

The ordinary tape-worm (Tænia solium), the long-jointed tape-worm, chain-worm, is a white or yellowish-white worm, twenty feet long or more, anteriorly thin, roundish, posteriorly flat, and from three to six lines broad, jointed. The joints are flat, square, and towards the distal end more and more oblong-square, resembling gourd-seeds with truncated apices. At the right or left margin, often alternately, is seen a wartlike projection marked by a pore with a raised brink. This is the orifice of the sexual organ, which represents a cavity dendritically branched throughout the joint. The head constitutes at the very thin anterior termination, a nodule-like intumescence, with four lateral black points in relief. There are four suction-pores; and between them is seated, upon a slightly raised circle, a double coronet of hooklets. The annulate neck is studded with numerous lime corpuscles of the most various size. Tænia solium inhabits the small intestine in man, almost in all districts, except where the Bothriocephalus latus occurs. Commonly solitary, as many as nine (Rokitansky) or forty (Küchenmeister) may co-exist in the same individual. This worm occasions the well-known annoyances, but no visible anatomical mischief.

The broad or broad-jointed tape-worm (Bothriocephalus latus, Tænia lata) resembles the last in many particulars, equalling it in length, and being in like manner jointed. Its joints are usually broader than those of T. solium; this alone, however, is not a diagnostic mark. The wartlike projections are not, as in the other worm, seated at the margin, but at the centre of the ventral surface. Their pore leads to a branched, rosette-shaped sexual organ. The head, differing from that of T. solium, has no suction-pores, but two longish grooves. This worm rarely parts with single joints or links, but usually with a greater or lesser chain of them. It inhabits the small intestine in man, but is strictly limited to Russia, Poland, Prussia (trans Vistulam), Switzerland,

and the South of France. If it occur elsewhere, it is assuredly imported from one of those countries.

Tænia mediocanellata—so named by Küchenmeister, from the uterus being a thick-walled, straight and median canal, and fully described by this observer—is thus mentioned by Nicolai:* "Capite inermi aculeato sessili, articulis dilatatis brevioribus, marginis utriusque medio latiore, alterius osculato, majoribus transverse striatis, emarginatis."

"If," observes Küchenmeister, "the head of this tænia did not resist attempts at expulsion with such extraordinary obstinacy, it would long since have been recognised." Segments (proglottides) of the body are passed more abundantly than those of T. solium; from which it may be inferred that the reproduction and growth of T. mediocanellata is peculiarly rapid. These segments are constantly and extensively passed without fæces; they occasion a troublesome itching on the sphincter ani muscle from within; having passed, they feel cold and clammy, and are apt to cling to the thighs, on which they deposit their eggs, which appear like white damp sand. Segments passed with the fæces may also immediately lay their eggs on them, whereby the fæces look as if sprinkled with white sand. This tænia, inhabiting the human intestine, occurs in Europe and Africa. A variety is found at the Cape of Good Hope (Küchenmeister).

Tænia nana (Bilharz, Von Siebold) is thus described by Küchenmeister:—"Corpus filiforme, depressum; caput antice obtusum, collum versus sensim attenuatum, acetabulis subglobosis, rostello pyriformi uncinulorum bifidorum corona armatum. Articuli transversi; cirri unilaterales, ovula globosa, testa laevi simplice (?) instructa $\frac{1}{100}'''$ magna. Longitudo totalis 6—10'''. Patria Ægyptus, in hominis intestino tenui semel reperta numero permagno."

Cestoid or tape-worms may be found in *immature* states.

---

* Neuer Zeitschrift für Natur und Heilkunde, Von Ammon, Choulant und Ficinus, i. p. 464.

For example, the larva or scolex state of Tænia solium is known as the Cysticercus cellulosus. Taking Rokitansky's description, it consists of a conical, snow-white, transversely rugous body, and of a vesicle which is its caudal extremity. The vesicle is oval, spherical or square, in muscles—cylindrical, parallel to the muscular fibres, and of the size of a pea or haricot bean, in rare instances—for example, in the ventricles of the brain—of a hazel nut. When the animal is retracted into this vesicle, it appears as a white, spherical, solid body, seated somewhat eccentrically on its inner surface, whilst upon the vesicle itself is observable, externally, a delicate point-like fold or depression at the same spot. When the animal is external to the vesicle, a condition easily brought about by puncturing the vesicle and pressing the hardish spherical body between the finger and thumb, a pore becomes perceptible, which leads to the interior of the animal pouch. Taking the size of the caudal vesicle at the ordinary one of a pea, the animal itself, *i.e.* the trunk, would about equal the diameter of the vesicle, both together measuring from six to twelve lines in length. The neck is short, very thin, and, like the body, wrinkled. Upon it is seated the largish, bulb-shaped, or rhomboidal head, upon which there is at each angle a circular suction-cup; and midway between these a proboscis, cone-shaped, in its protruded state, with, at its extremity, a coronet of hooklets, consisting of a double row, about thirty-two in all, which, when retracted, pack up into a funnel-shaped cup. The two circles of hooklets are identical in shape; those of the outer circle are, however, much smaller, while both are so disposed that the larger and smaller hooklets alternate with each other. The above-mentioned transversely-wrinkled anterior portion of the creature is covered with fine black contoured molecules, and studded with several superimposed layers of lime corpuscles, which close to the caudal vesicle, suddenly and entirely disappear. This anterior portion, and the caudal vesicle, consist alike of membrane almost structureless. The latter is besprinkled with countless fat-molecules, and contains a watery, slightly albuminous, and neutral fluid.

Wherever the cysticercus may be found in textures, it is enclosed within a second cyst of fibrous texture. This membrane is permeated by delicate blood-vessels, and is rendered transparent by acetic acid. If the cysticercus be free, within a cavity, as within the cerebral ventricles, it is then uninvested; showing that the outer cyst, in other localities, is adventitious.

When the creature perishes, as frequently happens from disease of the outer cyst, the caudal vesicle becomes semi-opaque, collapsed, its contents turbid, displaying the said lime corpuscles and hooklets, which, together with a granulate mass, are found floating in its fluid. The entire creature softens and liquifies, afterwards condenses, and eventually settles into a cretaceous concretion. Meanwhile, the outer cyst shrivels into a thick-membraned capsule, which isolates this concretion.

Mature tæniæ are found only in the intestine, but the immature in other parts of the body.

Thus, Cysticercus cellulosus occurs in the brain, in the striated muscles, including the heart, and in the areolar tissue. It may also be free—without its outer cyst—in the ventricles of the brain, and in the chambers of the eye. It sometimes occurs in the muscles and brain simultaneously, in great multitudes. Even in the brain it is usually borne imperceptibly. When present there in great numbers, however, it often occasions vertigo, and it has proved fatal by exciting inflammation in its vicinity.

A variety—Cysticercus tenuicollis, or visceralis—is now rare. It has been found in the human mesentery, and in the liver (Küchenmeister.)

Echinococcus hominis, the E. altriciparіens of this last-named authority, is regarded by him as another form of immature tænia. This entozoon, and the cysts in which it is formed in great numbers, have, according to Rokitansky, the following characters:—Within a sac of fibroid texture is enclosed a solitary, independent, thoroughly distended vesicle, containing a limpid, serous fluid, or else inclosing, as a parent vesicle, other similar vesicles, spherical or flattened by mutual compression, either floating at large in the

contained fluid, or sessile upon the inner membrane of the said parent. These sub-vesicles range in size from that of a poppy-seed to the magnitude of a goose's egg and more. In number they may amount to hundreds, so that the serous fluid of the parent vesicle is but little.

In very voluminous sacs, the parent vesicle commonly appears to be wanting. Either it has split up and mixed with the younger vesicles, or it has disappeared by the excessive attenuation consequent on its enlargement. The sub-vesicles occasionally contain others similar, of a third, and these again, in rare instances, of a fourth generation. On the inner surface of the sub-vesicles is frequently found a whitish, opaque, gritty efflorescence, and densely-nestled animalcules. They are the echinococci. Their characters vary with their development.

When developed, this entozoon (Echinococcus) has a tænioid head, with four lateral suction-pores, and a proboscis garnished with a double coronet of hooklets. The head is distinguished from the thicker, spheroid trunk, by an annulate indentation. From the proboscis a longitudinal striation runs to the posterior part; and, commencing from these striæ, the body of the animal is transversely striated. The posterior termination is a transverse cleft, in which is inserted a cordlike formation, by means of which the creature maintains its seat upon the vesicle. Between the striæ of the trunk are spherical or oval limelike corpuscles, resembling those upon the cysticercus.

Such being its developed state, it is met with under various other shapes. For example, it appears as an elongated sphere, in the centre of which the coronet of hooklets appears perspicuous when the head is retracted; or it assumes the shape of a heart, a pitcher, or even of a horse-shoe.

Echinococcus cysts are particularly frequent in the liver, less and less so in the sub-peritoneal areolar tissue, peritoneum, omentum, striated muscles—including the heart, in the brain, the spleen—mostly in concurrence with others, in the liver, in the kidneys; very rarely in the lungs and bones. Not unfrequently they occur

in several organs simultaneously. Thus, they may infest, in vast numbers, both the peritoneum and the abdominal viscera. In magnitude, the sacs sometimes attain, or even exceed, the diameter of a foot. Echinococcus cysts occasionally become perilous through their volume, and when present in great numbers prove fatal through exhaustion and general wasting, or by inflammatory and suppurative processes.

A variety—Echinococcus scoliciparíens, E. veterinorum—has been seen in the human eye by Gescheidt and by Eschricht.

Acephalo-cysts are regarded by Rokitansky as nothing more than those vesicles which the echinococci inhabit, but which are in some instances sterile. These cysts are, therefore, not independent formations, and such is Siebold's opinion. Küchenmeister now considers them independent living beings. "They are," says he, "six-hooded cestode embryos, the growth of which has proceeded without hindrance, but which nevertheless have remained barren, or, more correctly, have never attained to proliferation and the production of scolices."

Another species of Cysticercus—according to Creplin, the Cysticercus vesicæ hominis, so named because he supposed it came from the urinary bladder of man—is probably Echinococcus altricipariens, and comes from the kidney (Küchenmeister).

Næmatoid or round-worms, thread-worms, present many varieties as parasitic entozoa in the human subject.

The cylinder-worm—Ascaris lumbricoides—is quill-shaped, tapering towards both extremities, especially towards the anterior; from five to twelve inches long; has four longitudinal striæ, two of which are more marked; densely marked with transverse striæ; semi-transparent, so that the intestinal canal and the organs of reproduction are transpicuous. The junction of the head to the body is indicated by an annular groove, and on the head are three little valves, which encircle the mouth. The caudal extremity is incurvate, especially in the male. Sexes distinct, the male being smaller and narrower, and having at its caudal extremity a thin penis, sometimes double. The female, larger, exhibits at its upper

third a fissure, as the orifice to the organs of generation, which contain ovaries and oviducts of enormous length. This entozoon infests the ileum, often in extraordinary number, forming groups and conglomerate masses. A brood is never seen; the ova, therefore, are probably hatched extraneously to the human body, to re-migrate thither afterwards, as the living brood. Perforation of the intestine (migration extraneous to the intestine) and its consequences, are, to say the least, extremely rare.

The hook-tail, maw-worm—Ascaris vermicularis (Rudolphi), Oxyuris vermicularis—is a little thin white worm. Sexes distinct. The male, very rare and small, with spiral convoluted tail, annulate. The head of either has a transparent swelling, which, under the microscope, appears as a wing-like membrane.

It inhabits the colon, and especially the rectum, occasioning both here and in the vagina, into which it creeps, an intolerable itching. As it is never accompanied by a brood, it probably migrates as the impregnated female.

The hair-head or whip-worm—Trichocephalus dispar—is filiform, the anterior part hair-like, the posterior considerably thicker, from one and a half to two inches long. Sexes distinct. The male at its posterior part is spirally convoluted, and its penis is contained in an elongated, funnel-shaped, violet-coloured sheath. The posterior part of the female is not spiral.

This entozoon infests the cæcum, singly, and frequently also in multitudes (especially in the dead bodies of persons who have died of protracted typhus or similar diseases), without occasioning any extraordinary symptoms. The females are loaded with ova, which are not, however, developed in this locality.

Trichina spiralis is regarded by Küchenmeister as the progeny of Trichocephalus dispar; and by Rokitansky as a strayed nematodon, which, without coming to maturity, encysts itself, perishes, and cretifies within a second cyst thrown out from the textures. In the inner cyst, amidst a granular, viscid, transparent fluid, lies the worm, perfectly free, and generally rolled up in two and a half spiral convolutions. When extended,

it is from one-twentyfifth to one-thirtieth of an inch long, and about one-sixhundredth broad, lumbricoid, thread-like at both extremities, although more pointed at the one than at the other. Internally is a winding canal, supposed to be the intestine, and a granular organ, the designation of which, as an ovary, is without doubt erroneous (Rokitansky).

Occasionally, the cyst contains two, or even three worms.

Trichina spiralis inhabits the voluntary muscles, and always in vast multitudes, the muscles appearing to the naked eye studded with little white specks. The cysts always lie with their long diameter in the course of the muscular fibres.

This worm and the Cysticercus cellulosus are the two parasitic worms which infest the voluntary striated muscles in the human subject; but the latter worm inhabits the muscular fibres of the heart also, besides other textures and organs. Trichina spiralis is related to Trichocephalus dispar, a næmatoid or round-worm; and Cysticercus cellulosus is the scolex of Tænia solium, a cestoid or tape-worm.

The guinea-worm—Filaria medinensis—is another næmatoid worm, about the thickness of packthread, whitish, from half a foot to several feet long, at the broader end obtunded, terminating behind in a pointed curve. Peculiar to tropical countries of the Old World, but especially to Guinea, it inhabits the sub-cutaneous cellular tissue, particularly of the lower extremities, but occasionally also of the scrotum, the trunk, and the throat. Having spent its earlier period out of the body, it burrows beneath the skin, and resides in the areolar tissue for a considerable time, say several months; then it again perforates the skin from within, in order to deposit its offspring, or, it may be, to migrate for this purpose altogether. It would seem that hitherto none but females have been observed; accordingly, they must have introduced themselves in the impregnated state. These migrations are attended with inflammation and ulceration, which continue for a considerable period.

Other species of næmatoid parasitic worms are comparatively unimportant, being either rare or questionable. Of these are—the

giant strongle—Strongylus gigans—a cylindrical worm, very large, from five inches to three feet long, and from two to six lines in thickness; when recent of a fine red colour; it inhabits the kidneys, but is rare in man and brutes. Filaria bronchialis, once seen by Trentler in a degenerated bronchial gland in the human subject. Filaria oculi humani, in the liquor morgagni and in the cataractous lens (Gescheidt, Nordmann). Ancyclostomum duodenale (Dubini), in the duodenum. Certain filaria in the blood (Kleuke). Spiroptera hominis (Barnett), in the urine. And the Dactylius aculeatus (Curling), also in the urine.

Trematode, or suction-worms, are especially characterized by their peregrinations and metamorphoses. None of these parasitic worms are commonly found in man.

Liver-fluke—Distoma hepaticum, and D. lanceolatum—are flat, melon-seed or lancet-shaped, soft worms, of a yellowish-white colour, with two suction-pores, one of which is seated at the head extremity; the other, which terminates cæcally at the belly between the two, is the sexual orifice. They are hermaphrodites. D. hepaticum is the larger, being from four to eight, or even fourteen, lines long, and from one and a half to six broad, with a branched intestinal canal. D. lanceolatum, as the smaller, is from two to four lines long, and about one broad. Its intestinal canal is bifurcated. Both infest the liver of the herbivora, but rarely of man. The latter species has been seen only once in the human subject.

Other species of Distoma are even more unimportant; and being fully described in special works on parasites, I need but mention them, and the parts of the human body in which they may be found.

Distomum heterophyes (Von Siebold), discovered by Bilharz, is peculiar to Egypt. It has been found twice in the small intestine, where however large numbers were present.

Distomum hæmatobium (Bilharz), also peculiar to Egypt. Found in the vena portae and its branches, and in the walls of the urinary bladder. Küchenmeister adds—"in venis meseraicis reperi-

untur mares feminam in canali gynæcophoro gerentes, in venis intestinalibus et hepatico, in vena lienali, semper vidui."

Distomum ophthalmobium (Diesing), D. oculi. Discovered once in the eye of a child five months old, born with lenticular cataract, and who died from infantile atrophy. Four specimens of Distoma were found between the lens and its capsule (Gescheidt).

Infusoria occur as the lowest forms of animal parasites.

Denticola hominis (Ficinus) (?) is an aciliated infusorium, with its mouth on the ventral surface, and probably furnished with a carapace. It is regarded by Ficinus as a distinct genus, allied to the Monades and Vibriones. The species, according to this observer, are numerous, as almost every mammal has its peculiar species.

This parasitic infusorium inhabits the tartar of the teeth. Ficinus discovered his Denticola in all persons having teeth, especially in the interstices of the molars; more rarely on the mucous membrane of the mouth, scarcely at all in the saliva, but especially when the teeth were neglected, and, in immense numbers, in hollow teeth. In spirituous and acid fluids, and fluids mixed with cigar smoke and spices, they do not live in the superficial strata, but still exist in the lower ones.

The so-called Trichomonas vaginalis occurs only in women with gonorrhœal discharge; but its animal nature is uncertain. It is possibly or probably only ciliated epithelium.

Vegetable parasites are prone to infest the human body; they all belong to the great class Thallogens (Lindley), and to the orders Algæ and Fungi, exclusively.

The characters of their structure, and the pathological appearances of the textures or parts affected, are fully described by Küchenmeister, from the most recent observations and authentic sources of information respecting these parasites. I have therefore selected the more important particulars from his description of them, as follows:—

Parasitic Algæ found upon living animals (including man),

consist of cylindrical or flattened filaments, single or branched, frequently with dissepiments, or apparently articulated at certain distances, and containing greenish or grayish molecular granulations in varying quantities. Each of these granular masses is called a "gonidium," whilst the granulated cell-contents are called the "endochrome." These algæ have no special apparatus for fixing them in the mucus of the affected animal, they are held firm by the crossing of the fibres.

The reproductive system consists of the "sporangium" and the "spores."

The sporangium, conceptacle, or spore-case, is the organ in which the spores originate, are developed, and enclosed. It is formed from a variously-shaped vesicle, which is universally of larger size than the cells of the vegetative system, and originates in the extreme cell of a tube whose contents serve for the production of the spores.

The spores, sporules, corps, reproducteurs, sporidia, spora, sporulæ, corpora or cellulæ gonimicæ, spermatia, &c., are round or oval bodies, containing universally in their interior finely-granulated corpuscles. They vary in size, but are easily distinguished either by their appearance or their germination.

Man, according to Robin, grows on his body ten species of Algæ, distributed in five genera; or, if the five species of Leptomitus are to be regarded as one, then five species in five genera; or, if the genus Leptomitus is a depauperated fungus, incapable of fructifying, because deprived of air (Robin), then four species in four genera. They all belong to the class Isocarpeæ, and to the order Eremospermeæ with the exception of Miresmopædia ventriculi, which Meyen has placed in the tribe Palmelleæ.

Cryptococcus cerevisiæ, Torula cerevisiæ—ferment or yeast-plant—is a parasitical plant composed of round or oval cells, which often present in their interior one or two little corpuscles more like globules of oil, or the nucleus of a cell, than a vesicle. They are propagated by small projecting bodies on the sides of the cells, which, when they attain the size of the parent cells, give

origin to new germs, and form a row of from three to five elongated cells, but never a cylindrical stem. In the air it immediately decomposes; it therefore does not fructify (in the air) as Fungi do. The presence of one or two brilliant, highly refractive globules within the cells, and which are often regarded as globules of oil, is very characteristic.

This plant is not peculiar to yeast; it forms in diabetic urine, and is developed morbidly in the secretions of the mouth, œsophagus, stomach, and intestines, or is introduced into these parts by means of beer. It has also been found in the black fur of the tongue of persons labouring under typhus (Hannover); in the mouth of a woman who had long previously suffered from disease of the womb (Lebert); in fæces and vomited matter (Vogel); in the bitter fluid vomited by a woman who, after fasting many weeks, ate some decomposing apples (Robin); in a woman who for eight years had laboured under hysteria, accompanied during four years with daily vomiting—the vomited matter contained the yeast plant (Gruby); in the vomited matter from a cholera patient (Bennett), and constituting the so-called "cholera fungi" of Swayne, Brittan, and Budd, which they discovered in the stools and vomited matter of cholera patients, and which the latter observer discovered in the water and air of the affected place. The identity of this "fungus" and the yeast or ferment alga has been demonstrated by Baly, Gull, Griffith, Bennett, Robertson, Robin, and others. This "fungus" may occur in the urine of persons affected with scarlet fever (Vogel, Ilmoni); thus showing that sugar is not necessary for its production. It has also been seen in the urine of cholera patients (Herapath, R. Quain).

The relation of this plant to fermentation is not that of cause and effect. In all cases fermentation has commenced previous to the development of the cryptococcus. Decomposition of saccharine matter, or liquid acids at a favourable temperature, as in the intestine, is accompanied with its development.

Merismopædia ventriculi (Meyen), Sarcina ventriculi (Goodsir), is a transparent plant, formed of cubical, elongated, pris-

matic, or irregular masses, which are ordinarily composed of eight, sixteen, or sixty-four cubical cells. Each cell is divided on its surface by slight furrows, into four prominences (pustula, Goodsir). The neighbouring cells touch, or barely touch each other, and are usually of a faint red colour. The internal nucleus has the bright brown colour of the entire mass.

Discovered in vomited and fæcal matters, and coming therefore from the stomach or intestine, this plant may also occur in urinary deposits, "et pene tabido et abscessum gangrænosorum ex c. pulmonum"—(Küchenmeister).

Leptothrix buccalis consists of small, semi-transparent, finely granular, yellowish masses, of variable shape and size; and of numerous round, straight filaments, free at one end, and by the other planted in the granular mass. When highly magnified, small round granules—spores, are seen between the filaments. The filaments depend sometimes from a kind of stem, but there is no branching or movement of the filaments, nor are there sporangia, or clearly, spores present. Vibriones are associated with this parasite, but they are very small, and always mixed with epithelial cells, mucus and pus-globules, and molecular matter. Individual filaments may be free in the saliva (Lebert).

The soil on which these plants grow is the decomposing deposits of food which lie between the papillæ of the tongue and their processes; also in great abundance, and very fine, on the soft masses of food which collect between the teeth when unbrushed, and especially if such matter is allowed to accumulate for some days; between the tonsils in a dead body, they were found by Wedl; in the stomach and small intestines frequently; and Robin observed them in the stools of typhus patients.

Leptomitus urophilus, the nature of which is still doubtful, occurs "in urina morbosa cum filis emiosa" (Rayer).

Leptomitus (?) Hannoverii is developed upon the living body on the tongue and pharynx. The structure of this parasite has been differently described by Hannover, Meyer, and Robin.

Leptomitus (?) epidermidis—in white vesicles, like those of

eczema, after poultices, and which formed on the back of the hand—a reddish fluid, contained a number of "byssoid filaments," which were very long, frequently divided transversely; but were less clearly transparent and articulated than the filaments of "muguet." Partition walls could be clearly seen, especially towards the ends of the primitive filaments, and in the secondary branches. Spores were not seen within the filaments, but free in the sporidia. The latter had an elliptical form, straight or slightly bent, and were divided into two cavities by a partition wall. These facts are according to the observations of Gubler, who discovered this parasite.

Leptomitus uteri, discovered by Lebert (1850) in uterine mucus, has since been more accurately described by Robin.

This parasitic alga consists—(1) of naked tubes, which are more or less elongated and branched, and without partition walls and granulations in their interior. (2) Of tubes a little broader, articulated, and furnished with partition walls of varying length, sometimes branched, and which are terminated by granulated masses or spores. (3) Of spores which are sometimes formed of ovoid, elongated, granulated cells, with one or two clear drops in their interior, and sometimes of ovoid or spherical cells with prolongations. The last cell of the receptaculum which bears the spores is ordinarily more swollen than the others, and a little granulated.

Leptomitus (?) muci uterini—discovered by Wilkinson in a mouriform discharge from the uterus of a woman seventy-six years old. It consisted of primary and secondary filaments. Their edges were colourless. They were of various lengths and bent. Rendered transparent by acetic acid, these filaments were then seen to consist of elongated cells, disposed end to end, as in many fresh-water confervæ. In some of the filaments this cellular structure disappeared, and they appeared as simple filaments.

The primary filaments were from two to six times larger than the secondary. The broadest were shortest, and terminated at

one end bluntly, and at the other with a bundle of six or seven long secondary filaments. The blunt ends of the primary filaments seemed to be adapted to form partition walls and spores. Associated were certain ovoid or spherical corpuscles, which, when treated with acetic acid, mostly presented a nucleus.

Leptomitus (?) Oculi.—After inflammation of both eyes, which was attended with a sudden "sanguineous enlargement" in the left, but which subsided under warm fomentations and a foot-bath, epiphora and a flashing in the eye remained. By resting the eye this also disappeared; when suddenly, and without any obvious cause, figures of a constant form were seen with the left eye, and *muscæ volitantes* with the right. The last affection disappeared, but there remained in the field of vision of the left eye a constant form, which moved itself in a definite manner in various directions. Subsequently, the patient had a fall from a carriage, when the movements of the figure became more free. Helmbrecht, who had observed all these facts, now made a puncture in the lower part of the cornea, to allow of the passage of the body supposed to be loosened by the fall. In the fluid which came away, there was found, by microscopic examination with a power of 280 diameters, a branched vegetable body, divided into four parts, which consisted of confervoid cylinders and rows of spores. After this operation the patient quite recovered. Hannover, in his recent work on the eye (1852), has related a very similar case.

This parasitic plant much resembles the ferment alga.

Oscillaria intestini is another parasitic alga, composed of a number of elongated filaments, with partition walls which cross each other in all directions. Each of the cells is considerably elongated, and contains a quantity of green matter. According to Farre, the spores of this alga must have been taken into the intestine by drinking water. He found it enveloped in membranous reddish masses, which came away during an attack of colic in a dyspeptic woman.

Fungi are the only other order of vegetable parasites, and

include several species. According to Lindley's definition,* they are "cellular, flowerless plants, nourished through their thallus (spawn or mycelium) living in air; propagated by spores, colourless or brown, and sometimes enclosed in asci, destitute of green gonidia." To render this definition clearly intelligible, it is necessary to bear in mind the precise meaning of the technical terms employed in it. Thus, "thallus" represents the fusion of root, stem, and leaves into one general mass; "asci" are tubes in which the spores—"reproductive spheroids"—are contained, and which are then named sporidia; "gonidia" are reproductive germs of a green colour.

Trichophyton tonsurans (Malmsten) is the fungus of Porrigo. P. circinata and P. tonsoria are synonyms for the disease accompanying this fungus.

The filaments placed in rows in which the spores originate, have undulated edges, and show in their interior, at small intervals, the spores; round, transparent, and half as large as blood-corpuscles. Many have, in their interior, a distinct spot or vaguely-defined nucleus; many, when they are elongated in shape, appear to have a constriction in their middle.

This fungus is not found between the cells of the epidermis, but in the substance of the root of the hair itself; though it is doubtful whether the fungus thrives only in diseased, or also in healthy hair, after its spores have once penetrated the substance of the hair. The spores form at first a round heap, which spreads more or less upwards into the hair, which thus becomes enlarged (tinea or herpes tonsurans). The fungus continues to grow with the hair, and when it has grown 2 or 3 mm. above the skin together with the hair, the latter breaks off.

Effects of the parasite:—Small rugged elevations on round spots are produced, chiefly on that part of the head which is covered with hair, and which give to it the appearance of seal-skin. Wherever the hair has broken off, the part is bald. In such

---

* Vegetable Kingdom, 1846.

patches the skin is dry, firmer, and more contracted than the surrounding integument. Small round inequalities, resembling goose-skin, may be seen and felt. The colour of the skin is slightly bluish, and when scratched it becomes covered with a white dust like fine bran. The disease begins in a very small spot in the middle of the circle which it afterwards forms, and spreads from thence eccentrically. The same mode of diffusion is observed when the patches are eventually uniting into one. Sometimes this disease spreads over all the hair of the body, and even attacks the nails.

Trichophyton, or Mycoderma (Günsburg) Plicæ polonicæ.—Different views are entertained by observers respecting the import of this parasite: some regarding it as a pathognomonic sign of Plica polonica, while most modern writers—J. Müller, Münter, Baum, Simon, Hessling, Skoda, and F. Müller, regard it as merely an accidental concomitant in the case in which Günsburg originally made his discovery.

This parasite consists of articulated filaments. The spores are very numerous, round or oval, with a smooth surface, but sometimes articulated by "umbilical spots."

The following changes in the hair, according to Günsburg, are produced by this parasite:—The root of the hair becomes thickened; its shaft enlarged by the upward growth of the fungus in it, the hair splits and assumes the appearance of a hedge-hog's brush, the spores escape therefrom, and the hair disappears, or adheres in tufts. Hebra and Wedl never found spores within the "hair-canals," but masses of parasitical plants on and between the plicated hair. Spores also were distinctly seen everywhere on the adherent mass. The hair itself was brittle, and split.

Trichophyton (?) ulcerum.—The scabs showed here and there dry, yellow spots, and looked like mould. The fungus consisted of spores, round or slightly elliptical and large, with nuclei. Other spores were full of small globules. The former joined, and formed threads like strings of pearls, some of which were branched. Every

transition from the simple globules to the threads and branches was discernible.

Microsporon Audouini (Gruby)—fungus of Porrigo decalvans, Trichophyton decalvans—unlike T. tonsurans, forms a tube *around* each hair, and surrounds the hair outside the follicle. It consists of filaments, branched, and bearing spores. The hair becomes less transparent, thicker, very finely granulated, and eventually breaks. If the hair has become gray from its root, it breaks off about a week after the spot where the sheath of the plant begins, and is followed by baldness. The "hair-epithelium" likewise falls off. Around the follicles, masses of the fungus heap up, which have been mistaken for pustules or secretion of the sebaceous glands. Light gray crusts cover the bald places, and these crusts consist of the parasite mixed with epithelial cells. Its contagious nature is thus explained, and the contagious matter of P. decalvans is nothing more nor less than the spores of Microsporon Audouini.

Microsporon mentagrophytes—fungus of Mentagra—is distinguished from the last species by larger filaments, branches, and spores, and by its seat.

The spores, which are in countless numbers, very small and round, hang with one part on the inner surface of the sheath of the hair, and the other on the hair itself. The filaments or stalks are granulated inside, and are divided in the shape of a fork. The branches are annulated.

This fungus penetrates into the follicle of the hair to its very root, between the latter and the wall of the follicle. It settles neither in the substance of the hair which lies in the follicle, like Trichophyton tonsurans, nor around the part which is exposed to the air close to the skin, like Microsporon Audouini. Thus, Microsporon mentagrophytes forms—according to Gruby—a kind of vegetable sheath surrounding that part of the hair which is imbedded in the skin, and whose spores are never produced above the surface of the skin. All the diseased parts of the hair are covered with white, gray, and yellow scales, which are penetrated in all places with hair.

This parasite grows in the follicle of the hair of the beard, more especially of the chin, the upper lip, and cheek; and, according to Bazin, also in the tufts of hair of the skin in general.

Microsporon furfur, fungus of Pityriasis versicolor—consists of elongated and branched cells, partly of spores which are heaped up in groups. They are highly refractive. Caustic ammonia added to the crusts or scales of the diseased skin renders the parasite more distinctly visible.

This fungus is ushered in by the formation of yellowish or yellowish-brown spots, of various sizes, but never rising above the level of the skin, are pulverulent on their surface, and are constantly scaling off and itching. The whole constitutes the Pityriasis versicolor. These spots, at first the size of a pea, enlarge and spread to the breadth of two hands and uninterruptedly from the thorax to the stomach. It is sometimes found on the extremities, but never on parts exposed to the air. The disease is contagious, being propagated by the parasite (Sluyter and Eichstädt). It has never been known to occur previous to puberty, but always after the fourteenth to the sixteenth year; and it seems more particularly to attack those persons who have tuberculosis.

Achorion Schöenleinii—Fungus Porriginis, Fungus of Favus—consists of filaments with spores. This parasite attaches itself to the bottom of the hair-follicles in the direction of the hair; more commonly, however, to the cells of the epidermis. Here spores only, or closely articulated filaments, are met with. These spores adhere for the most part to the hair, and create on its surface circular enveloping masses, which spread out and form a kind of sheath for it. The spores may form single, double, or triple rows, constituting a kind of network which adheres firmly to the hair. Sometimes the spores penetrate into the root of the hair, which then becomes disfigured, dried up, and fibrous. Spores are likewise found at that part of the hair which is free, outside the follicle, on the angles formed by the hair in its folds.

If the parasite be accumulated in the depressions of the skin, forming a cup, "godet or favus;" then, not only spores, but

every part of the plant, together with spores, are found. These lie at first beneath the epidermis; they gradually penetrate, still under the epidermis, into the follicle, and unite with those of the neighbouring hair, whilst the skin becomes thinner. When the favus is very large, it generally throws off the dried epidermis, whereby the parasite is exposed; and when the parasites of several infected hairs run together, large crusts of favi are found, underneath which the skin is changed to a large extent.

The favus is hard, dry, and brittle; its fracture shining; its interior whitish yellow, and paler than the outer surface. It is spongy, easily rubbed to a yellowish white dust, representing under the microscope a mixture of mycelium receptacles and spores; which shows clearly the various steps of transition among themselves.

The favi are principally found on the head covered with hair, but also on all other parts of the body; on the face, shoulder-blades, external ear, front of the thighs, penis, and scrotum; and not merely on those parts of the skin which are more or less hairy.

Eventually, the favi become associated with the products of inflammation; fungi, pus, and serum together forming scabs.

Oidium albicans—Fungus of Aphthæ, Soor, Muguet, Thrush—is a parasite consisting of tubular filaments bearing spores, and of spherical or at first oval spores. Its presence is denoted by a disease of the mucous membrane, which manifests itself sometimes in the form of small points, rings, conical and semi-spherical elevations; sometimes in the shape of large spots, and able to form a compound membranous envelope. This envelope is originally of a milk or pearl-white colour, passing into gray or yellowish when the disease pursues its own course or occurs in weaned children, but rarely assuming a darker colour in children, which happens only when a foreign colouring matter acts on it. The external portion has a cheesy consistence, and the thickness of very fine paper only to half a line or more; it adheres firmer at first than after a little while, and finally peels off spontaneously without injuring the continuity of the mucous membrane.

This disease occurs alone, or simultaneously, on the inner edge of the lips, where the mucous membrane begins; on the inner side of the cheek, on the gums and palate; on the tongue, above and below; in the throat, and in the œsophagus, down as far as the cardia.

Fungus of the Lungs (Bennett).—The mycelium of this fungus is composed of long tubes, provided with partition walls and unequal articulated intervals, bearing several branches, which sometimes consist of one cell, set into the stem at the end of the last cell, and parted in the shape of a fork, sometimes simply separated into two or three elongations at their point of articulation. The spores are numerous. Bennett saw them lengthen and form tubes.

This fungus was found in the expectoration, in the cavities and their tuberculous matter, in a case of pneumo-thorax.

The Aspergilli species of fungi are apparently peculiar to certain parts only of the human body.

Thus, a species of Aspergillus was observed by Mayer in the external auditory meatus of a little girl affected with scrofulous otorrhœa. It consisted of round oval cysts, of the size of a cherry, the walls of which were fibrous, white externally, internally greenish and granulous. Viewed with a magnifying power of 300 diam. these granulations were shown to be organized productions.

The stem of this fungus is long, transparent, having little globules in its interior, and terminating with a small, swollen, round, and greenish little head, which sits like the cap of the fungus on a small inflation of the stalk (Robin, Atlas, iii. I.) It is covered with a layer of simple or double nuclei, or spores, on its free edge. Between the stalks are other filaments, which are deprived of mycelium, and spread here and there, isolated or in bundles. Amongst them are seen filaments in all stages of development.

Nail-fungi are perhaps other species of Aspergilli (Meissner and Virchow).

A copious plexus of variously entwined filamental fungi was discovered by Meissner on the nails of an octogenarian, which were broad and thick, strongly convex, resembling claws, striped with a yellowish-white or brownish colour, and which were moveable in their sockets; they were also characterized by having a soft and brittle, but not lacerated appearance, and by being fissile, like wood. The nails of all the fingers, excepting that of the forefinger of the right hand, were thus affected. The fungus was not found on any other part of the body.

Three instances of fungi on the nails of the toes are mentioned by Virchow, under the head "Onychomycosis."

Mucor mucedo, so called, but probably another species of Aspergillus (Robin), was found by Baum, Litzmann, and Eichstädt, in a cavity, in a case of inflammation of the lungs.

This parasite consisted of a black mass of filaments, interspersed with round globules, adhering to the walls of the cavern. Each filament had a process on the outer surface of the mass, terminating in an enlargement, surrounded by a row of oval cells.

Puccinia Favi (Ardsten) is, according to Robin, a parasitic fungus of rare occurrence; it is merely an accessory epiphenomenon, and when it does occur, is generally found on the fungus of Favus (Achorion Schöenleinii), or more frequently on epidermal scales. It may also occur in other diseases of the skin—as on the fine scales in pityriasis.

Next to human parasites, animal and vegetable:

Foreign bodies, as *dead* substances, in the human organism represent the last and most extreme degree of 'adventitiousness' in relation to it.

Their variety is endless: indigestible portions of food, animal or vegetable, as bones and fruit-stones; portions of dress; or mineral substances, as needles, a knife-point, shot and shell, bits of glass, tobacco-pipe, coins, rings, &c.

They are introduced, sometimes by the natural apertures and passages, as by the mouth, nostrils, auditory meatus, larynx, urethra, vagina, anus; sometimes by violence, as in gun-shot wounds.

Having been introduced, accidentally or designedly, the migrations of foreign bodies are very extraordinary; travelling through textures and organs, possibly to parts distant from that of their introduction. In some cases these migrations are obviously due to gravitation, in others not so, and the movements are less explicable. In their course, foreign bodies occasion mischief, either by injuring the mechanism of parts through which they pass or in which they become impacted; or by irritating such parts, provoking inflammation and its consequences. Eventually these bodies may be ejected through the natural passages, or discharged by suppurative inflammation. This effort of Nature failing or not supervening, more rarely they become invested with a capsule of inflammatory fibrin, consolidated; and being thus located and isolated, are tolerated for years or for life, with little or no inconvenience. Foreign to the last degree, as any such encysted body still is, *per se*, in relation to the human body; yet this organism has become reconciled, in a measure at least, to its presence, without ever acknowledging the incarcerated substance as a part of itself.

Taking a retrospective view of the foregoing Elements, I propose the following classification:—

ELEMENTS OF PATHOLOGICAL ANATOMY.

The Textures, Organs, and Fluids of the body, as compared with the standard of Health, may undergo and present—

ALTERATIONS OF DEGREE.

PHYSICAL.

Situation—Elevation and prolapsus.
Protrusions and herniæ.
Dislocations.
Hemorrhage, extravasations.
Number—Of double organs—*e. g.* kidneys.
Position—Versions—*e. g.* uterus—heart.

Configuration—Internal parietes of cavities.
Sacculations of hollow organs.
Inflexions of hollow organs.
External alterations of contour—
Flexions—of uterus, spine.
Expansions and bulging of cavities.
Retraction and depression of cavities.
Solutions of continuity—
Fractures, Wounds.

Colour — Changes.

Consistence, Elasticity } Induration, softening.

Size . . { Volume—rarefaction, condensation. Capacity—aneurism, stricture.

Weight —Hypertrophy, atrophy.

CHEMICAL CONSTITUENTS.

Increased, diminished.

STRUCTURE.

Hypertrophy, atrophy.
Plethora, anæmia.

Congestion, Determination, Inflammation } Local anæmia.

ALTERATIONS OF KIND.

MORBID PRODUCTS.

Supplemental Formations.

Plastic—Are Retrogressions of Healthy Structural Elements of Textures

(by) Arrests of their Developmental Conditions.

False Tissues . Connective tissue.

Deposits . . . Tubercle. Exudation-corpuscles. Pus, Lymph.

Growths—Non-infiltrating—Fibro-cellular tumour. Painful subcutaneous tubercle.

Fibrous tumour { fibro-nucleated. recurring-fibroid.

Fatty tumour and its varieties.

Cartilaginous tumour and myeloid variety.

Cysts—simple and proliferous.

Vascular tumour and its varieties.

Infiltrating—Cancer — encephaloid, scirrhus, colloid, epithelial.

Aplastic Formations.

Concretions.

Calculi.

TRANSFORMATIONS OF TEXTURE.

Substitute Tissues—Degenerations. Retrogressions of Structural Elements, by Relapse.

Fatty, Pigmentary, Fibrous, Amyloid (waxy lardaceous), Granular ? Calcareous ?

Non-substitution—Disintegration, Débris of Textures.

FOREIGN BODIES.

Parasites :—Animal—Insects, Arachnida, Annulata (worms), Infusoria.

Vegetable—Algæ, Fungi.

Dead-Substances—Organized, Unorganized.

THE PRINCIPLES OF CLINICAL SURGERY;

OR,

THOSE OF DIAGNOSIS, ETIOLOGY, AND PROGNOSIS.

PRELUDE.

The Surgeon at the bedside.—The preliminary Questions to be determined on behalf of Diagnosis.—Etiology, respecting internal causes, and their operation; and Prognosis.—Their Pathological character, and mutual relation to rational Therapeutics.—The *earliest* and *most exact* solution of the Questions alluded to, and the establishment of corresponding Principles, in each of these Departments of Surgery, by the Analysis of Pathology, will become apparent in the course of this Work.—The immediate bearing of these Pathological Principles on the Preventive and Conservative Practice of Medicine and Surgery re-explained and enforced.—The Analytical Method of Investigation, explained by reference to the Principles of Diagnosis.

When the surgeon stands at the bedside of a patient, he virtually asks himself these preliminary questions:—What is the structural condition, situation, and extent, what the origin and operation, what the course and tendency of this disease or injury? For example: is this case one of ulceration of the articular cartilages of the knee-joint, and is the ulceration limited to the margin of one, or more, such cartilages, or does it extend over it, or their, free surface; respecting the origin of this disease, is it primary, or is it secondary to either synovitis or caries; is its origin local, as from external violence, or does it proceed from a lithic acid (gouty) condition of the blood, or from any other constitutional cause,—of which it is only a local manifestation; does the ulcerative condition operate locally only—thereby interrupting the (mechanical) function of

the joint, or does its influence extend so as to induce constitutional disturbance, *e. g.*, hectic fever; and lastly, is its course and tendency to, or towards, reparation, with a sound articulation, or anchylosis, with a stiff joint? Again, to take an example of injury: is this case one of fracture of the neck of the femur; is its situation without or within the capsular ligament; in extent, is it complete—through the bone, or incomplete, and with or without entire rupture of that ligament; is the fracture due only to external violence, or has it originated in previous softening and absorption of the bone; is there but little functional derangement beyond inability to use the limb, or much constitutional disturbance—shock of injury—present; and lastly,—in respect of this fracture,—is its course and tendency to, or towards, bony union, with a useful limb, or ligamentous union with a useless appendage?

The detection and discrimination of diseases and injuries—in respect to their particular structural condition, situation, and extent—is Diagnosis; the detection of their origin, as constitutional or local conditions, and their operation as internal causes, is the most essential branch of Etiology; the foreknowledge of their individual course and tendency to or towards a favourable or an unfavourable issue, is Prognosis: and, in considering any particular case, the surgeon is imperatively compelled to solve the pathological questions involved in *each* of these preliminary Departments of Surgery, *before* he can fairly undertake to conduct its curative treatment, guided by *rational* Therapeutic Principles. The *earliest* and *most exact* solution of the questions alluded to, and the establishment of corresponding Principles in each Department of Surgery, will be shown in the following pages to emanate from Pathology by analysis. The Principles derived from this source also impart a Preventive and a Conservative character to the Practice of Medicine and Surgery—to Therapeutics—medical and operative.

A little reflection will show this result in a clear light, agreeably to the terms of that definition of Conservative Surgery and Therapeutics in general, which I advanced in the Historical Introduction.

Starting, as nearly as possible, from the earliest condition of the disease or injury, the Therapeutical indications to be fulfilled are proportionately less, not merely as to the kind of our interference, but also respecting the amount of assistance—the least amount only, it may be, being then required—to conduct the case to a favourable issue. To this end the equally essential importance of, as nearly as possible, the most exact knowledge is obvious, and needs no comment.

In the event of complications having already supervened, or of their being present from the very commencement,—as in many forms of injury,—a *relatively* early and exact knowledge of the structural condition, situation, and extent, origin and operation, course and tendency of the disease or injury, is still absolutely necessary to enable the surgeon to anticipate and circumvent *worse* complications, and thus to employ remedial measures, whether medical, operative, or both, which shall be *proportionately* Conservative.

If the question of diagnosis be delayed until a later period, or if, not being delayed, it be inexact, and not rectified until a later period; or again, if the origin of the disease or injury be undiscovered, and its operation, itself as an internal cause of other morbid conditions, be unknown; or lastly, if its particular course and tendency, and this also in the first instance, be unforeseen; then,—being proportionately hindered by all this delay, and misguided by all this inaccuracy respecting the preliminary knowledge of Clinical Pathology—when the Restorative Power is invoked, it will have become proportionately less and less responsive, and any kind or amount of Therapeutic assistance may prove inadequate to overcome the confirmed condition of the disease or injury, or be neutralized by the more and more complicated conditions which would then have to be overcome in favour of a successful issue.

And all this *anticipation*, and all this *exactitude*, turns upon an equal knowledge and application of Clinical Pathology. Pathological Diagnosis, knowledge of Etiology—in respect of internal causes and their operation, and foreknowledge of the particular

courses and tendencies of diseases and injuries—Pathological Prognosis,—must therefore, and in this order of investigation, necessarily precede the fulfilment of the guiding Principle of Rational and Conservative Therapeutics.

I therefore propose instituting an original analysis of the pathology of the various kinds of injury and (surgical) disease, with a critical exposition of its guidance at the bedside, in the first place, in order to discover the Principles of the *earliest* and *most exact* Diagnosis, Etiology, as explained, and Prognosis. Such preliminary knowledge of the structural condition, situation, and extent, origin and operation, course and tendency of the various kinds of injury and disease, will determine how far to rely on the provisions and resources of the Restorative Power, and concurrently indicate the kind and *least* amount of Therapeutical assistance—medical, operative, or both—which is then required;—thereby fulfilling the leading Principle of Conservative Therapeutics.

The analysis of Pathology, as applied to establish the Principles of the earliest and most exact Diagnosis, will be conducted as follows:—Firstly, I shall endeavour to establish certain Negative Principles—namely, the *incompetency* of Anatomy and Physiology, and the *insufficiency* of pure Pathology (functional disturbances) severally as guides to even an early and exact Diagnosis.

But Diagnosis is only the threshold of clinical inquiry, and these Negative Principles being acknowledged in this department of Surgery, will direct the mind aright towards Etiology and Prognosis. In subsequent portions of this work, I shall endeavour to determine how far (pure) Pathology is accessory and subservient to the earliest and most exact detection of internal causes, and their operation; and to a similar Prognosis of the course and tendency of diseases and injuries.

So far, analysis having resulted in Negative Principles, as regards Diagnosis, will conduct to Positive Principles, through the guidance of Pathological Anatomy, and its application *during life*, as severally illustrated by the earliest and most exact diagnosis of fractures, aneurisms, and dislocations. I shall then endea-

vour to establish the Principles of this standard of Diagnosis, by the further and systematic analysis of *Clinical* Pathological Anatomy.

To thus discover the *positive* and *comparative* diagnostic value of physical, structural, and chemical evidence, I shall select certain kinds of disease, from *General* Pathology—the 'physical,' the 'structural,' or the 'chemical' conditions of which are *not constant*, but variable in the same disease; or in which one or more of these conditions are *not peculiar* thereto.

The analysis of diseases, thus constituted, will show:

Firstly. How far each of these conditions is peculiar to any given disease, and represents the *essential* morbid element.

Secondly. How far each condition may be relied on, to identify and distinguish diseases; or the value of each condition—*absolutely* and *relatively*, as a *method* of diagnosis—the earliest and most exact.

Thus, analysis being applied to diseases of nutrition, in respect of certain quasi-malignant varieties of non-malignant growths—tumours—illustrates the insufficiency of 'physical' characters on behalf of such diagnosis.

Extension of the analysis to other diseases of nutrition—*e.g.*, to various species of growths or tumours—discovers the superior value of 'structural' conditions on behalf of such diagnosis.

Further extension of this mode of investigation to the entire range of diseases of nutrition, as manifested by all kinds of growths, deposits, and false tissues, shows the potential value of 'chemical' conditions on behalf of such diagnosis.

The analysis of a large section of Pathological Anatomy having yielded these results, their guidance collectively at the bedside represents the Principles of the earliest and most exact Diagnosis.

That 'physical' characters are most readily recognised at the earliest period of a disease, and possibly therefore at a time when the morbid condition itself is least complicated, and most remediable by the simplest Therapeutic measures; but these characters are nevertheless the most equivocal and inexact signs. Unaided

physical diagnosis therefore fails to fulfil the (diagnostic) requirements of Conservative Therapeutics.

The diagnostic value of certain physical characters, as signs of disease, is moreover practically further restricted, by the difficulty or impossibility of recognising them during life.

Changes of 'minute structure' can be detected, during life, at an early period, by puncturing growths and deposits situated near the surface of the body, and examining with the microscope these and all other morbid products found in discharges and secretions issuing from internal parts and organs. Moreover, the alterations of structure thus detected are more constant, in any given case, than those of physical properties, and are therefore more exact signs of the particular morbid condition in question; and being also discoverable at an early, if not the earliest period, before the supervention of complications, supply the chief method of diagnosis, whenever this method is practicable.

If, however, the results of analytical inquiry demonstrate the superior value of minute structural characters, yet experience shows that they *apparently* suffer the disadvantage of being inconstant; for the same disease may present different phases of minute structure. No two growths, for example, that are apparently alike, agree exactly in respect of their minute structure. It becomes, therefore, an object of supreme importance to reconcile these discrepancies, if possible, by a fair interpretation of facts, and thus to extend the value of minute structural characters, as supplying the more exact method of diagnosis.

Now the pathological law of Structural Retrogression* represents plastic morbid products to be merely so many rudimentary conditions of analogous healthy tissues—*i.e.*, of their structural elements, by arrests of their development. By this law, the science of healthy histological anatomy is applied to interpret plastic morbid products, which are morbid, not by virtue of their structure being peculiar, but only in respect of their pathological course and tendency.

---

* P. 92 *et seq.*

In conformity with this law, I shall hereafter show the identity of many minute structures that are apparently distinct, but which are only different stages in the development of one and the same species of structure; and thus endeavour to reconcile the discrepancies of microscopic examination, as the method of more exact diagnosis.

Alterations of 'chemical composition' represent the most essential condition of disease, but they are also the least certainly detected and identified, and least open to detection.

Many collateral *circumstances* may singly or collectively verify the evidence derived from Pathological Anatomy.

Diagnosis may be corroborated by Pathology—*i.e.*, the functional disturbances accompanying the particular disease or injury; also by connecting the morbid condition with its external cause, and by the characteristic effects of the Therapeutic measures employed.

For example, the fact of an individual having been exposed to malaria, is evidence presumptive that he may have ague; whilst the fact of his disease disappearing during the administration of quinine, alone is, among *other* evidence, corroborative of that disease being ague.

But the diagnostic value of all such *circumstantial* evidence is regulated by its more or less *constant* and *exclusive* association with the disease or injury which is sought to be identified.

Viewed in this light, the evidence of Pathology—functional disturbances—of external causes, and the effects of therapeutic measures, possess different degrees of diagnostic value; but the assurances derived from all these resources are, at the best, equivocal, and therefore misguiding. Here it is that Pathological Anatomy, applied during life—Clinical Pathological Anatomy, comes to our rescue.

By an appeal to the 'physical' properties of organs and textures, as discovered *during life*, we can *partly* corroborate or rectify our interpretation of mere functional symptoms.

By the 'structural' character of morbid products and secretions discharged from the natural passages—as the mouth, œsophagus,

stomach, and intestines; the lungs, urinary bladder, kidneys, uterus, and vagina; those also which are externally yielded by or through the skin, or procured by puncture, as from tumours; possibly, in some cases, by 'chemical' analysis of some such materials, we gain *direct* insight into and evidence respecting the structural conditions of *most* internal organs and of morbid products issuing from them.

Such application of Pathological Anatomy, therefore, directly indicates the *exact* structural condition, the situation, and even the extent, of the disease; and by virtue of this qualification, as a method of diagnosis, is more trustworthy than the evidence of Pathology, or any other collateral testimony.

The superiority of physical diagnosis, by virtue of the *earliness* with which the physical characters of a disease become obvious during life, is cancelled by its inferiority in respect of *exactitude*, as compared with structural diagnosis.

Duly weighing all these considerations, we arrive at this conclusion—that it is only by careful and repeated clinical observation of the 'physical,' the 'structural,' and, if possible, the 'chemical' conditions,—which taken *collectively*, are the *constant* signs of disease; by a similar observation of any collateral *circumstances* of evidence, *i.e.* alterations of function; precurrence of external causes, and the effects of therapeutic measures; and by connecting *all* this evidence during life, with certain pathologico-anatomical alterations, as demonstrated by the scalpel, the microscope, and possibly, by chemical analysis after death,—that we can rationally hope to establish the *earliest* and *most exact* diagnosis.

This connexion, oft-recurring, at length begets self-confidence; so that with the accumulation of such experience our diagnosis, although determined during life, and therefore by evidence not infallible, yet having been repeatedly verified by *post-mortem* examination, now supplies a proportionately sure *basis* for Therapeutics.

THE PRINCIPLES OF DIAGNOSIS,

SHOWING

THE *EARLIEST* AND *MOST EXACT* DETECTION AND DISCRIMINATION OF THE STRUCTURAL CONDITION, SITUATION, AND EXTENT OF DISEASES AND INJURIES.

NEGATIVE PRINCIPLES.

Chapter I.

Anatomy and Physiology are severally incompetent Guides to an Early and Exact Diagnosis.—This Principle is illustrated by the Diagnosis of Wounds, and by that of Burns.

Were I about to advocate the claims of Anatomy to the confidence of surgeons, there would be no difficulty in finding ample proof of their full acknowledgment in the numerous works on Surgery which have been issued since the time of Vesalius, the father of human descriptive Anatomy.

Formerly the error prevailed—not yet overcome—that Anatomy is the very foundation of Operative Surgery; and that without the light of its guidance in surgical operations, the simplest proceeding —say, puncturing a pointing abscess—would be no less perilous and culpable than making a stab in the dark. *How far* Anatomy is entitled to this claim, I shall hereafter amply consider; but I shall now endeavour to determine the value of purely anatomical conditions to fulfil an exact Diagnosis.

This inquiry is most forcibly illustrated by the diagnosis of those lesions which most obviously and conspicuously contrast with the standard of healthy anatomical conditions, and of which therefore the distinctive characters, as suggested by anatomical

knowledge, would be presumed to be most unquestionable. Such are the various *kinds* of wounds, fractures, aneurisms, and dislocations; all of which lesions would be presumed, by virtue of their apparently anatomical nature, to exhibit the guidance of anatomical knowledge in respect of their diagnostic characters. But it might also be presumed that the exact diagnosis of Wounds, more especially, would illustrate the (diagnostic) value of anatomical distinctions, and for this reason:

A simple *incised* wound is a sudden and recent breach of continuity of the soft tissues in any part of the body, and effected by a sharp cutting instrument. Such lesion is accompanied with more or less pain and hemorrhage; and the lips of the incision gape more or less, owing to the elastic retraction of the cut tissues, aided, it may be, by muscular contraction and the weight of the parts divided.

Guided by Anatomical considerations, we should infer that incised wounds would differ in their pathological import and tendency, according to the particular tissues or parts which are divided and exposed; and that they ought, therefore, to be distinguished by virtue of the anatomy of the various regions in which they occur. The exact diagnosis of incised wounds would thus be determined by their situation, and their surgical importance also measured by the particular *parts* divided. In the early days of Surgery, Anatomical knowledge—coarse indeed, but still this kind of knowledge—suggested the differential characters of all wounds.

Richard Wiseman, Serjeant-Chirurgeon to Charles II., and who is justly regarded as the father of English Surgery, acknowledged this method of Diagnosis. "Those," says he, "that look most like *essential* differences, are they which are taken from the subject, in which the wound (which is itself but an accident) inhereth, viz., from the flesh, skin, brain, nerve, tendon, artery, vein, gristle, bone, &c.; all which being the several subjects of wounds may well be allowed to specify them; and so much the rather, because from the nature of them we raise our greatest indications of *altering* the method of cure."

These distinctions are quite true within certain limitations. A sabre-cut in the thigh, dividing the superficial femoral artery, vein, and branches of the crural nerve, represents a certain degree of danger, by the very nature of this lesion, and apart from the influence of other circumstances; while an incised wound in the abdomen, and extending to the intestines, implies another degree of danger, and this, again, differs from that of a wound in the chest, and implicating the lung, or of a wound in the throat, and passing through the larger blood-vessels. Incised wounds might so far be distinguished by reference to their anatomical characters; but the usual course and tendency of *all* such wounds is to one and the same issue—'union by adhesion' of their sides, when neatly and evenly adjusted in contact.

This mode of healing by the "first intention," as it is sometimes incorrectly named, is the prevailing characteristic of all *incised* wounds, under favourable circumstances. Their pathological significance may vary in *degree*, according to the extent and functional importance of the parts divided; but all such lesions *tend* to unite by the first intention, without loss of substance or other disfigurement, without also much, if any, accompanying sympathetic fever. True it is that wounds of the throat rarely, if ever, admit of primary union; and this, owing to the frequent motion of the parts during efforts of deglutition: but wounds of the chest and abdomen are no exception to the general rule of primary union. This fact was well known to the surgeons of old. Cases are recorded of wounds of the thorax, in which not only was the cavity of the chest opened, but also a portion of lung protruded; which being cut off, adhesion took place between the lung and the pleura in the situation of the wound. Tulpius mentions the instance of a man who received an extensive wound just below his left nipple. On the third day, three inches of lung protruded. The poor man underwent two days' journey to Amsterdam, for the purpose of obtaining hospital relief. The protruding lung had begun to mortify; it was ligatured and removed with scissors, and the portion thus removed weighed three ounces. The wound,

however, healed in a fortnight, and the patient recovered his usual health, with the exception of an occasional cough. He survived the accident six years, but led a dissipated life, which perhaps induced his death prematurely. Post-mortem examination showed that the lung and pleura had *adhered* and *united* in the situation of the wound. Wounds of the abdomen are no less subject to the law of primary union. John Bell* states that M. Rosiere, a French surgeon in Lower Normandy, put back the intestines into the belly of a peasant boy, who had been gored by a bull. The poor lad came the next day on foot three miles from his village, carrying in the skirts of his shirt and in his hands a great bundle of the intestines which had protruded again; they were again replaced, the wound was neatly sewed, and the boy being kept quiet for some time, made a very perfect recovery. In this case, the cavity of the abdomen had been opened extensively, but the intestines themselves were not wounded.

That the abdominal viscera also may be wounded extensively, and yet adhere, is attested by a conclusive instance which occurred in the practice of Mr. Littre, in 1705, and is narrated in John Bell's admirable work on Wounds. A madman stabbed himself with eighteen deep wounds in the belly, and of these eighteen wounds, made with a long and sharp-pointed knife, eight penetrated the cavity of the abdomen. The vomiting of blood, and clots of blood passed by stool, proved that the wounds touched the stomach and intestines; yet in two short months, the man had entirely recovered. But the man's madness returned, and about eighteen months after (recovery from the first injuries) he threw himself from a high window and died upon the spot. And, observes Bell, here lies the important point, what did the post-mortem examination reveal? First, that the liver had been wounded, but had *adhered* in its middle lobe to the inner surface of the peritoneum. Secondly, the jejunum had been wounded just below the stomach, with an incision half an inch in length, across the

* Nature and Cure of Wounds, 3rd Edit., p. 231.

gut, and this portion of intestine lying deep, was not pressed against the internal surface of the belly, but was kept in close contact with a contiguous turn of the jejunum. The two turns of intestine *adhered* to each other; on the one intestine was the scar of the wound, while the other portion of intestine to which it had adhered was sound. Thirdly, the right side of the colon had been wounded with an incision an inch in length, but had *united* and *adhered* to the inner surface of the peritoneum, by about a score of long thread-like tags of false membrane, issuing from the inner surface of one of the largest scars in the abdomen.

These records, authentic as I believe they are, incontestably verify the law, that incised wounds of all parts of the body, however apparently dangerous, have a natural tendency to one and the same favourable issue—primary union.

If therefore we were guided by Anatomical characters in our diagnosis of incised wounds, and distinguished them according to the anatomical nature of the parts which are divided, we should thus draw an artificial distinction between lesions, which are naturally associated, by their tendency to the same happy issue; and moreover, we should overlook the operation of the great law of adhesion, as the first principle of therapeutic guidance in the management of wounds. This argument illustrates also the insufficiency of functional, or Physiological, distinctions in diagnosis. All textures and organs—skin, muscle, tendon, blood-vessel, bone, intestine, lung, &c., however various their functional nature and importance—are associated by the law of primary union, and therefore should be so regarded in the surgical treatment of their wounds, irrespective of the particular functional character of the parts that have been wounded.

But now let us suppose that a wound is inflicted by a wedge-shaped weapon, say a bayonet, thrust into the thigh. The same textures perhaps are injured as by a deep incised wound. The main artery may be lacerated, and branches of the crural nerve may not have escaped. The muscles are more or less divided, especially if the wound has an oblique direction, and the skin be

penetrated. This anatomical lesion coincides with that of a deep incised wound of the thigh, inasmuch as we will suppose that the *same parts* are divided. But the artery and nerves are *torn;* the muscles, perchance, sorely *bruised* by the forcible entry of a distending weapon, and where it penetrated, the skin presents a ragged aperture, having inverted edges, and encircled by an areola of *ecchymosis.* Such are the usual characters of a *punctured* wound; and its natural tendency is (unlike that of an incised wound) not to union by adhesion throughout its course, but to 'suppuration,' often profuse, and 'sloughing,' accompanied with the emaciation, sweating, and trembling weakness of hectic fever.

Trusting, however, the guidance of Anatomy alone, and apart from pathological experience, this punctured wound would be supposed to resemble an incised wound of the same parts, from which it differs in its course, tendency, and more fatal termination. Dissimilar lesions would thus be associated by the anatomical method, an error which also, *pari-passu,* would result from the guidance of Physiology; and I have shown how that lesions of a similar nature might thus inadvertently be regarded as essentially different. Anatomy and Physiology are, therefore, blind guides in Diagnosis, being quite incompetent either to recognise *identity,* or to distinguish *differences,* of pathological character.

The same general facts and inferences are true also of other varieties of *contused* and lacerated wounds. A punctured wound may not be contused, or at least only in an unappreciable degree, as when made by a sharp-pointed and double-edged knife; but this lesion is necessarily lacerated, owing to the tissues being detruded before the point of the instrument, and the more so were the instrument blunt pointed. A gun-shot wound is both contused and lacerated, and its history well illustrates the insufficiency of anatomical and physiological considerations to fulfil an exact diagnosis.

A man is shot in his thigh, and the ball passes through, wounding, in its course, the main artery and accompanying saphenus nerve; nevertheless, but trifling hemorrhage occurs at the time, and the injury occasions, at first, but little pain to arrest attention.

The entrance opening made by the ball is small, circular, inverted, and surrounded by a rim of lividity; the exit opening is perhaps larger, ragged, everted, and begirt with a deeper and darker circle of ecchymosis. Such are the ordinary characters of gun-shot wounds. Would it not be supposed from mere anatomical or physiological considerations of the *parts* injured, that the comparatively insignificant external wounds made by the ball would heal by the first intention, no less readily than the lips of a large, slashing, incised wound of the thigh, and in which the main artery and branches of the crural nerve are also divided? Could it be anticipated that the invariable tendency of a similar gun-shot wound is to profuse suppuration, sloughing, and hectic fever? Yet, so constant is this distinctive pathological character (one which could not have been predicated by mere anatomical or physiological considerations), that it may supervene when least expected, and when the wound has almost healed.

On the retreat of the army from Fuente Guinaldo, in 1812, a skirmish took place at the convent of Saca Farte, between the advance of the French and the fourth division of the British army, aided by the cavalry. The wounded were given in charge of Mr. Guthrie. Among them was a man, a stout, handsome soldier, who had been shot through the thigh, the ball entering below the femoral artery, and passing through and outwards close to the bone. This wound progressed favourably for a fortnight, so much so that the man actually got up and walked about. Mr. Guthrie saw him at 1 P.M., and as he was standing, desired him to keep himself quiet. He answered, that he felt quite well. But little or no inflammation had supervened about the wound, and the limb was soft. In the evening, the man was reported to be suffering some pain, and an opiate and poultice were ordered. He died early in the morning. The limb was carefully examined within twenty-four hours of the man being in comparative health. "Inflammation came on in the night, internally, deep, and hardly affecting the skin with redness. On dissection, the thigh appeared swollen, although not particularly so; but on cutting deeply through

the fascia in the course of the wound, the whole thigh was so stuffed or gorged with blood, that the texture of the parts, muscular as well as cellular, was soft, and readily gave way to a moderate pressure of the fingers; I (says Mr. Guthrie) can only compare it to the appearance of a part just falling into a state of gangrene."* This case is open to criticism. It may be suggested that perhaps the swelling gorged with blood was a false aneurism, resulting from the ball having grazed the femoral artery, in the first instance; and that sloughing of the vessel and secondary hemorrhage occurred at about the time (fifteenth day) when these consequences usually ensue. The condition of the main artery should have been recorded; but one or more similar cases of apparent gangrene occurred at Salamanca, and I am not inclined to believe that Mr. Guthrie overlooked the possible contingency to which I have alluded, or that he mistook a false infiltrating aneurism for bloody succulent gangrene.

The history of this case may, therefore, be admitted as conclusive evidence that mere Anatomical or Physiological considerations of the nature of parts which have been wounded cannot supply an exact method of Diagnosis; that, guided by such knowledge alone, we should be induced to associate lesions different in themselves—*e. g.*, gun-shot (contused) wounds and simple incised wounds of the thigh—an error no less fatal than the creation of an artificial distinction betwixt incised wounds as they chance to be made in different parts of the body.

At length, the *true* criterion of *resemblance* and *difference* between Wounds came to light through Pathology.

The great law, to which I have already alluded, of union by adhesion, was established chiefly by the observations and experiments of John Hunter, and its operation was regarded by him as a process of "adhesive inflammation." All *incised* wounds were found to be subject to this law, and to possess this attribute of healing by "the first intention," without loss of substance or dis-

* Gun-shot Wounds, p. 96, 3rd Edit., 1827.

figurement. The old artificial distinctions inculcated by Wiseman and others, as to the essential differences of wounds in different parts, gradually became obsolete; for wounds of all parts, whether of skin, flesh, arteries, or veins, nay, even those of the lungs, intestines, and other viscera, were observed to alike naturally tend to obey the law of union by adhesion; and thus it came to pass that lesions whose nature and tendency are essentially the same were no longer regarded as essentially different.

To the acute penetration of Hunter is also due the merit of having more clearly defined, although not of having first discovered, the true course and tendency of all contused and lacerated wounds; whether punctured, as they are occasionally, or, more commonly, gun-shot wounds. He showed that a 'slough' follows the course of a ball, or the passage through the flesh of a blunt penetrating instrument. In the case of a gun-shot wound, Hunter believed that a slough does not always follow to the same depth, nor in every part of the same wound. John Bell subsequently maintained that the whole course of such a wound is mortified, and this statement was supported by Dupuytren's observations;* but more recently Guthrie has, I think, justly remarked, that no one has ever *seen* from a superficial or deep-seated wound of three inches in length, with two openings, a slough or sloughs of the same extent as the wound.† The observation as originally made by Hunter is therefore confirmed.

But should it be disputed whether or not a slough extends *throughout* the whole length of a gun-shot wound, nevertheless its presence there, and in the track of many punctured wounds, cannot be gainsaid. About the fifth day of a gun-shot wound, the slough begins to loosen from the margin of either opening, if two exist, and the line of demarcation between the living and dead tissues is clearly visible; about the tenth day, the slough or sloughs may be seen hanging out of the wound, and come away in the dressings. A tubular casting of slough had hitherto *inter-*

* Clin. Chir., t. ii., p. 450. † Op. cit. p. 61.

vened between the living tissues surrounding the course of the wound, and prevented its union by adhesion.

This one pathological condition altogether changes the course and tendency of a wound, the anatomical and physiological conditions of which may be the same as those of an incised wound. For example, a gun-shot wound and an incised wound of the thigh, in both of which we will suppose that the same parts have been divided, are very different lesions. Guided by Anatomy or Physiology alone, we should be led to regard them as similar lesions, but the *Pathology* of contused wounds at length corrected the incompetency of these methods of diagnosis. The Anatomical method of Diagnosis which formerly prevailed was abandoned, and wounds were henceforth distinguished by their Pathological course and tendency, either to union by adhesion, or to sloughing.

These results of inflammation became the grand criterion of distinction, and the pathology of inflammation gave importance to circumstances which, in the opinion of the old surgeons, were frivolous. Distinctions of wounds, says Wiseman, taken from the weapons, whether sharp or blunt, sword, rapier, or bullet, cut, prick, or tearing, are but accidental. Yet such distinctions were found to be all-important in the modern diagnosis and corresponding denomination of these lesions: for the terms—*incised, punctured, contused,* and *lacerated,* point to the two grand distinctions of 'adhering' and 'sloughing' Wounds.

I have purposely omitted "poisoned wounds," because they are not wounds properly so called. Their relative importance is not measured by the nature and extent of the local injury, but by the introduction of anything which "holds enmity with the blood of man." Should 'inoculation' not be accomplished, the lesion ranks as an ordinary wound—punctured, contused and lacerated, or incised; and the most severe wound, if it bleeds freely and cleanses itself, may prove the least hurtful; while the most trifling puncture may convey a fatal dose of the poison, whatever it be. Thus, the bites of rabid dogs and cats are of themselves contused and lacerated wounds, and not necessarily poisonous,

although one person out of a number bitten by the same animal may become affected, showing that a poison has been introduced. Dr. William Hunter gives an account of twenty persons who were bitten by the same mad dog, yet only one out of the whole number was afterwards attacked with hydrophobia. On the other hand, a cut or mere prick received in dissection sometimes causes death, and even the sting of a bee or wasp occasionally proves fatal. Dr. Gibson mentions the case of an old lady who died in a quarter of an hour from the constitutional disturbance induced by the sting of a wasp; and also an instance of death from a bee having been accidentally swallowed in a piece of honeycomb.

To conclude—the whole history of poisoned wounds, so called, renders them inappropriate illustrations of the incompetency of purely Anatomical and Physiological distinctions to determine an exact Diagnosis; but the history of all other wounds shows that by the guidance of these distinctions alone we should be led—

Firstly. To dissociate those wounds which are of similar nature, although affecting different parts of the body.

Secondly. To associate those (wounds) which, although affecting the same parts, are of dissimilar nature; namely, incised, punctured, and contused, including lacerated wounds.

The incompetency of Anatomical and Physiological knowledge alone for the purpose of exact Diagnosis, is further and fully illustrated by considering the effects of Burns and Scalds.

Formerly, these lesions were distinguished and arranged according to the different degrees of disorganization they produce—inflammation, suppuration, sloughing, and ulceration. Fabricius Hildanus, Boyer, and Dr. J. Thomson, observed only three gradations of disorganization; Heister and Callisen recognised a fourth; but Dupuytren* was dissatisfied with both these classifications, on the ground that they regarded only the intensity of the effects of burns, considered in a general way, while the nature of the organs affected—the textures injured or destroyed, were altogether disregarded; and maintained that, if a classification of burns is

* Leçons Orales de Clin. Chir., 1832, tom. i., p. 423.

to be established with clear ideas and upon correct principles, its foundation must be—the effects of heat on different kinds of organs. Dupuytren therefore proposed the following classification:—Burns producing—1stly. Erythema, or simple reddening of the skin. 2ndly. Vesication, by raising the cuticle in blebs, filled with serum. 3rdly. Incomplete destruction of the skin. 4thly. Complete destruction of the skin, extending down to and involving the subcutaneous cellular tissue. 5thly. Conversion of muscles, nerves, vessels, and other soft tissues into eschars, to within a variable distance from the bone. 6thly. Charring and complete disorganization of the whole substance of the burnt part. This classification recognises not only the various degrees of disorganization which burns present as they extend from the surface to deeper parts, beginning with erythema and ending with charring; but the various kinds of tissues—*i.e.* anatomical differences—are also recognised as grounds of distinction. Thus, burns of the skin, of the cellular tissue, of the muscles, nerves, vessels, and so forth. If, however, the burnt appearance of each of the various tissues should present distinctive characters, does the *depth* of a burn, as it successively involves each tissue, correspond with a certain kind and amount of functional disturbance, and its probable issue, so as to supply data whereby a natural classification of these lesions may be established?

To decide this question, I cannot do better than narrate some of M. Dupuytren's own clinical Observations. And first of the *deeper* burns—namely, his 4th, 5th, and 6th degrees.

M. Dupuytren's 14th Observation was of a burn to the 4th degree of the whole upper extremity. The suppuration was abundant, yet the patient *recovered.*

The 13th Observation was of a burn to the 5th degree of the external part of the left shoulder, and to the 6th degree of the left side of the face. This deep burn was followed by the destruction of a portion of the parotid gland, by salivary fistula, by necrosis of a portion of the bone 'de la pommette' and of the zygomatic arch; yet this patient also *recovered.*

The 12th Observation* was of a burn to the 5th degree of the right hand. The amount of injury sustained was most severe, while the clinical history of this burn contrasts favourably with that of superficial burns, which I shall presently refer to.

Clinard, aged thirty, a servant, of good general health and constitution, was afflicted with a mental malady, the result of violent grief. Under treatment she recovered her reason, but the dejection of spirits continued. Finding herself alone one evening, she made a very large fire in the kitchen grate and put her hand into the burning charcoal. By chance some one came to her, when she appeared very agitated, but did not move. These circumstances, and the smell of the burning flesh which pervaded the kitchen, aroused the madness of the unhappy creature. With considerable trouble she was dragged from the hearth and removed to her bed. She uttered cries of agony during the night, and begged that they would end her days, as she had nothing to live for. The next day she was removed to the Hotel Dieu.

The right hand appeared burnt to the very bone; on every part of it were black eschars—hard, thick, and separated from each other by crevices, which yielded no blood. These eschars extended to the middle of the metacarpus; while the other portion of the hand was covered with a large phlyctena full of serum. A bright red ring encircled the wrist. The radio-carpal articulation was moveable, the fingers and the thumb were bent upon the hand; one of the joints of the ring-finger, and one of the little finger, were open. The patient was much excited, the countenance animated, the eyes were immovable. Delirium continuing, a strait-waistcoat and venesection were had recourse to. Topical dressings were used. The third day she continued in the same state. Blood was let from over each mastoid process. On the fourth day she was no better. A purgative lavement was ordered. The seventh day brought some improvement. The eschars on the palm of the hand, and on the back of the fingers, began to separate, and there was hope that the

* Op. cit., tom. i., p. 499.

tendons might be saved; the matter, which escaped in but small quantity, was fetid.

On the eighteenth day the cries of the girl had ceased; she sang in a low voice, and spoke in a subdued tone, and if her attention was much excited, she would reply. The mental malady had completely disappeared on the thirtieth day; and during the last forty-eight hours the invalid was fretting for what had befallen her, but had no recollection of the occurrence, and her astonishment was great when she was told all the circumstances of her illness.

The report thus concludes:—The extent of the burn is well defined; the last phalanx of the ring-finger and finger "auriculaire" have fallen off, as if eschars; and cicatrization has begun here and there. Many small portions of tendons having sloughed away, have been removed with the dressings, and the fingers are contracted. They were placed on a board, so as to gradually extend them, care being taken that they were not in contact, in order to avoid adhesions.

Since this event, observes M. Dupuytren, any accident may admit of cure. The girl had entirely *recovered* by the 20th of February, being 103 days after the accident. No signs of insanity were apparent, and on the 5th of March the patient left the hospital quite well.

It is impossible to escape from the conclusion which these and similar cases suggest. Here are instances of deep burns to the 4th, 5th, and 6th degrees, extending successively through tissues having *very different anatomical* characters, and occurring in various parts of the body, these lesions being accompanied with the constitutional disturbance of shock and reaction in their various phases; and yet we observe Nature detaching the eschars, slowly but successfully separating the dead from the living, and eventually restoring the balance of the constitutional functions, with perfect *recovery*. Who can venture to affirm, after such experience, that "the nature of the parts affected, the textures injured or destroyed," that, in fact, Anatomical distinctions have aught to do with a na-

tural classification of burns, or lead to their *exact* diagnosis? In like manner we experience the incompetency of Physiological distinctions. The textures which are successively destroyed by a deep burn, serve *very different functions* in the animal economy,—some mechanical, some more essential to life, and yet the relative importance of burns cannot be estimated by their depth—is not proportionate to the functions of the parts destroyed. Nor is the guidance of Anatomy or Physiology competent to fulfil an *early* diagnosis; for a diseased condition, or injury—such as a burn, being a deviation at least, if not a further alteration, from the healthy standard—structurally and functionally, cannot be announced by that which is lost.

Nor, again, is the surgeon concerned with the Pathologico-anatomical differences of burns. He estimates not the effects of heat on the body, by observing the different degrees of disorganization produced in the various textures, as the geologist or mineralogist examines specimens of igneous rocks; that this eschar is yellow and hard, and that that is black and brittle. The insignificance of pathologico-anatomical differences (no less than of those derived from anatomy and physiology) will become more apparent, and the basis of the most exact and early diagnostic distinctions of burns supplied by observing their *Pathology*, as exhibited in the following cases of more *superficial* burns.

M. Dupuytren's 8th Observation relates to burns of both feet, from the 1st to the 4th degree, occasioned by a foot-bath, and *death* resulting on the seventh day.

A young woman, seventeen years of age, having been disappointed in love, attempted suicide by the fumes of burning charcoal. She so far succeeded as to become insensible, when she was discovered and restored by appropriate remedial measures. Among others a foot-bath was used, but the feet were accidentally plunged into boiling water, in which they remained for half an hour, the poor girl being insensible. She then complained of the heat of the water, and that her feet were numbed. She was at once removed to her bed. The next day her feet were found to

be scalded nearly up to the malleoli; the toes had lost their cuticle; yellow, hard eschars were seen up to the ankle-joints, and the lower parts of the legs showed the limits of the burn by numerous phlyctinæ, which were filled with a reddish serum. Above these the skin was reddened, slightly swollen, and very painful.

In three days the symptoms of asphyxia had entirely subsided, but the shock of the burn continued. The fifth and sixth days brought with them no improvement, either local or constitutional; obscure fluctuation was perceptible over the right leg, the inflammation had travelled upwards to the knee and the thigh; delirium supervened, and death followed on the seventh day.

It may be urged that this case was *complicated* in its origin with asphyxia, and in its issue with erysipelas. I therefore subjoin—

Dupuytren's 4th Observation, which was of burns to the 4th degree in many parts, by boiling soup and by fire; *death* following during the period of reaction at the end of the fourth day:

—— Bison, aged forty, affected for many years past with epilepsy, was sitting near the fire, when, being seized with a fit, she fell upon a pot of boiling soup. The left side of the face and the whole of the right hand were burnt to the two first degrees; the left side of the neck, and upper part and left side of the chest, which had come in contact with the fire, were burnt to the 3rd and 4th degrees.

The usual constitutional disturbance followed, and prevailed, by terminating fatally on the fourth day.

Mr. Travers* narrates the following case, of which, for my purpose, I omit the treatment:—"A lad, carrying a pail of hot soup upon his head, fell, and scalded his right arm, face, neck, and side of his chest as far as the margin of the ribs. The accident happened at about 10 A.M. The cuticle was destroyed, but the cutis appeared sound. In one hour his pulse was 104 and sharp. He complained of excessive thirst, and had constant shivering as

* Constitutional Irritation, 1826, p. 90.

from cold. Half-past 1 P.M., shivering continues; pulse 112, smaller and feeble. 10 P.M., makes no complaint, but is restless and thirsty; and although not actually in a state of delirium, approaches near to it; upon being asked questions, he makes no reply; the shivering has nearly subsided, and the surface is recovering warmth; pulse 136, thrilling feebly. Second day, 10 A.M., was restless and delirious the greater part of the night, but has been for some hours in a dozing, half-comatose state, from which it is difficult to rouse him. He appears attentive to questions; expresses no pain; breathing rather hurried; temperature natural; pulse small and feeble, scarcely perceptible at the wrist, rather creeping under the finger than distinctly pulsating, about 150 in the minute. The cutis has lost its florid colour, and seems hard and insensible, with little surrounding inflammation. At 10 P.M. he expired, having remained much in the same state as last reported, and survived the injury about forty-eight hours."

Here, then, is a series of cases wherein burns, *superficial*, but *extensive*, were accompanied with shock and collapse so severe as to terminate *fatally*, and this also so speedily as to have almost anticipated the commencement of reaction. Yet the skin was alone involved, the cuticle alone destroyed. These cases therefore corroborate the conclusion we gathered from the history of previous ones—that the depth of a burn, as suggesting the different kinds of tissues involved, and their functions respectively, is no measure of the vital importance of the lesion—that Anatomy and, by parity of reasoning, Physiology are not guides to an exact and early diagnosis of burns; nor, therefore, to their natural classification.

It may be said that this conclusion is not, perhaps, *generally* true, seeing that it is derived from the comparison of only a limited number of burns. But the argument is cumulative. The cases I have adduced to support it are not peculiar, and they all point in one and the same direction. The more superficial the burn, if only extensive—the more the skin *alone* is involved, the more urgent is the constitutional disturbance, and the more dangerous and significant is the burn.

A negro employed at the Bains Vigier, in Paris, wishing on one occasion to warm his limbs, which were benumbed with cold during a rigorous winter, immersed himself in a bath heated to a high temperature. In a short time he experienced a general feeling of uneasiness with acute pain in the skin. He was immediately withdrawn and carried to the Hotel Dieu, where he expired in thirty-six hours! It is reasonable to suppose in this case, that although the water was heated to a high temperature, yet it was not at the boiling point, and that it acted only as a general rubefacient, without raising or destroying the cuticle; and yet here is a burn of only the 1st degree, but very extensive, and therefore accompanied with a shock of the nervous system, so sudden and overwhelming as soon to have proved fatal.

We have already seen the value of Pathologico-anatomical distinctions, in the early and exact diagnosis of wounds; but the distinctions of burns can alone be estimated by Pathology,—by the kind and degree of *functional* disturbance they induce,—only, therefore, by observing the living, moving, sentient body, "to mark its hunger and thirst, its sleeping and waking, its heat and its cold, to hear its complaints, and to register its groans." Clinical observation of the 'functional disturbances' pertaining to burns and to other injuries and diseases, brings to light a new order of phenomena, which—unlike those that have hitherto chiefly occupied our attention in this chapter—are *only* to be studied *during life*.

A *general* inquiry of critical moment here opens up. How far are Functional disturbances competent to determine the earliest and most exact diagnosis; and as compared with the evidence of Pathological Anatomy, so far as it is available during life?

To anticipate the result of this inquiry, I may state that, while the former will prove to be equivocal and misleading, the latter is an infallible guide.

CHAPTER II.

NEGATIVE PRINCIPLES OF DIAGNOSIS (CONCLUDED).

Pathology—Functional Disturbances only—is an insufficient guide to an early and exact Diagnosis. Functional disturbance does not *invariably* accompany, and does not at an *early* period accompany, injury or local disease; and the same disturbance of function is no *measure* of the structural lesion existing—may also accompany the same disease or injury simultaneously in different *parts* of the body, or may accompany very different *kinds* of injury or disease. Functional conditions, therefore, are only 'symptoms'—*i. e.*, casual coincidences, not exact signs of morbid conditions. This *negative* Principle illustrated by the Diagnosis of injuries of mechanism—*e. g.*, fractures, aneurisms, dislocations, respectively; also by diseases of nutrition—*e. g.*, inflammation of internal parts,—strangulated herniæ and ileus; tumours of various kinds; and degenerations of textural structure.

"How idle and insignificant do we find the opinions of physicians, and many, too, of high endowments and great repute, when we examine them, not in their books, but in the dead body. The practice of Physic has been long excluded from the circle of the exact sciences; but if the study of the dead body and its morbid appearances be combined with a constant, cautious, and rigorous observation of the living body in a state of disease, it will have a clear and undisputed title to be admitted among them, and especially in virtue of its Diagnosis.

"This is the direct and undeviating line that reasonable minds now incline to pursue, and it will, I doubt not, be generally pursued. What may observation avail us, if we know not the *seat* of the evil? We may make clinical notes from sunrise to sunset for twenty years, on the diseases of the heart, lungs, and abdominal viscera, at the bedside of our patients, and what shall we find in them? What but a detailed and circumstantial account of *symp-*

toms, which cannot be referred to any legitimate source, and therefore exhibit only a series of unconnected phenomena? Let us study the *dead* body, and the obscurity which observation alone cannot dispel will quickly disappear in the evidence it affords."

Thus wrote Xavier Bichat.* Since then, the study of Pathological Anatomy *has* been combined with Clinical observation—constant, cautious and rigorous. With what result? We still continue to read detailed and circumstantial accounts of symptoms which cannot be referred to any legitimate source, and therefore exhibit only a series of unconnected phenomena. Whence this continued failure of effort, pursued by so many investigators? Because Clinical observation has itself been, *in part*, misdirected. The "unconnected phenomena" are functional disturbances, and they are inconstant coincidences—*i. e.*, *symptoms* only. Thus it is that Physic *still* remains excluded from the circle of the exact sciences, and, so far, especially in respect of its diagnosis; for the *Principles* of diagnosis cannot be established on such insecure data.

The *inconstancy*, and therefore diagnostic insufficiency, of Functional disturbances, is a *negative* Principle, which admits of very general illustration.

Morbid conditions of function are significant only as being manifestations of alterations of structure (including those of physical properties and chemical composition). These are the *essential* conditions of disease. It may be that structural alterations are minute and likely to escape detection; it may be that they relate only to the quantity of blood in the tissue or organ, as compared with its solid matter, and that this proportionate vascular condition, being mutable, is almost or altogether effaced before a *post-mortem* examination (in the event of a fatal issue), and thus certain nervous diseases may appear to be purely functional; but instances of functional disturbance *alone* are very doubtful, and are, one by one, being referred to altered structure. Amaurosis, for example, in

---

* General Anatomy.

some cases, is now seen by the ophthalmoscope to be connected with certain structural alterations of the retina or optic nerve.*

The presence of altered structure is therefore more essential than functional derangement, which derives its diagnostic significance from Pathological Anatomy.

How far, then, are Functional manifestations faithful guides (during life) to disorganizations of Structure? In reply, I might point to common experience as reminding us of many instances of sudden death, after which chronic organic lesions have been discovered, the existence of which had never been even suspected, by functional disturbances, during life. Thus, the heart degenerated into fat may yield no warning palpitation, pain, or feeble first sound —may throw out no functional symptom—previous to its final stroke in sudden and unexpected death.†

Cases such as these, showing the *absence* of all functional disturbance, clearly denote the diagnostic powerlessness of functional conditions, and their incompetency, therefore, to fulfil even an early and exact diagnosis; but short of this total inability, the *insufficiency* of functional conditions is shown by their general *inconstancy*.

That functional disturbances do not invariably accompany a local disease or injury; and, moreover, that the same disturbance of function may accompany very different local diseases and injuries. Functional conditions are, therefore, to be regarded as only accidental coincidences or symptoms (Συμπτωμα, from συν, *with*, and πτοω, *to fall*). The truth of this proposition is well illustrated by the functional disturbances which casually accompany, but do not indicate, various injuries of the natural mechanism of the body, and various diseases of nutrition. Such are, respectively, Fractures, Aneurisms, and Dislocations; Tumours and Degenerations.

The functional concomitants of Fracture are pain and inability to

---

* Ophthalmoscopic Surgery. J. Hogg, Ed. 3, 1863, p. 155 *et seq.*

† See an elaborate monograph On Fatty Diseases of the Heart, by R. Quain, M.D., Med. Chir. Trans., vol. xxxiii.

use the part which has been injured. But these conditions are very equivocal. The pain may be insignificant at first, and the power of motion may be retained in the case of an impacted fracture—say, of the neck of the femur, of which several remarkable instances are on record. In the fourth volume of the "Mém. de l'Acad. de Chirurgie" a case is related, in which the patient walked home after the accident, and even got up the next day. Desault mentions a similar example, and others were seen by Dupuytren.* I have seen two instances of impacted fracture of the cervix femoris with the power of progression retained. Again, when at the seat of fracture there are two companion bones, as in the forearm and leg, and only one of these bones is broken, the other acts as a splint, and, by supporting the fractured portions pretty well in the natural position, preserves also the natural power of motion.

When pain and loss of the power of motion *are* experienced, these functional conditions may arise from other causes besides fracture; and, so far, a dislocation, a bruise, or an attack of rheumatism, may feign fracture.

The functional symptoms of an Aneurism are concomitant with its enlargement. The tumour now slowly, yet constantly and progressively, presses on surrounding parts, inducing various functional disturbances. The artery below the aneurism is, in course of time, gradually obliterated, and for a short distance becomes a fibrous cord. The flow of blood is thereby more obstructed, the veins become turgid, and swelling supervenes. The nerves are flattened into ribands, thus occasioning severe pain, and eventually partial paralysis. The muscles waste and lose their power of motion, while by their displacement, also, their lines of action are disadvantageously changed. In due time the tumour bursts, and with the occurrence of more or less hemorrhage, all these functional disturbances are temporarily relieved.

But observe, these symptoms (pain and loss of muscular power)

---

* Clin. Chir., t. ii., p. 96.

do not occur until a somewhat late period of the aneurism; and, moreover, do not accompany it throughout its career. They therefore are neither constant nor primary conditions, and cannot be used in aid of an early and exact diagnosis.

Passing on to Dislocations, we have to notice the pain and powerlessness of the limb which an individual experiences who is the subject of this kind of injury. But these, again, are mere casual symptoms, and very inconclusive evidence of a dislocation having occurred.

Pain in such a case arises from the pressure exercised by the displaced bone on the nerves in its vicinity, and is felt therefore in proportion to the number and size of the nerves which are thus affected, and in proportion also to the tension of the parts. An oft-recurring dislocation of the shoulder-joint begets such laxity of the articulation, especially in an old feeble subject, as to admit of the displacement occurring with comparatively little pain, while the head of the humerus being dislocated for the first time into the axilla of a young muscular man, and pressing with unyielding resistance on the leash of nerves which form the brachial plexus, occasions the most severe pain. Such pressure, indeed, is sometimes sufficient to obliterate pain, and induce temporary paralysis of the limb. Other varieties of functional disturbance may accompany special dislocations. For example, the sternal end of the clavicle being dislocated backwards has been seen so to compress the œsophagus as almost to prevent deglutition. But the insufficiency of *all* derangements of function in relation to diagnosis here holds good. The pain or other inconvenience may be absent; on the other hand, if present, such symptoms may attend other kinds of lesions and diseased conditions. The same restrictions apply to mere powerlessness of the limb — a symptom which supervenes slowly after the accident, and may not therefore be well marked. Every practical surgeon must concur in the experience of Sir A. Cooper, who examined a dislocation of the thigh-bone into the foramen ovale, a few minutes after the accident had happened. The dislocated part was still very moveable,

and continued so for the space of nearly three hours, when it became firmly fixed in its new situation by the tonic and permanent contraction of the muscles. But if the symptom in question be present, it may result from other causes than dislocation. Thus, loss of power to move a joint may indicate chronic rheumatism.

If, then, the functional disturbances to which I have alluded are present, and are regarded as sufficiently early symptoms, they are not sufficiently constant evidence to aid, much less to determine, an exact diagnosis.

The same inconclusiveness belongs to whatever disturbances of function may accompany the production and progress of any species of Morbid Growth.

The mechanical results of pressure on surrounding parts cannot become obvious until the tumour has attained some size. The obstructed circulation, or the embarrassed respiration, or the constipation from pressure of a tumour on the intestinal canal,—these and similar disturbances of function may accompany the growth of a tumour so placed as to impede the free working of some portion of the vital machinery; but such symptoms cannot arise from a small tumour which admits of easy accommodation to the part in which it is placed. The symptoms alluded to are therefore not *early* symptoms; nor are they sufficiently *exact*, because not sufficiently constant. A slow-growing tumour, such as are many instances of the fatty, cystic, fibrous, and cartilaginous tumours, may trespass so stealthily on the mechanism of surrounding parts as to allow of their functions becoming adapted, as it were, and reconciled to the inconvenience to which they are submitted. The experience of most surgeons will recall some such instances of tolerance; and this tolerance of all slow-growing tumours—a numerous class of growths—suggests the general insufficiency, because the general inconstancy, of functional disturbances, for exactly determining the presence of a tumour.

Again, the usual situation of many tumours is such as to preclude the possibility of their occasioning much functional disturbance. Thus, a fatty tumour is usually found under the skin, and slip-

ping about, probably on the back, so as to occasion no other inconvenience than the weight of a burthen of its own size and substance. Cartilaginous tumours very frequently select the bones of the fingers and toes, or the extremities of larger bones; and tumours so placed only interfere with the free motion of these parts. Their influence is purely local. Again, the chosen *habitat* of the fibrous tumour is the uterus—a passive organ, whose use is occasional, and not required day by day to maintain the organic life of the individual. A fibrous tumour may lodge quietly in the uterus for months and years without inconvenience. Lastly, if functional disturbances should perchance indicate, and at an early period announce, the presence of *a* tumour, it does not, and cannot, indicate the *kind* of tumour. Its nature may be benign and sociable with neighbouring parts, or malignant and aggressive.

To more particularly illustrate the insufficiency of functional disturbance in relation to the earliest and most exact diagnosis, let us glance at the clinical history of one functional condition—*pain*, considered as an accompaniment of various species of morbid growths.

Pain is a subjective symptom, and very capricious. It is neither an early nor a constant symptom of any morbid growth. Thus, very rarely indeed does a fatty tumour, advanced in life, suppurate in its centre, and become painful. An ordinary cyst or collection of cysts is painless, and a proliferous cyst in an early stage, and so long as it continues unbroken, may remain painless for years. The cyst I have already mentioned as having been removed by Mr. Lawrence from the thigh of a woman is an example. This tumour had been growing slowly and without pain for nine or ten years. Yet the fungous protrusion which eventually springs from a proliferous cyst is painful. Such was the condition of a very large protruding tumour of the breast, removed by Mr. Lawrence from a lady fifty-five years old. But these protrusions are late as well as inconstant productions.

A fibro-cellular tumour and an ordinary fibrous tumour are both painless; so also is a cartilaginous tumour: and when any

variety of either of these tumours is painful, it has become so after the lapse of time. Pain, therefore, is a late symptom in the varieties, and absent in the types, of these growths.

Lastly, if pain *does* accompany the growth of a non-malignant tumour, the presence but not the origin of which is so far indicated by this symptom, yet it is impossible, by any *character* of the pain, to recognise the kind of tumour. *A* tumour, not the tumour, is made known.

A malignant tumour is usually no exception to these general restrictions, respecting the diagnostic value of pain.

The word cancer is to many persons suggestive of perhaps the most exquisite torture. "Molten lead," a "hot dart" passing through the breast,—these and similar expressions are used by cancer patients; and, as Mr. Paget truly remarks, the pain is generally described by reference to some imaginary sensation, rather than by anything the individual ever felt before, as if actual experience failed to supply an adequate illustration of the kind of torture.

But cancer is not *always* painful. "Among the many inconstancies," observes Mr. Paget, "in the life of cancers, none I think is more striking than that which relates to the attendant pain. One sees cases, sometimes, that run through their whole career without any pain. In a case of deeply ulcerated cancer of the breast, the patient, who had also a cluster of cancerous axillary glands, begged that the disease might be removed, but only because it was 'such a terrible sight.' It had never once given her the least pain. In another case, a patient with a cancer involving the whole mammary gland, was quite unaware of any pain or other affection in her breast, until, a few weeks before its excision, some friends called her attention to its diminished size." The largest hard cancer of the breast which Mr. Paget had ever removed was equally painless; and another patient who died from rapidly progressive and ulcerated cancer, never experienced a pain in its two years' duration.*

* Op. cit., vol. ii., 1853, p. 339.

Pain then is not a constant, and therefore not an exact symptom of cancer. Neither is it an early symptom. Mr. Paget goes on to state that, in the early part of its course, for instance in ordinary cases for the first year or year and a half, the hard cancer of the breast is either not painful at all, or gives only slight and occasional pain, or again, only becomes painful by handling. Nor, if present, is the kind of pain uniformly the same. 'Darting' and 'lancinating' are terms in common use, and as if always descriptive of the kind of pain. But, during the first year or year and a half, the pain of *scirrhus* cancer of the breast has usually no peculiar character; is not generally lancinating, but usually, and especially after manipulation, dull and heavy: after this time, the cancer becomes more and more painful, and the pain acquires more of the darting and lancinating character; this pain is generally increased when the cancer grows quickly, and especially when it is inflamed or ulcerating, or about to slough; lastly, the pain becomes yet more intense while the cancer is progressively ulcerating, and now adds to its lancinating character, or substitutes for it the hot, burning, or scalding sensation.

No one kind of pain, then, is characteristic of Scirrhus cancer (of the breast) in all stages of its progress—neither dull, aching, lancinating, nor scalding pain.

In respect of *encephaloid* cancer, pain is by no means a constant nor an essential condition. Mr. Paget's experience is again explicit on these two points. "The history," says he, "of some of the medullary cancers which grow as distinct tumours, may teach us that the pain is not an affection of the cancer itself, but of the organ which it occupies. Such cancerous tumours in the subcutaneous cellular tissue are, I believe, rarely the source of pain; often they are completely insensible: yet the same kind of tumours seated among the deeper parts of limbs, or enclosed in the testicle or in bone, seem to be usually painful, and often severely so. This difference indicates that the varying pain is not of the cancer, but of the part it fills."*

* Op. cit., vol. ii., p. 405.

The clinical history of Inflammation exhibits the insufficiency of functional symptoms. The definition which has prevailed since the days of Celsus is an assemblage of four conditions—redness, heat, pain, and swelling. This definition is far from being unexceptionable. Redness, heat, and swelling are the physical characters of inflammation; pain alone, its local functional symptom. But the occurrence of pain, and its intensity, are regulated by many circumstances. When the capillary vessels of any part, about to be inflamed, become turgid with arterial blood, redness and heat are the first accompaniments of this hyperæmia. Whether pain shall succeed as an early symptom, and its degree of severity, are alike uncertain.

The painfulness of inflammation is chiefly regulated by the more or less unyielding character of the texture or part affected, whereby the interstitial effusion, of lymph, serum, and exudation-matter, is more or less readily accommodated. If in a freely distensible texture—as the spongy parenchyma of the lungs—little or *no* pain may be experienced. Incipient pneumonia is so far latent, because unfelt. Pain therefore may be absent altogether, or not an early symptom of inflammation. And when this symptom does arise, its degree will depend in some measure on the quantity of arterial blood flowing through the part—in fact, the degree of inflammatory hyperæmia—and partly on the nervous endowment of the part, and the sensitiveness of the individual. The occurrence of pain is therefore no measure—no exact symptom of the intensity of the inflammation. "And, of all symptoms," writes a watchful clinical observer,* "mere pain is the most inconstant and uncertain, whatever be the disease. It is so in pericarditis. It is present in one case and absent in another, strangely and unaccountably. I have known much pain where the disease has been of little severity, of short duration, and of easy cure; and I have known the severest pericarditis pass through all its stages without pain. All other symptoms have been present to mark its reality

* Latham's Lectures on Clin. Med., vol. i., p. 141.

and its progress: the murmur, and the præcordial dulness, and the fluttering heart and the respiratory anguish. And sometimes the patient has died, and sometimes he has escaped by a tardy and precarious convalescence. But from first to last there has absolutely been no pain.

"Do not be surprised at this. Pleurisy may exist without pain; even acute, rapid, pus-effusing pleurisy. Peritonitis may exist without pain; even acute, rapid, pus-effusing peritonitis. And so too if, in pericarditis, there is sometimes no pain, it fortunately happens that there are other signs by which we can fix our diagnosis of the disease equally well without it.

"See what a strange, unequal, and uncertain light pain throws upon diagnosis and treatment. We find it where we do not look for it, and look for it where we do not find it. Its presence is no sure proof, its absence is no sure negation, of disease."

If then the local symptom—pain—is not trustworthy, are the general symptoms, known as the inflammatory or symptomatic fever, more reliable? The chief elements of this fever are, general excitement of the arterial system, as denoted by a quick, forcible and hard pulse; and a general arrest of the secretions, as denoted by a dry, hot skin, scanty urine, and constipation. These functional disturbances arise most probably from an influence transmitted through the nerves from the inflamed part; hence, the commencement and subsequent severity of inflammatory fever is much regulated by the condition of the nerves at the seat of inflammation. In loose textures and parenchymatous organs, the nerves included are not submitted to pressure, certainly not at an early period of inflammation; consequently, inflammatory fever is not readily excited—is not an early symptom. Pneumonia and hepatitis severally proceed to some extent without inducing this constitutional disturbance. For the same reason inflammatory fever is not an exact measure of the severity and extent of the inflammation. The basis of both lungs, or a large organ like the liver, when inflamed, may *not* excite much fever, while the limited inflammation of tonsillitis arouses the most violent fever.

But if such constitutional disturbance should be well pronounced, it refers to inflammation in *any part* of the body, without guiding to the real source or seat of all the mischief. For example, the symptoms of strangulated hernia are those of ileus: if, therefore, an irreducible hernia be present, such symptoms might *possibly* mislead one to refer them to it, rather than to the ileus; or to infer that the case is one of strangulated hernia. The following illustrative case occurred in the practice of Percival Pott.*

An old gentleman, who had for many years had an irreturnable rupture of the mixed kind, was seized with symptoms of obstruction in the intestinal canal. He complained of great pain in his whole belly, but particularly about the navel; was hot and restless, and had a frequent inclination to vomit; his pulse was full, hard, and frequent; and, contrary to his usual custom, his bowels had not been relieved for three days. Mr. Pott examined this rupture very carefully; the process was large and full, as usual, but not at all tense or painful upon being handled; the belly was much swollen and hard, and the patient could scarcely bear the light pressure of a hand about his navel. Mr. Pott rightly inferred that the rupture had no share in the present sufferings of his patient; that the hernia was simply irreducible, and the operation was not performed. The patient died on the sixth day, and a post-mortem examination disclosed the real source of all the symptoms.

The hernial sac was thick and hard, and contained a large portion of omentum, a piece of the ileum, and a portion of the colon, all perfectly sound, free from inflammation or stricture, and irreturnable only from quantity. But the *jejunum* was greatly distended, highly inflamed, and in some parts sphacelated!

An analogous case was that of a man about thirty years of age, whose bowels had not been relieved for three days, although he had taken purges and used glysters; he vomited almost incessantly; his pulse was hard and frequent, but not full; and his countenance bespoke death.

There was a rupture on the right side; it was clearly intestinal,

* Chirurgical Works, 1790, vol. iii., p. 330.

soft, easy, occasioned no pain when handled, and seemed to be capable of reduction; but, after many trials, Mr. Pott failed to return it, although he used "his utmost endeavours," all which gave the man no uneasiness, and therefore satisfied Mr. Pott that the symptoms did not arise from the hernia, which was also the patient's own opinion. A counter opinion prevailed, and the operation was performed.

The hernial sac was formed by the tunica vaginalis; it contained a portion of the ileum, which, although slightly adherent to the testicle, was so perfectly free from stricture, that, when loosened from its connexion, it was returned into the abdomen without dividing the tendon. Death occurred the following day, and then a portion of the *colon* within the abdomen was discovered to have undergone mortification, and had become quite black!

In estimating the diagnostic value of functional conditions, as symptoms of Morbid Growths, it may be alleged that the proved faithlessness of such evidence refers to structures which are adventitious or superadded to the parts affected; but functional conditions are not more constant, in connexion with textural Degenerations of the various normal tissues of the body.

I cannot undertake thus to consider every kind of degenerative transformation, to which every organ or part is liable; but I shall select the most common—namely, fatty degeneration, and as it affects an organ of vital importance—the heart. Clinical observations respecting the diagnostic value of functional evidence of fatty degeneration of the heart are contradictory; but the anomalous conclusions of different observers are, in a measure, capable of explanation.

The literature of this transformation shows, beyond a doubt, that it was for a long time confounded with *interstitial* deposit of fat in the muscular substance of the heart, and around that organ. Therefore in considering the question of functional evidence, we may disregard certain clinical observations which referred indiscriminately, sometimes to true degeneration, sometimes to false. The results of more recent observations do not entirely concur, yet

they preponderate against the constancy of functional conditions as symptoms of true fatty degeneration of the heart.

Dr. Quain says:* "In general, *weakness, irregularity,* and *slowness of the pulse,* are the characters which we most frequently find. The irregularity may *not* be *constant;* I have seen it present during slight attacks; I have seen it disappear altogether while the patient was in tolerable health, to return as the effect of any depressing cause, the more marked because that cause may be far too insignificant to affect a sound heart. The *impulse* is *feeble,* and this symptom is proportioned to the extent and degree of the disease; a *feeble first sound,* scarcely audible in some cases (when the heart is enlarged there is extended dulness); and in two cases the second sound is said to have been feeble or imperfect." Dr. Quain dwells on the occurrence of *pain,* as a symptom; pain, either confined to the region of the heart, or radiating over the chest, and down the arm, as *angina pectoris.* The symptoms of an enfeebled heart and power of circulation are mentioned; such as *syncope, coma,* and *shortness of breathing.*

Concerning the diagnostic value of all these symptoms, Dr. Quain states,† that many cases are recorded in which patients have died and in whose hearts this condition has been *unexpectedly* found. Some of the individuals are said to have been previously in the enjoyment of perfect health; others had been ailing, but not in connexion with the heart; others ailing likewise, and the heart being suspected has been examined, and failed to give evidence of the existence of the disease, which post-mortem examination has subsequently shown to be present. Dr. Quain has not met with such cases. On the contrary, even in some instances in which the individual dying suddenly was said to be in good health, he found on inquiry, that symptoms, more or less marked, had existed, and might have been previously recognised, if attention had been called to them.

Now I pass over pain, and the remote symptoms of an en-

* On Fatty Diseases of the Heart, Med.-Chir. Trans., 1850, vol. xxxiii.

† Page 32, foot-note.

feebled heart and circulation; as syncope, coma, and breathlessness. Such symptoms are avowedly inconstant, or possibly due to other causes than fatty degeneration of the heart.

A feeble first sound, impulse, and pulse, which is also irregular and, generally speaking, slow, are symptoms immediately referrible to the heart's function; and they have been currently reported to be constant symptoms of true fatty degeneration, and also that they point to no other disease of the heart, unless, indeed, accompanied with some valvular murmur, extended dulness, or other indication of an organic lesion. But this general conclusion is disputed by other observers equally eminent, and whose evidence is more recent.

If, says Dr. Stokes,* it be inquired how far we have gone since the time of Laennec in establishing the diagnosis of this affection, it will appear that as yet but little has been done. Laennec declared that he knew of *no means* by which the diagnosis of fatty degeneration of the heart could be made; and Dr. Ormerod, writing in 1849, observes that the most extreme cases detailed show that the diagnosis on general or physical grounds is almost *impossible*. We cannot, he says in another place, predict with *certainty* in any case that we shall find this lesion after death; but it is difficult for any pathological observer not to be led to suspect the existence of a disease in the repetition of the same circumstances under which he has seen it occur previously. This etiological means of diagnosis has nothing to do with the estimate Dr. Ormerod had formed of functional conditions in relation thereto. Somewhat at variance with this authority, Dr. Stokes continues, the diagnosis of this condition is not only possible, but often free from difficulty, at least where the disease is *confirmed*. On the other hand, minor degrees of the affection are to be determined less by direct signs than by some general characters.

The functional conditions of the heart's impulse and sounds agree, according to Dr. Stokes, subject to unimportant variations,

---

* Diseases of the Heart and Aorta, 1854, p. 320.

with those of other observers already mentioned; so also do the characters of the pulse, excepting that it may be rapid in certain cases, as well as being permanently slow in others.

More unqualified is the incredulity of Dr. Walshe as to any constant functional symptoms denoting true fatty degeneration or "fatty atrophy" of the heart, as he calls it. "The clinical aspects of this disease vary greatly with the superficial extent and degree of the atrophous change. That fatty metamorphosis may be found, and to no insignificant amount, where neither subjective nor objective cardiac symptoms had awakened attention during life, is indisputable. And it is equally certain that hearts have been carefully examined during life, and pronounced free from disease, which almost immediately afterwards (death arising from other causes) have been found very sensibly fatty. The disease may then, to the observer of the present day, at least in its minor and moderate degrees, be *latent*."*

To this concurrence of statements founded on the experience of distinguished clinical observers, may be added the particulars of a case related by Dr. Semple, which I lately read in a paper by Mr. Canton, on the Arcus senilis.† A medical man died at the age of seventy-one, and the muscular fibres of his heart, which exhibited no morbid appearances to the naked eye, were examined by the microscope, separately, by Mr. Canton and by two practised microscopical observers. They all agreed in having discovered true fatty degeneration of the heart. Dr. Semple remarks, there can be no doubt this very serious disease of the heart had probably existed for a long period before death. "Yet it is a curious circumstance, that although fatty degeneration was naturally suspected, no symptom which could be referred to that lesion was ever detected during life."

The balance of evidence, therefore, goes to show that *all* functional disturbances referrible to the heart may be *absent* when that

---

* Diseases of the Lungs and Heart, 1854, p. 674.

† Lancet, September 12th, 1857.

organ has irreparably degenerated into fat, however strange this separation of functional and structural conditions appears as regards a disorganization so extreme, and of an organ so functionally essential to life. But if *present*, the functional disturbances may not be due to fatty degeneration, but to other organic disease of the heart; possibly interstitial deposition of fat, and its accumulation around the organ. The diagnosis by Dr. Hope* is pretty well that of the present day. The following symptoms concurring probably denote this sub-pericardiac accumulation of fat:—Diminution of the sounds, especially the first. Irregular pulse, without valvular disease. "Oppression," or even pain, in the præcordial region, with general signs of a retarded circulation, producing cerebral, hepatic, and other congestions. Three cases illustrative of this diagnosis are adduced by Dr. Hope; but the first two are presumptive only from the symptoms during life; the third alone is verified by dissection. More recently another case was similarly verified by Dr. Walshe.† "A sensation of oppression, or even pain, about the præcordial region; syncopal feelings on exertion; inability to walk quickly on level ground, and to get up hill, except with great and powerful effort; inclination to coldness in the extremities; feeble but (as far as I positively observed) regular pulse, of about medium frequency; sluggish action of the liver and bowels; occasional giddiness, and feeble cardiac impulse, with a too extensive dulness under percussion; the sounds, especially the first, being weak and toneless: these were the symptoms and signs in the only person, a male, aged sixty-four, I happen to have watched professionally during life and opened after death, whose heart was at once loaded with sub-pericardial fat, and positively free from serious softening or notable amount of intra-sarcolemnous oil."

The functional disturbances thus accredited by Dr. Hope and Dr. Walshe as symptoms of this *false* degeneration, agree, except-

---

* Diseases of the Heart and great Vessels, 1839, p. 347.

† Op. cit.

ing as regards the rhythm of the pulse; and the more explicit account given by the latter authority clearly shows how nearly these functional symptoms resemble those of *true* degeneration of the heart. Yet the prognosis of these two conditions is as different as they are themselves unlike: the one a substitution of fat for muscle—the muscle of the heart; the other, a mere super-addition of fat, which may come and go. The guidance of functional symptoms is, therefore, so far treacherous. But more than this, the same symptoms may be due to *softening*, without degeneration, of the heart. This disease,—softening of the heart's muscular texture from malnutrition, gives out a weak first sound, a feeble impulse, and pulse which is also faltering, and is accompanied with faintness and much anxiety. But these are also the symptoms of true degeneration as well as of softening alone; and by such, therefore, it is impossible to distinguish simple softening from that of fatty degeneration, and scarcely from dilatation without either.* Yet the prognosis and treatment should be different, perhaps very different, and are more hopeful in simple softening without the substitution of lifeless fat. Here, then, we reach the climax of functional misguidance. The diagnosis of fatty degeneration of the heart cannot be considered exact when this disease is thus liable to be confounded with other and dissimilar diseases—as false degeneration by the interstitial deposit of fat and simple softening; nor can the diagnosis be early when the absence of symptoms precludes suspicion.

Neither can it be said that the diagnostic obscurity of fatty degeneration, as it affects the heart, or indeed of this *kind* of degeneration, is exceptional. The same obscurity prevails in respect of other degenerations, and as occurring in other situations. Atheromatous patches are found after death within certain arteries,—say those of the brain; and calcareous transformation is then found to have made them brittle, and to have resulted in fatal apoplexy, without one premonitory symptom

---

* See op. cit. by Walshe, p. 667.

during life. I knew a lady, about thirty years of age, who, although pale and weakly, enjoyed even health; yet, without a warning symptom of any kind, she suddenly fell in her bedroom, and expired in two hours. The basilar artery and its branches were found so brittle as to crumble under the fingers; and blood had freely escaped into both lateral ventricles of the brain, breaking down the septum lucidum, and throwing these cavities into one. This fatal hemorrhage, without any previous notice, is in keeping with the general insufficiency of functional symptoms to fulfil an early and exact diagnosis.

To conclude:—besides being late or absent—if present, the same functional symptoms may be intense when the disease or injury is trivial, and insignificant when either is grave, perhaps irreparable; or again, they may be due to two or more different diseases or injuries, or to the same disease or injury in different parts of the body, but which is the part affected, they do not tell. These discrepancies seriously impair the diagnostic exactitude of functional symptoms. On the other hand, they may be absent, or not happen sufficiently early in the course of a disease or after an injury.

But if we cannot trust the evidence of Functional disturbance to guide our Diagnosis, such disturbance serves at least the humbler purpose of telling *the individual* that all is not well, of announcing that *something* is going wrong; and when, by that normal degeneration which accompanies old age, the natural energy of mind and body subsides gradually, and fails without struggle or shock, it may be that this mortal puts on immortality imperceptibly and not without resignation and even thankfulness. Such, indeed, would appear to have been the experience of one who, in apparent health, at seventy-three years of age, when writing to a near relative, within two hours of his sudden and unexpected death, thus expressed himself:—"The appetite for any of the enjoyments of youth declines day by day as age advances, and it is comforting that it is so, for we leave the world with little reluctance."

While, then, as by the law of nature all who live must die, so

death, approaching insensibly, without sign or symptom, by degenerative transformation of structure, seems to be the appointed mode of passing through Nature to Eternity.

POSITIVE PRINCIPLES OF DIAGNOSIS.

Chapter III.

Pathological Anatomy advanced as the guide, during life, to the *earliest* and *most* exact Diagnosis.—The diagnostic value of this fundamental Principle illustrated by the diagnosis of the several varieties of Fracture and Aneurism, respectively.

What has Pathological Anatomy done for Therapeutics? is a question often suggested, and easily answered, as to the relation involved. It is chiefly that Pathological Anatomy has done much, very much, for Diagnosis. The truth of this general proposition will have been already *inferred* from the conclusions established in the two preceding chapters; but the superior diagnostic value of Pathological Anatomy, and the comparatively inferior value of Functional symptoms (no less than of Anatomical conditions), in relation to the *earliest* and *most exact* method of diagnosis, will become more apparent by a critical examination of the general pathology of Fractures, considered in reference to their diagnosis.

Fracture has been defined to be "a solution of continuity of one or more bones, produced in general by external force, but occasionally by the powerful action of muscles, as is often exemplified in the broken patella." This definition, laid down by Sir A. Cooper, is not unexceptionable. It would include those injuries of bones which are made by sharp cutting instruments. A sword-cut of the shaft of the femur, for example, partakes rather of the nature of an incised wound than a fracture. Herein we recognise one essential element of a fracture—that, if regarded as a wound of a bone, it is always either a *contused* or *lacerated* wound. I shall presently show that this most important condition lies at

the very root of the pathology and diagnosis of Fractures; and, for this reason, I am inclined to adopt the old definition of Guido de Cauliaco:—"Continuitatis solutio in osse non a re qualibet, sed ab ea quæ contundit." Contusion and laceration are very similar lesions; and this definition, therefore, fairly includes those fractures which are produced by the accidental bending of a bone, and its breaking, either completely across, or incompletely through a portion only of its girth. The latter has been aptly denominated the greenstick fracture.

Fracture is either incomplete or, more usually, complete. If complete, then certain varieties have been distinguished apparently with much precision, and to these distinctions much importance has been attached. It is said that a complete fracture is, as regards its direction, either "transverse," "oblique," or "longitudinal." These terms convey their own meaning, and need no explanation. So long ago as the time of Galen, fractures were recognised by their figure:—First, "quæ fit per longitudinem;" secondly, per latitudinem; and thirdly, an intermediate direction—the oblique fracture. And these distinctions have ever since been acknowledged. How far they possess any practical importance, is a question I shall now endeavour to determine.

Keeping these distinctions prominently in view, the surgeon examining a fracture is guided by *anatomical* considerations to his diagnosis.

An ordinary complete fracture can be readily detected by certain infallible signs, which, to the educated eye, the hand, and even to the ear, are singularly convincing. These, the 'physical' signs of fracture, as they may be termed, are, either singly or collectively, conclusive evidence.

If the fracture be in one of the long bones of a limb, the natural length of the limb may be shortened by the involuntary contraction of the muscles acting on the lower fragment. For example, the humerus being broken betwixt its head and the insertion of the pectoralis major, that muscle chiefly, aided by the latissimus dorsi and teres major muscles, draws the lower portion

of bone inwards towards the thorax. Again, when both bones of the leg are broken, the lower portions of the tibia and fibula are drawn up behind the upper fragments, by the combined action of the gastrocnemius and soleus muscles. A similar shortening of the fore-arm is scarcely ever observed, or in a very trifling degree, in fractures of the radius and ulna; and if only one of either bones of the leg be broken, the other acts as a splint and prevents retraction. Moreover, shortening may possibly arise from dislocation; or the limb may be shorter than its fellow, owing to a previous fracture having been badly adjusted.

When, however, *shortening* does occur, what is its diagnostic value as a physical sign of the *kind* of fracture? Let the thigh-bone be broken in its middle third, the limb is generally shortened, and a certain fulness is both seen and felt in the upper part of the thigh, owing to the attachments of the adductor muscles being approximated. The lower portion has slid upwards and inwards behind the upper third of the femur, and the injury is most probably an oblique fracture of that bone. If there be not shortening and concomitant enlargement of the thigh, the broken portions of bone may project outwards in the situation of fracture. The angular deformity of the limb now seen is due to the action of those adductor muscles which, being attached to the lower portion of the femur, draw it inwards. The injury is said to be transverse fracture without displacement; and the alteration of *contour* which the limb now presents is another physical sign, supplying the place of shortening.

The surgeon having handled the seat of fracture, further assures himself that this injury has occurred. He both feels and hears *crepitation* of the broken portions of bone, when their ends are (gently) rubbed together; but he remembers that *a* (not similar) crepitation may attend the play of the tendons in their sheaths when thickened by old inflammation, and that crepitus is not felt when the ends of the broken bone are much displaced, or if soft tissues intervene, or if the fracture be examined some days after the accident. Whenever true crepitation can be felt, it is the surest

sign of fracture, and the *mobility* of the part gives further assurance that it is not a case of dislocation.

The most difficult case to determine is a suspected fracture of one of either companion bones in the leg or arm of a muscular subject, and examined some days after the injury, when considerable swelling has supervened. There is no shortening, and no angular deformity; no crepitation can be felt, and the mobility of the broken portions is obscured by the swollen mass of soft tissues in which they lie buried. Where is the surgeon, says Boyer, that has not sometimes hesitated to deliver an opinion in certain cases of this description; but I would add to this acknowledgment of occasional doubt and difficulty, that with the precautions of an educated tact, the diagnosis of fracture may be safely completed.

Not so the various *kinds* of fracture. They cannot be discriminated, nor their relative importance determined, by anatomical considerations of the length and outline of the limb, or by the bare fact of crepitation and mobility. These physical alterations are signs of a fracture having occurred, but not of its pathological course and tendency.

It was formerly believed that the anatomical conditions of fracture—as "transverse," "oblique," and "longitudinal," were most *essential* conditions of distinction; and accordingly great pains were taken in examining a fracture to most exactly ascertain these particulars, and as soon as possible, in order to fulfil the earliest and most exact diagnosis. Now, it is quite true that an oblique fracture, of the femur for example, is more liable to re-displacement after reduction than a transverse fracture. The anatomical condition in question favours displacement, but beyond suggesting the necessity of using appropriate mechanical contrivances to prevent this casualty after reduction, the fact of more or less mobility implies no essential difference in the injury. Simple fracture, whether transverse, oblique, or more longitudinal, alike tends to a speedy union. If therefore we make the mere anatomical direction of a fracture the ground of our diagnosis, we at once draw an

artificial distinction between lesions which, in respect of their pathological course and tendency, are naturally akin.

The false Pathology, if indeed it merited that name, which prevailed for so many centuries respecting the nature of the process by which broken bones unite, led to many frivolous distinctions of fractures, no less than to grave errors in their treatment.

Albucasis, and others of the Arabian School of Surgery, imagined that the ends of a broken bone exuded an inorganic juice which, if left undisturbed, set and hardened like concrete or cement, and eventually acquiring the consistence of bone, formed the *callus* whereby the broken portions were reunited. The literature of a later date bears ample testimony to the perpetuation of this erroneous doctrine. The great Paré affirmed, "Car ainsi que l'on joint les pièces de bois avec de la colle, ou les potiers d'estain leurs pots, ainsi nature cement les os rompus avec le callus;" in like manner our own countryman, Wiseman, advised the application of "such remedies as may dry the proper nourishment into callus.*

These false conceptions respecting the nature of callus gave an equally fictitious importance to the particular *direction* of a fracture. It being imagined that broken bones unite by the exudation of a juice which set and hardened like plaster of Paris, the joining of a fracture might be compared to the mending of broken china with cement, the soldering portions of leaden pipe, or the gluing together pieces of wood. This operation required for its successful completion absolute rest; and therefore that the mobility of an oblique fracture implied a far more serious injury than the steady position of a transverse fracture. But the progress of Pathology at length shed light on the true nature of callus. It was slowly discovered, and more slowly acknowledged, that callus is not a mere inorganic concretion; and of all those who contributed to reform this coarse mechanical pathology, no man deserves more honourable mention than John Bell. He prepared the way for other observers. He

---

* Principles of Surgery. John Bell, 1826, vol. ii., p. 13.

attacked the clumsy notion which had so long prevailed; he disproved the doctrine which would degrade the Surgeon to the level of a potter, a plumber, or a carpenter; and, by a few finishing touches of his vigorous pen, effaced for ever the absurd theory of an inorganic concreting callus.

He accomplished more than this. The writings of John Bell afford ample proof that he indeed perceived the blunder of the old Surgeons—that the callus they imagined would have been virtually a *foreign* body (although produced in the part), and that had it been the inorganic concrete they supposed, it could have held no connexion with the contiguous living bone; but Bell, moreover, clearly recognised the fundamental pathological truth, that callus is a true regeneration of bone, organized by the same action as that by which the original bone itself was formed.*

The formation of callus is analogous to the production of other false tissues; and the union of a simple fracture, whether transverse, oblique, or longitudinal, is analogous to the union by adhesion of an incised wound. This reuniting of living textures, unlike the joining by cement, is a plastic process of growing together. The details of this reparative process were in due time discovered, and will be hereafter fully described.† A layer of lymph is deposited between the fractured ends of bone; which becoming organized, re-establishes their continuity, and eventually forms an intermediately connecting portion of bone—an "intermediate callus." This also is the *only* callus where the fragments are not in even apposition, but overlap.

The observations of Duhamel, Dupuytren, and others, were made from experiments on animals; and the conclusions drawn from such observations are not applicable to the repair of fractures in the *human* species, excepting under special circumstances. Neither an external nor an internal callus is produced—as was formerly supposed—excepting when the fractured limb is subjected to unusual motion, during the process of repair, or when the original

---

* Op. cit., vol. ii., p. 15.

† See chapter 15.

bone is diseased. Then, callus may be thrown out round the seat of fracture—ensheathing the fragments as with a clasp; and deposited also within the medullary canal. But even in these cases, this external and this internal callus are temporary or provisional. They serve the purpose of two splints, to retain the fragments in apposition, while the permanent, intermediate callus is forming; and, when no longer required for such purpose, are gradually withdrawn—the bone being fashioned off by absorption, and eventually restored to nearly its original symmetry of outline.

Thus is a 'simple' fracture united within a limited period, without regard to its particular direction or figure. The mere anatomical conditions of direction—"transverse," "oblique," and "longitudinal"—are contingencies of minor consideration. The same process of union, similar to that whereby an incised wound is healed, reunites all varieties of simple fracture. Let a bone be broken in two, three, or more pieces, making a "comminuted" fracture; the same mode of union ensues, although perhaps more slowly, owing to the requisite supply of blood being cut off from these more isolated portions of bone. Even in this unfavourable condition, the resources of the reparative power of adhesion do not fail.

I have said that fracture is always a contused, or it may be a lacerated, wound of a bone. This *pathological* condition, affecting the formation of callus, lies at the very root of the most exact diagnosis of fractures. If fracture be the result of direct external violence, as a heavy blow or kick, the bone is smashed—comminuted fracture—or any prominent portion of bone is knocked off. The malar eminence may thus be crushed in a prize-fight, or the olecranon process struck off by a smart blow with a stick. Such fractures—resulting from direct external violence—are necessarily *contused* wounds of bone. But more generally, a bone is broken by indirect external violence—by force applied at a distance from the situation of fracture. A fall on the shoulder may snap the clavicle in either of its curves; or, in jumping from a height,

on coming to the ground the tibia or the femur may give way. The bone breaks in these cases with an irregular surface, unlike the clean division of a sword-cut. Such fractures are *lacerated* wounds of bone. Muscular action occasionally produces the same result; thus the patella has been broken transversely in the act of jumping, and the humerus snapped by a powerful blow which missed its aim.*

Certain circumstances predispose to the occurrence of fracture; and, moreover, subsequently *retard* the *union* of a broken bone.

Of the predisposing causes of fracture, I have already mentioned—the situation of the bone; and generally speaking, superficial bones—*e.g.*, the tibia—are more readily broken than those which are cushioned and protected by surrounding soft parts. The particular function or use of certain bones exposes them to fracture. For example, the radius is liable to be broken in the manifold use of the hand; so also is the clavicle—the keystone of the shoulder-joint, and on which turns all the motions of the upper limb. Age is a telling circumstance. In youth the bones are flexible, and bend or break easily; hence the incomplete (or 'greenstick') fracture is more likely to occur at this period of life; but fracture in childhood as readily unites and forms a permanent callus. In adult age and advanced life, the animal matter of bone is withdrawn, and the earthy portion then prevailing, the bones are brittle, liable therefore to break, and do not unite. Certain blood-diseases also predispose to fracture, and retard or arrest the formation of callus. Rickets, syphilis, cancer, scrofula, and scurvy, have severally this twofold effect; while febrile disturbance, erysipelas, starvation, and perhaps pregnancy, severally tend to prevent union.

Beyond these constitutional conditions, if either be present, but little else retards, and certainly does not arrest, the formation of callus and the union of *simple* fracture. Spasmodic action of the muscles tends to shift the fragments; and so far, therefore, the direction of fracture—an anatomical condition—suggests watchfulness during the process of repair; but in simple fracture—whether

---

* Dict. of Prac. Surg., 1838, S. Cooper, p. 535.

transverse, oblique, more longitudinal, or comminuted—the broken portions of bone still adhere to the vascular periosteum, and the whole lies buried in a soft, succulent mass of living, growing tissues, which speedily regain connexion with and reacknowledge, so to speak, the broken bone. All simple fractures, however *affected* by general or local circumstances of distinction, alike *tend* by this process of adhesion to a speedy union and the formation of a permanent callus. This pathological law overrules all other circumstances of distinction, excepting those of contused and lacerated fractures, considered in relation to age and certain blood-diseases, to determine the varieties of simple fracture. I would, therefore, speak of a *contused*, or of an ordinarily *lacerated*, 'simple' fracture, as the case may be, associated with a certain age and state of the general health. Now, these are *Pathological*, not Anatomical, grounds of distinction and diagnosis; and they refer to corresponding differences respecting the callus-forming tendency. Other fundamental distinctions of fracture emanate from this source.

The crushing force which occasions a simple contused fracture may bruise and tear the soft tissues down to the bone; or again, the broken ends of bone may lacerate the soft parts and protrude through the skin. In either case a wound is made, leading from the surface down to the seat of fracture, which is then said to be 'compound.' This lacerated wound is in effect an extension of the fracture to a similar lesion of the neighbouring soft parts; it scarcely ever heals by adhesion, but is followed by suppuration, often profuse, sloughing, hectic emaciation, and the slow formation of callus. Here again, therefore, pathology is our guide to diagnosis, and supplies another important variety of fracture—the compound—easily recognised by the wound through the soft parts.

Lastly, fracture may be 'complicated' by some further injury which, besides its own importance, impedes or impairs the formation of callus. The fracture of a bone in either of the extremities may be complicated by the rupture of a large artery or nerve, by

laceration of the muscles, or it may extend into a joint, or be accompanied by dislocation. Fracture of the pelvis may implicate the bladder, broken ribs wound the lung or pericardium, and a depressed fracture of the skull injure the brain. It would be idle to insist on the significance of these *pathological* circumstances of distinction. Only one conclusion can be drawn from the analysis we have made of these lesions.

The simple, the compound, and the complicated fracture represent certain conditions which promote or retard the formation of a permanent callus. The 'simple' fracture implies no important rupture of the surrounding soft tissues, and therefore suggests speedy union; the 'compound' fracture signifies their laceration, and therefore tardy or imperfect union; and the 'complicated' denotes important injury of contiguous parts, often essential to life, and, moreover, proportionate evil influence on the formation of callus. These are pathological distinctions, and open to detection during life; they are also the most exact and earliest reliable grounds of distinction. The diagnostic value of Pathological Anatomy is therefore fully verified.

The guidance of Pathological Anatomy to the earliest and most exact Diagnosis is illustrated by the diagnosis of Aneurisms.

The diagnostic value of Pathological Anatomy can scarcely be *established* more clearly than as illustrated by an analysis of the ground of diagnosis, in respect of fractures; but the truth of this principle will be more fully exemplified by tracing the pathology of Aneurisms in relation to their diagnosis.

Aneurism is essentially a collection of blood within a dilated portion of an artery, which thus becomes a sac containing arterial blood, and communicating with the artery from which it springs. Such is "true" aneurism, so named to distinguish it from a spurious kind of this lesion, occasioned either by the rupture of a true aneurism or by the wound of an artery; and in either case, an effusion of blood within the adjacent cellular tissue. This tissue may be loose and permeable, so that being infiltrated with blood it presents a diffused swelling, or becoming condensed

the blood is thereby enclosed in a kind of circumscribed sac. "False" aneurism, therefore, presents either a *diffused* or a *circumscribed* swelling, occasioned by the escape of arterial blood, and its accumulation within the cellular tissue external to a ruptured artery; while true aneurism, not being formed by the actual extravasation of blood, signifies its progressive accumulation within a dilated portion of an artery.

But whether true or false, diffused or circumscribed, aneurism is a collection of arterial blood in direct connexion with the portion of artery from which it has emanated; and as this unexceptional condition gives rise to all the symptoms which are peculiar to, and therefore characteristic of, this lesion, I am inclined to adopt the general definition of Lisfranc, and speak of aneurism as "a tumour formed by arterial blood, and communicating with an artery."

In the early days of Surgery, authors took a one-sided view of this subject. The literature collated by Mr. Erichsen* under the auspices of the Sydenham Society, shows, that while the old surgeons, with one or two exceptions, believed only in aneurism by *rupture*, their descriptions actually referred to aneurism by *dilatation*. The observations of Galen,† Gorræus,‡ Paré,§ Guillemeau,‖ and those of Wiseman at a still later period, are, I think, sufficient evidence of this inconsistency. The latter writer states, that "not having been able by my practice to discover one aneurisma made by dilatation or relaxation of the outward coat of an artery, I am apt to believe that there is no such thing, but that it takes its rise from the blood bursting quite through the artery into the interstices of the muscles, where it raiseth a

---

* Observations on Aneurism. 1844.

† Claudii Galeni, Opera Omnia, tom. viii.: fol., Venet. ap. Juntas, 1609, vol. v., p. 84. De Tumoribus præter Naturam, cap. xi. De Aneurysmate et Sydratione.

‡ Joanni Gorræi, Opera Medica: Paris, 1622, fol. Defin. Medic. p. 56.

§ Les Œuvres d'Ambroise Paré: fol., Lyon, 1641. Liv. vii., chap. xxxiv., p. 184.

‖ Les Œuvres de Chirurgie de Jacques Guillemeau: Paris, 1612, fol., p. 698.

tumour suitable to the cavity it findeth, growing bigger or less, of this or that shape, as the muscles give way. But (adds Wiseman) this tumour consists of blood extravasated, the artery lying undilated the while." Yet the *description* given by this author of such a tumour coincides with that of aneurism by dilatation. "It is," says he, "a tumour, soft, white, and yielding to the touch, but riseth again upon the removal of your finger, and is for the most part accompanied with pulsation of the artery."*

On the other hand, certain of the earlier writers took an entirely opposite, and equally exclusive, view of this question. Fernelius speaks of aneurism as being the dilatation of an artery, and it is scarcely credible, says he, that some imagine that in these affections the vein or artery is ruptured or opened; for if the blood had escaped from the vein or artery it would soon putrefy, and give rise to a tumour of a different kind.† In like manner Sennertus appears to have confined his attention to this description of aneurism, and to have been biassed and restricted accordingly in his views. He imagined that if the tumour were formed by an effusion of arterial blood collected under the skin, the blood ought certainly to be diffused far and wide all round, and discolour the skin, as we see happen in contusions; and that the effused blood, being in an unusual situation, would soon putrefy, as also occurs in ecchymosis.‡ Sennertus then proceeds to urge that aneurism is invariably produced by an opening in the internal tunic and dilatation of the external coat of an artery, of which two coats he conceived the vessel to be formed. Fabricius Hildanus followed with similar opinions;§ while Lancisi, in 1728, just a century after the observations of Sennertus, distinctly recognised

---

* Chirurgical Treatises, ed. 6, 1734, p. 116.

† Joan. Fernelii, De Morbis universalibus et particularibus. 8vo. Lugduni, Batavorum, 1645.

‡ Danielis Sennerti, Opera Omnia: Lugduni, 1650; fol., lib. v., part i., cap. xlii., p. 306.

§ Gulielmi Fabricii Hildani, Opera: Francofurti, 1646; fol., cent. 3, obs. 44.

the rupture of an artery in certain cases, and its dilatation in others, as lesions alike productive of aneurism.*

The distinctions of true and false aneurism, as now understood and accepted, were therefore clearly anticipated in the writings of Lancisi; but on turning to those records, it will be observed that he used the terms true and false, with reference to aneurism, in a different sense to that which is now attached to them. Petit† first asserted these distinctions in their *present* acceptation; they were subsequently adopted by William Hunter,‡ and have ever since prevailed. The latter authority also notices a "mixed" kind of aneurism, the result partly of dilatation, and partly of rupture—a condition of an artery to which I shall presently more particularly allude.

I have made these references to the bibliographical history of aneurism with the view of introducing this proposition—that while neither of the one-sided opinions formerly held respecting the nature of aneurism are now tenable, the *present* distinctions, 'true' and 'false,' do not denote any important pathological differences, and cannot always be clearly recognised in practice.

The demonstration of this proposition will indirectly bring out the value of Pathological Anatomy, in relation to the most exact diagnosis; from a due consideration of which I shall proceed to offer certain distinctive denominations of aneurism, founded on a critical examination and discrimination of the various pathological conditions of this lesion. To this end, I shall first observe the characters of aneurisms (as I have described other kinds of lesion) at the bedside; and then, taking their morbid anatomy in connexion with their signs and symptoms, endeavour to determine

---

* Joh. Mar. Lancisi, De Aneurysmatibus Opus Posthumum: Romæ, 1728. Prop. v.

† Observations Anatomiques et Pathologiques au sujet de la Tumeur qu'on nomme Aneurysme. Par M. Petit, de l'Acad. Roy. des Sciences. Paris, 1736. p. 244.

‡ The History of an Aneurism of the Aorta, with some Remarks on Aneurisms in General. By W. Hunter, M.D. Med. Obs. and Inquiries. London, 1757. Vol. i.

to what kind of diagnosis we are thereby fairly and honestly conducted.

The signs of "true aneurism" are modified somewhat by its duration; but, as I have said, all of them are referrible to the direct communication of the sac with the current of arterial blood. True aneurism, say of the popliteal artery, at first presents a small, soft, yet circumscribed swelling, pulsating in unison with each beat of the heart; but this pulsation ceases, and the tumour itself subsides, if the flow of blood through the artery be arrested by compressing the femoral in the thigh. Moreover, at this early period, the aneurismal sac can be emptied by pressure, and the blood returned into the artery. In time, as the tumour enlarges by the pulse-wave current through the artery, the accumulation within the sac is withdrawn more and more from the influence of the current, and is therefore more and more at rest. This comparatively undisturbed state of the blood within the aneurismal sac favours its coagulation, which gradually supervenes. A clot is slowly formed by the successive deposition of concentric layers of fibrin, proceeding from the interior of the sac towards its opening into the artery. The innermost portion of this clot is loose and semi-fluid, and has the appearance of currant jelly; the adjacent layers acquire the consistence and appearance somewhat of damson cheese; while the outermost portion eventually becomes friable, and resembles boiled beef in consistence and colour.

The aneurismal sac, enlarging and borrowing more and more fibrin from the passing stream of blood, is now partially consolidated. The tumour, as felt externally, is therefore no longer soft, but still circumscribed; its pulsations are fainter and less expansive, and the swelling cannot be reduced by either of the manipulations I have mentioned. This semi-solid swelling may remain tolerably quiescent—illustrating the natural cure of aneurism; but usually, the tumour continues to enlarge, and by its constant and increasing pressure produces various functional disturbances and alterations in surrounding structures. The artery below the aneurism becomes partially obliterated, or quite impervious for a

short distance, as a fibrous cord. The current of blood, therefore, is obstructed or altogether intercepted, the veins are turgid, and œdema supervenes. The nerves are, by pressure, gradually flattened into ribands, occasioning intense pain, or eventually partial paralysis; the muscles waste, and the bones erode. This disintegration of the osseous structure resembles caries, but is unaccompanied by the formation of pus.

Such are the general consequences of aneurism. It is unnecessary for my purpose that I should notice the *special* consequences of this lesion when it occurs within either of the great cavities of the body.

In the course of time, aneurism bursts and discharges itself, probably into the cellular tissue, constituting a "*diffused* false aneurism." The tumour now resembles a large 'ecchymosis;' its superimposed integuments are discoloured like a bruise; its size is much greater than that of the original aneurism, which appears to have suddenly become much enlarged; its outline is no longer circumscribed; the pulsations are yet fainter and more indistinct, or quite imperceptible; and the blood, being effused, cannot be returned into the aneurismal artery. The functional disturbances consequent on true aneurism may persist or be temporarily relieved. Such is the ordinary history of true aneurism. It is unnecessary for me here to describe its ordinary termination, further than to mention that, by sloughing and rupture of the skin, or by a similar destruction of the mucous membrane,—in the issue of certain internal aneurisms, or by the fissure of a serous membrane—should the tumour point towards the peritoneum, the pleura, or pericardium, hemorrhage occurs, either sudden and fatal, or recurring again and again, at length closes the unhappy patient's sufferings.

Aneurism which has burst into the cellular tissue may, for a time, stop short of fatal hemorrhage, and form a circumscribed and smaller tumour, which pulsates, and subsides under pressure.

The production of this "*circumscribed* false aneurism" depends on the size of the artery and the kind of opening in it, and, moreover, is regulated by the force and rapidity of the circulation.

When the arterial system is bloated, and the flow of blood impetuous,—as in sthenic plethora, when the artery is a main one, and its rupture sudden and considerable,—then the hemorrhage, not being restrained, soon infiltrates the cellular tissue to a considerable extent, and forms a large *diffused* aneurism; but when the circulation is feeble, the artery smaller, and it has yielded gradually with a small lacerated opening, then the hemorrhage is less extensive, and being also delayed, the blood tends to coagulate, the interstices of the cellular tissue are soldered up with half-coagulated fibrin, and a *circumscribed sac* is thus constructed, which slowly enlarges, like a true aneurism, as the pulse-wave continues to beat upon its interior, in this case gaining access *through* the aperture in the artery.

The dilatation of *all* aneurisms is, generally speaking, slow. John Bell* has established this fact by a series of cases collated with his usual judgment. He particularly refers to one of Guattani's patients, where a "true" aneurism of the femoral artery underwent dilatation for a year ere it burst into the cellular texture of the thigh; another instance from "Warner's Cases in Surgery," where three months elapsed before a popliteal aneurism burst; and an aneurism of the aorta, which, although arising in this case from the largest artery in the body, and through which the flow of blood is most impetuous, yet slowly dilated apparently for several years!

During this slow dilatation of aneurism, all the coats of the artery may yield together and burst simultaneously, or yield and give way one after the other; and this latter transitional condition, consisting, probably, of the rupture of the internal and middle coats of the vessel, and the dilatation of its external cellular investment, represents the "mixed aneurism," mentioned by William Hunter and described by Scarpa. This intermediate form of aneurism, neither true nor false, did not escape the notice of an earlier observer, Dr. Donald Monro, who, in 1760, with the anatomical acumen for which his family were distinguished, made

---

* Principles of Surgery, ed. by C. Bell, 1826, vol. iv., p. 375.

the dissection of one John Parker. There were at least six aneurisms; three of the right femoral artery, one of the left femoral, and one of either popliteal artery. The smaller aneurismal sacs of the femoral arteries appear to have been perfect, each having the circular fibres of the muscular coat continued entirely around its circumference; but in the left ham this investment was wanting at least in a portion of the sac, which appears therefore to have been a mixed aneurism; while the largest sac, that in the right ham, and which had burst with fatal hemorrhage, presented no traces of the muscular coat. The anatomical condition in this series of aneurismal sacs shows that while "mixed aneurism" was formed by the successive rupture of the coats of the artery undergoing dilatation, their laceration proceeded from within outwards. And such is the way in which this variety of aneurism is generally produced. On the other hand, there are rare instances of aortic aneurism, recorded by Dubois and Dupuytren, where the external cellular coat of the artery having been first removed by absorption, the internal and middle coats yielded slowly, and were at length dilated in the form of a pouch.

Considering the recognised place accorded to mixed aneurism in books on Practical Surgery, we should presume that it was a pathological condition *per se* of special signification, and capable of being detected during life. Yet what are the *distinctive* signs of mixed aneurism? None whatever. The tumour presents the same circumscribed outline as true aneurism, and, like it in an early stage, pulsates, but subsides under pressure. No surgeon, however gifted by nature or guided by the *tactus eruditus* of experience, can discriminate during the life of a patient the signs of true, from those of mixed, aneurism. These anatomical distinctions are utterly hopeless in practice; and the description of hernia, with its many successive layers or "coverings," is not more frivolous and delusive than are the pretensions of mixed aneurism to exact diagnosis. It is a trivial *post-mortem* distinction, without any symptomatic characters peculiar to itself.

Nor if we regard the tendency and termination of mixed aneurism, do we discover anything special or peculiar in its history.

The prognosis is that of true aneurism. Like it, a mixed aneurismal sac eventually bursts into the surrounding cellular tissue. If the laceration of the artery be inconsiderable, and the first hemorrhage be not sustained by the bounding pulses of an active circulation, the little eddy of blood burrows but slowly, and eventually forms circumscribed false aneurism.

The signs of *this* condition also are in no way peculiar. They are those of mixed, those of true, aneurism; equally with either presenting a circumscribed swelling, and, in its early stage, beating and thrilling under the finger, but reducible. At the bedside of a patient, we search in vain for any pathognomonic sign whereby to determine our diagnosis. Whether the aneurismal sac consist of the cellular tissue of the part, that of the artery, its external coat, or of all three coats of the artery, the aneurismal signs are practically the same. Moreover, the appearances of these aneurisms, as seen during surgical operations, are very similar. "That every surgeon is able in bloody operations in the ham or thigh, in aneurisms lying close under the heads of the gastrocnæmii muscles, to distinguish what is the sac of a true, and what of a false aneurism; what arises from dilatation of the artery, and what from the cellular substance, is not to be believed." (J. Bell.)

We may therefore conclude that, for all practical purposes, the true, and the circumscribed false, aneurisms are indistinguishable. Both are circumscribed, &c.* The only variety, therefore, of false aneurism worth considering in reference to diagnosis is the diffused; and as the terms 'circumscribed' and 'diffused' express appreciable distinctive characters, I propose thus to designate aneurisms, and to abolish the old terms "true" and "false" from Surgical nomenclature.

Hitherto I have spoken of the circumscribed and diffused aneurisms as having a *spontaneous* origin; but these conditions may also be the result of wounds, strains, fracture, and other injuries, implicating an artery of some size. John Bell narrates the case of a young man whose femoral artery was wounded with a

---

See p. 201.

penknife. The opening in the vessel was here only a small puncture, and a day elapsed before the nature of the injury declared itself. Then an aneurism began to form; but as yet the wounded artery was not far from the surface of the thigh; external hemorrhage therefore occurred freely, and in a few moments no less than three pounds of blood escaped; but, on the third day, the coagula were so firm, the wounded artery so deeply buried under the coagulating blood, and the external wound so steadily compressed, that it healed. The aneurism then assumed its proper form of a pulsating tumour, and at the end of three weeks from the time of the wound the operation was performed upon an aneurismal sac, so small and circumscribed that the limb in general was but little swelled, and this small circumscribed and beating aneurism was entirely limited to the upper and fore part of the thigh.

The history of this and similar cases leads to an important conclusion, and one which from the nature of the injury may be anticipated. It is—that the signs of a *traumatic* circumscribed aneurism are similar to those of the spontaneous circumscribed form of aneurism. We might presume that much will depend on the force of the blood's circulation and on the size of the artery that is wounded, but more, perhaps, is due to the size, shape, nature, and direction of the aperture in the vessel. If the opening be a small puncture, the blood probably flows but slowly into the cellular tissue, and therefore slowly works out a moderate-sized sac, which has a tolerably well-defined outline, when examined through the integuments, pulsates, and is reducible, like a spontaneous circumscribed aneurism. More significant is the direction of the opening in the artery. If the aperture be oblique, then the flow of blood is yet further delayed by the valvular form of such an opening, and the signs are even more nearly those of a circumscribed aneurism arising spontaneously. The stretching, straining, or laceration of an artery is attended with similar symptoms. M. Saviard relates the case of a man who was driving a cart laden with corn; it stuck deep in a rut; he hooked his right arm

under the wheel to lift it out, and strained with such impatience and violence that he hurt his arm, and suddenly exclaimed, "My arm is broken." An aneurismal tumour immediately began to appear in the bend of the elbow, and gradually increased till he was no longer able to bear it.

But most of all will the characters and signs of a traumatic aneurism be influenced by the looseness of the areolar tissue amid which the blood infiltrates, and by the pressure of surrounding parts. If the cellular tissue is abundant and loose—as in the axilla, thigh, and buttock—a large diffused aneurism with its peculiar characters will most probably ensue. Mark the following well-known case by John Bell. A poor man, by trade a leech-catcher, fell as he was stepping out of a boat, and the long and pointed scissors which are used in that business being in his pocket, pierced his hip exactly over the place of the sciatic notch, where the great iliac artery emerges from the pelvis. The artery was struck with the point of the scissors, it bled furiously, the patient fainted; and so narrow and deep a wound the surgeon, when he came, found little difficulty in closing, and less difficulty in making it heal. The outward wound being cured, a great tumour soon formed, and the man travelled up from the North country, where the accident had happened, and in six weeks after arrived at the hospital with a prodigious tumour in the hip, his thigh rigidly contracted, the ham bent, the whole leg shrunk, cold and useless, as if it had been an aneurism rather of the femoral artery. This tumour contained, as it turned out afterwards on dissection, not less than eight pounds of blood; and by the very circumstance of its being one of the largest aneurisms ever known, it had lost all the characteristics of aneurism. Pulsation had ceased; the tumour was irreducible by pressure; there was nothing peculiar but this—the great and sudden distension occasioned intense pain.

Since the period when this remarkable case occurred, many similar instances of diffused aneurisms from wounds and other injuries have been met with; and the general conclusion to which we are led by an impartial consideration of all aneurisms having a traumatic

origin is, that as the signs of 'circumscribed' aneurism are the same —whether the origin of the lesion be traumatic or spontaneous, so also are those of 'diffused' aneurism—whether that condition arise spontaneously or be the result of a wound or other injury.

But it is no less unquestionable and significant that the rational treatment of aneurism is determined, not by reference to its condition —circumscribed or diffused—but by its origin, *spontaneous* or *traumatic*. In the latter case it is only necessary to lay open the tumour by a free incision—to turn out the coagula—to find the opening in the artery from which the blood flows, and then to arrest the hemorrhage by casting a ligature around the vessel above, and another below the bleeding aperture. Very different indeed is the rational treatment of spontaneous aneurism, whether circumscribed or diffused. The youngest student in Surgery must have heard of the memorable operation by John Hunter, who in 1785 first tied the femoral artery for a popliteal aneurism. And what first suggested this then novel mode of treatment? Guided by that knowledge of Pathological Anatomy which is the only sure basis of Operative Surgery, Hunter had observed the *fragility* of an artery from which a spontaneous aneurism arose. So long since as the year 1760—a quarter of a century prior to the famous operation referred to—Hunter dissected an aneurism of the aorta, taken from the body of an officer who had been a patient under the care of Sir John Pringle. "The aorta," says Hunter, "was not dilated about the aperture (leading to the aneurism); but its coats at that place were harder than natural, as if tending to ossify; and having lost their natural elasticity and toughness, were easily pulled asunder."

This and similar observations at length, in 1785, suggested that in performing the operation for spontaneous aneurism, the artery should be taken up at some distance from the diseased part, so as to diminish the risk of hemorrhage, and admit of the vessel being more readily secured in the event of such contingency. The force of the circulation being thus removed from the aneurismal sac, the cause of the disease would, in Hunter's opinion, be removed; and he thought it highly

probable that if the parts were left to themselves, the sac, with the coagulated blood contained in it, might be absorbed, and the whole of the tumour absorbed by the actions of the animal economy, a provision which would consequently render the opening of the sac unnecessary. Such is Everard Home's account of the reflections which induced this grand operation—grand, because it implied a new and general principle applicable to all operations for the cure of spontaneous aneurisms. I need not here enter on the comparative advantages of the more conservative mode of treatment—by compression. I am considering aneurisms in relation to the most exact diagnosis, and it is sufficient for my present purpose to revert to the fact which an analysis of their history amply testifies; that the signs of circumscribed aneurism are the same, whether its origin be traumatic or spontaneous; and that those of diffused aneurism are also similar, whether it arise spontaneously, or be the result of a wound or other injury; but to this generalization we may now add the equally important facts—that the knowledge of the origin of aneurism concludes its diagnosis (in relation to the appropriate treatment), and this by virtue of the anatomically *diseased* condition, or otherwise, of the artery, which it suggests.

Pathological anatomy, therefore, supplies the most exact, and earliest reliable, diagnosis of aneurisms; so that as we recognise the perceptible distinctions—'circumscribed' and 'diffused,' we complete the diagnosis, by associating that condition of the artery which is suggested by the origin of the aneurism—*spontaneous* or *traumatic*. But, thankfully accepting the infallible guidance of Pathological Anatomy in diagnosis, we should ever reject those frivolous distinctions which the wholesale and indiscriminate application of this science to Surgery is sure to entail. Rather should we restrict our attention, as surgeons, to the study of those morbid conditions which can be recognised *during life*, by clinical observation. Regarded from this practical point of view, the primary distinctions of aneurisms, which have hitherto held an acknowledged position in surgical works, are as unsurgical as they

are frivolous;—*post-mortem* conditions, such as are not *appreciable* by *clinical* observation; without which conjunction, Pathological Anatomy avails no more than the blind guide of Empiricism.

CHAPTER IV.

PATHOLOGICAL ANATOMY APPLIED DURING LIFE TO SUPPLY THE EARLIEST AND MOST EXACT DIAGNOSIS.

The Diagnostic Value of Clinical-Pathological Anatomy, illustrated by the Diagnosis of the Varieties of Dislocation.

THE *application* of Pathological Anatomy to Diagnosis implies the possibility of detecting and distinguishing the various kinds of morbid conditions, *during life*, whether they be structural, physical, or chemical alterations, which the organs and textures of the body may severally have undergone. How, then, can such alterations, removed, as for the most part they are, from direct appreciation by the senses, be detected and distinguished in the clinical observation and study of disease?

Firstly, and most exactly, by examination of the morbid products and secretions discharged from the natural passages—as the mouth, œsophagus, stomach, and intestines; the lungs, urinary bladder, kidneys, uterus, and vagina; those, also, which are externally yielded by or through the skin, or procured by puncture, as from tumours; all of which materials, coming from the diseased organ or texture, represent its pathologico-anatomical condition, and this, possibly or probably, at a period sufficiently early in its course to allow of therapeutic measures being employed with the most reasonable hope of success. This *direct* application of Pathological Anatomy is necessarily restricted to the minute structural and chemical conditions of *disease*, as distinguished from injury, and its value will be fully discussed and illustrated in future chapters.

But morbid products and secretions may not be discharged, or be procurable by puncture, at a sufficiently early period for therapeutic purposes; or, the physical conditions and structural relations of organs may be the object of clinical investigation. Under these circumstances, we are compelled to accept the less certain diagnostic evidence afforded by examination of the various textures and organs, as they lie buried and concealed during life by their integuments and surrounding parts.

Injuries are mostly in this disadvantageous diagnostic condition.

The value of any diagnosis, under circumstances so unfavourable to its completion, should be estimated by the comparative frequency and exclusiveness with which certain external characters (*i. e.*, signs) are found to be connected with the same essential internal condition—a connexion which can only be established by repeated observation during life, being as repeatedly corroborated or rectified by post-mortem examination. The knowledge thus guaranteed can be applied with confidence. Pathological Anatomy then speaks out, as it were trumpet-tongued, during life, in terms too plain to be misinterpreted, and as the earliest reliable announcement of a disease or injury, the mere functional symptoms of which are but as whispers.

This *indirect* guidance of Pathological Anatomy, through *signs* during life, may be inferred from the previous chapter; but the diagnosis of Dislocation very clearly illustrates its (diagnostic) *value*. The structural relations which constitute dislocation are made known by certain external 'physical' signs, and they supply at once the *earliest* and *most exact* ground of diagnosis, whereby, also, the varieties of this injury can be distinguished.

Dislocation signifies a displacement of the articulatory portion of a bone from the surface on which it was naturally received. This lesion is generally accompanied with pain and an inability to use the joint; the anatomical prominences of bone (condyles) contiguous to the joint, become in part less, in part more, perceptible; its normal configuration, therefore, is more or less distorted; the limb is, moreover, usually shortened, occasionally elongated, and its axis

diverted, but not by any voluntary movement, for the limb becomes fixed and immovable; and, lastly, when handled near the dislocation, it does not communicate that peculiar sense of rough crepitation which is so characteristic of fracture. These, the 'physical' signs of dislocation, are happily trustworthy evidence—more especially the *form* of the joint, the *length* of the limb, and the *direction* of its axis; to which may be added, as a negative sign, the *absence* of true crepitation.

These signs can be shown to be immediately dependent on dislocation, with which they are, in different degrees, invariably and exclusively connected, and of which, collectively, they may be regarded as the most exact and earliest reliable exponents. But to discern their diagnostic *signification* it is necessary to have acquired a correct knowledge of the general appearances which a dislocated joint presents on *dissection*.

The appearances in question are restricted to dislocations as ordinarily understood, in which the bones composing the articulation are held together by ligaments, and the joint admits of more or less free motion. Displacements of the cranial bones, or of the vertebræ, excepting the two upper cervical, and those of the pelvic bones, are therefore excluded from our present consideration, and I shall confine my remarks to those dislocations which the joints of the *extremities* undergo; and, moreover, endeavour to bring, as far as possible in the form of a regular series, the appearances found on dissection, with those signs by which the fact of dislocation having occurred is surely made known during life.

But dislocation *per se* seldom proves fatal, and this circumstance limits the opportunities for making those more exact observations which the scalpel can alone discover, and of acquiring that more accurate knowledge which can alone be gained by the dissection of a recent case; while, on the other hand, in the event of an old unreduced dislocation being examined after death, sufficient time may have elapsed, since first the accident occurred, to have allowed of certain reparative changes by which the parts then displaced are, not indeed restored, but mutually adapted to their new

positions, and reconciled to the injury they formerly sustained. Then, again, *all* the joints of either extremity are not equally liable to dislocation, and this additional circumstance further restricts our pathological knowledge (in the event of an opportunity for post-mortem examination) to such knowledge as can be obtained by the dissection of *particular* dislocations.

To estimate the bearing of this remark, we should remember that certain circumstances, whether normal or acquired by disease, predispose to dislocation. The anatomical and physiological conditions predisposing, are—the shape of the articular surfaces, laxity of the ligaments, feeble tonic power, or paralysis, of those muscles by which the articular portions of bone are held in apposition; and the length of the bones articulated; this latter condition having reference to muscular action as conducive to dislocation.

The ginglymoid (hinge) joints cannot, by reason of the shape of their articular surfaces, allow of so free a range of motion as the orbicular, and their ligaments are stronger. The knee, ankle, elbow, and wrist joints, for example, are more secure than those of the shoulder and hip; and the articular surfaces of the former allow of motion only in two directions, forwards and backwards. Their dislocations are therefore met with less frequently—far less frequently, indeed, than those of the shoulder-joint, in respect of which all the conditions predisposing to dislocation conspire to render this joint the most liable.

But opportunities for dissecting an *orbicular* joint dislocation —such as that of the shoulder-joint—in a *recent* state, are rare. True it is, that certain conditions acquired by disease, further predispose to dislocation. When the cartilages and bones of an articulation are softened by caries, and fretted away by ulceration, the ligaments soddened and loosened by suppurative discharges, and those muscles which aid in retaining the articular surfaces in apposition, are paralysed, dislocation is imminent; but the structural relations thus acquired by disease, are an inappropriate illustration of those which constitute dislocation, as the result of injury. Apart from such cases, our knowledge of the

appearances which dislocations present on dissection, has been unavoidably limited chiefly to the post-mortem examination of *old unreduced* cases; or of those—more favourable for observation—being recent, but which have been complicated by *other severe injuries*, and have occasionally terminated fatally.

The general results of dissections, made under these circumstances, will be described most advantageously, by taking the pathologico-anatomical conditions concurrently in connexion with the signs by which they are manifested during life.

Starting then with the first, because the most essential pathologico-anatomical condition of dislocation,—that of *displacement*, what is *its* sign?

The dislocated bone is dislodged from its natural relation to the companion bone or bones. Such displacement may be *complete*; orbicular joint dislocations are usually so, as those of the shoulder and hip, of the heads of the metacarpo-phalangeal bones backwards, and of the head of the astragalus from the scaphoid bone, occasionally. On the other hand, ginglymoid articulations, such as those of the knee, elbow, ankle, and wrist, are more subject to *incomplete* displacements of their component bones. And this is also due to the peculiar shape of their articular surfaces. In fact, the same anatomical condition which regulates the range of motion of a joint, and thereby its liability to dislocation, also predisposes to the more or less complete displacement of its articular surfaces, when dislocated.

But the fact of *displacement*, of whatever degree, is the essential element of dislocation, and that which the very term itself is understood to express. The alteration of structural relations, thus implied, is accompanied by a corresponding defacement of the *outline of the joint*, and this sign is the earliest and most unequivocal announcement of dislocation. The significance of this most important sign was fully recognised by Sir A. Cooper when he observed, that the natural prominences of bone near the joint either disappear, or become less conspicuous; as, for example, the trochanter at the hip joint. Sometimes the reverse

occurs; for in dislocations of the shoulder the acromion projects more than usual.*

The discrimination of dislocations and fractures in the neighbourhood of joints—with which injuries dislocations are most likely to be confounded—can be determined in many cases by observing the particular contour which the joint presents: in illustration of which diagnosis I may allude to instances of dislocations of the hip joint, as compared with fracture of the neck of the thigh bone; dislocations of the shoulder joint, as contrasted with fracture of the anatomical neck of the humerus; dislocation backwards of the radius and ulna, compared with fracture of the epiphysis of the humerus; and dislocations of the wrist joint, in relation to the appearances of fracture transversely through the lower articular end of the humerus.

Certain exceptions must be taken to the apparently unconditional value of that evidence which an examination of the joint itself contributes. The ginglymoid articulations, admitting as they do of motion only in two directions, present the most obvious deformity when dislocated. Moreover, certain of these joints have contiguous processes of bone (condyles) so prominent, that any alteration of their natural relative position can be easily felt. The orbicular articulations are less favourably circumstanced. The free range of motion which they naturally enjoy allows of their assuming every possible altitude without the suspicion of dislocation being suggested; and their bony processes are, if not smaller, at least less perceptible through the depth of the soft parts beneath which they lie buried.

These anatomical difficulties are frequently increased by the supervention at an early period of considerable effusion and swelling, which quickly conceals the boundaries of the articulation. The enveloping cushion thus formed of swollen soft textures may be too thick to transmit any very exact impressions, and also too sensitive to allow of much handling, while it encompasses the joint

---

* Surgical Essays, part i. p. 4.

so soon after dislocation as, possibly, to preclude the opportunity for making an immediate diagnosis. The peculiar shape which the joint assumes may thus become an inappreciable sign.

Let us, then, again refer to pathological anatomy for further evidence. In what condition are the *ligaments* found by dissection of a recent dislocation? On this point, reflection will almost anticipate the results of experience. When we consider how closely the ligaments of each joint are adapted to the particular shape of the articulation, and that with one notable exception—the shoulder joint, they are sufficiently tight to retain the bones in apposition, even without the assistance of muscular action; and, when we further consider how far removed the head of the dislocated bone is felt to be from its natural locality, it appears more than probable that the ligaments must be ruptured to have allowed of such displacement of the articular surfaces.

But what says dissection? Howship was led by experience to believe that in some instances rupture of the capsular ligament takes place, but not in the majority. This opinion is not supported by the experience of other observers. Two authentic cases of reduced dislocation of the femur on the dorsum ilii, are worthy of notice. One was accompanied with an injury of the head, which proved fatal the day after the accident. On dissection, the orbicular ligament of the hip which had been dislocated, was found entire at the superior and anterior part only, and irregularly lacerated throughout the remainder of its extent. The "inter-articular" ligament was torn out of the depression on the head of the femur.* The other case supplied similar testimony. Death from injury to the intestines ensued within forty-eight hours after the dislocation, and forty after reduction. The internal and upper part of the capsular ligament was ruptured; the external portion remained unbroken. The round ligament was torn, as in the preceding case, from its insertion into the depression on the head of the femur. The particular portion of the

---

* Dub. Hosp. Rep., C. H. Todd, vol. iii. pp. 396—401.

capsular ligament which was here torn does not affect the general question, and I pass on to other instances respecting the same form of ligament, but in which the dislocation had *not* been *reduced* before death. Two cases, in which the humerus was forced down into the axilla, as recorded by Sir A. Cooper, are illustrative. In one the capsular ligament was found torn along the whole of the inner side of the glenoid cavity, and the opening would have admitted a much larger body than the head of the humerus. In the other case, death had ensued apparently from injuries inflicted at the time of attempted reduction, and the dislocation was five weeks old when dissected. The capsular ligament had given way in the axilla between the teres minor and subscapular muscles. A third case of similar injury occurred in the practice of Sir Philip, then Mr., Crampton. The capsular ligament was found completely torn from the lower part of the neck of the humerus to the extent of more than half its circumference. I may add, that in an unreduced dislocation forwards, the head of the bone was observed to have passed out through a rent in the capsular ligament over the upper edge of the tendon of the subscapular muscle.* Lastly, the dissection of another dislocation showed that the same ligament was completely separated from the entire circumference of the humerus.†

These dissections lead to this general inference: that if a capsular ligament so loose as that of the shoulder-joint has been found thus extensively lacerated in various forms of dislocation, we may reasonably presume that the ligaments of other articulations *less lax* would also be found ruptured under similar circumstances if opportunity permitted of examination by dissection.

Taking for granted that the *ligaments* are *extensively lacerated by the act of dislocation,* in what way does *this* condition, discovered by post-mortem examination, supply the earliest and most exact ground of diagnosis during life? By virtue of the

---

* Dub. Journ. Med. Science, Nos. 7 and 8.

† Med. Obs. and Inq. vol. ii. p. 349, Thompson.

constant and exclusive connexion of this condition with certain external signs, and its rational explanation of these signs.

The bone having been *violently* dislodged from its natural tenement, is suddenly *plunged through ruptured ligaments* into tender, soft parts, and is then and there delivered over to the dominion of those muscles which soon imprison it in its new abode. Encompassed as the dislocated bone now is by nerves and muscles, among which it has been rudely thrust, the pain may be exquisitely acute; or the natural sensibility being numbed by continued stretching of the nerves, partial paralysis may ensue. Hence the powerlessness of a dislocated member. But the muscles soon resent the injuries they have received. The displaced bone having lost its companion articular surface, upon which it had long glided smoothly with supple mobility, is now at the mercy of the muscles, and they, by their tonic contraction, gradually induce that fixed immobility which betokens dislocation. The importance of this muscular contraction as *the* persistent cause of displacement, was first advocated by Pott; and many conclusive proofs can be adduced. Dissection shows that at least some articular surfaces are held in apposition by the action of muscles rather than by ligaments; witness the humero-scapular articulation; the capsular ligament of which is weak, and would allow the humerus to drop down somewhat, were it not for the tonic contraction of those muscles which brace up this joint. When dislocated, it is therefore reasonable to suppose that these same muscles will retain and maintain the displacement, and perhaps act with even greater mechanical advantage.

I remember, during my student days, a dislocation of the hip backwards into the sacrosciatic foramen, which had been produced artificially in a dead subject, at the University College, for the purpose of some observations which Mr. Quain was then making on dislocations of this joint. The integuments were removed, and the muscles, in the neighbourhood of the joint, displayed. I was one of many assistants to reduce this dislocation, and our united efforts were required, for some time to effect its reduction,

to overcome the resistance of the *rigor mortis* of the opposing muscles, although their tonic contraction must have been partly subdued in producing the dislocation.

To these more direct proofs respecting the point in question, may be added the fact, that, if a weakened or paralytic condition of the muscles concerned favours dislocation and its reduction—allowing movements *out* and *in*, so to speak, surely, that amount of tonic contraction which usually supervenes on dislocation, will fix and perpetuate the displacement. Agreeably to this view, various antispasmodic medicines, such as chloroform, opium, and other narcotics, which relax muscular contraction, favour reduction; and injuries inducing collapse, have the same tendency. Sir A. Cooper* mentions an instance, in which a man, having a dislocation of his hip-joint, had also an injury of his jejunum, and the bone was very readily reduced.

Under ordinary circumstances, however, the joint when handled, feels locked; or, if still slightly moveable, does *not* communicate the *grating crepitus* of fracture. But pathological anatomy telegraphs, by other and more *obvious* signs, the fact of dislocation. The head of the displaced bone is removed to some distance from its natural situation, and therefore the *length* of the limb is altered, and most appreciably so if the dislocation be complete. The limb is lengthened or shortened as the head of the displaced bone happens to be lodged either below or above, the level of the articular surface on which it naturally moves.

The form of the articulation will itself suggest whether it be possible for either lengthening or shortening to accompany a dislocation, or whether the latter can alone occur. An orbicular joint allows of a displacement in *any* point of its circumference. It is sufficient for practical purposes to recognise only four points of its compass. Thus, the femur may be dislocated upwards on the dorsum ilii, downwards into the thyroid foramen, forwards and slightly upwards on to the ramus of the pubes, backwards and

---

* Surgical Essays, part i. p. 20.

slightly upwards into the ischiatic notch. The three upward dislocations must obviously be attended with more or less shortening of the limb, the remaining one, downwards, being as evidently accompanied with elongation. The other great ball and socket joint may undergo three analogous dislocations; downwards into the axilla, with elongation of the arm, forwards, less frequently, on the inner side of the coracoid process, and very rarely backwards on to the dorsum of the scapula below its spine; the two latter displacements being attended with some shortening of the extremity, but in a less degree conspicuous when the displacement is downwards. On the other hand, ginglymoid joints cannot, owing to their construction, be subject to dislocation other than in three directions—backwards, forwards, and laterally. The two former displacements are necessarily productive of shortening, the latter dislocation does not, if incomplete, attract attention in this respect. Witness an illustration of these results, shortening of the leg by dislocation of the knee, and of the fore-arm by various displacements of the elbow-joint. But if the natural form of an articulation suggests the *possibility* of lengthening or shortening of the limb occurring, how much more clearly will dissection reveal whether the head of the displaced bone is lodged below or above the level of the articulation. An exact knowledge of this pathological condition supplies the only true explanation of that alteration in length—be it elongation, or shortening—which the limb has undergone; and this change (having been found by repeated observations to be constantly connected with dislocation) becomes one of the surest and earliest signs during life of that condition to which it points.

Yet, as if to demonstrate the insufficiency of *any one* feature for the identification of a disease or injury, clinical experience shows that fracture may, so far as the length of the limb is concerned, simulate dislocation. In cases of impacted fracture, the natural length of the limb remains unaltered; but in those of the unimpacted with displacement, the limb is necessarily drawn up by muscular action, and shortened. So also, and for a similar reason, all dislocations of ginglymoid joints, *forward* and *back-*

wards, and those also of the orbicular articulations, *upward,* present a similar appearance. It is therefore between *ordinary* cases of fracture, and *certain* dislocations, that the line of distinction should be drawn. The mobility or immobility of the limb supplies this distinction. With fracture, the limb can be drawn down to its natural length, returning, when left alone, to its former position; but with dislocation, the limb remains immoveably fixed, and cannot be temporarily relieved from its crippled dimensions.

Lastly, the same pathological knowledge which explains the meaning of other well-known signs of dislocation, also explains the *particular direction* which the *limb* assumes. Its direction is peculiar, and only to be interpreted by an exact knowledge of the new relations to muscles which the displaced bone has acquired.

Certain muscles are found to be thrown out of action by the displacement of that portion of bone to which they are attached, while others, enjoying a mechanical advantage, preponderate. The natural balance of opposing muscles—*e. g.*, flexors and extensors, is therefore lost, and the limb acquires that particular attitude to which the predominant muscles direct it. Should perchance this attitude suggest the suspicion of fracture, we again have recourse to the recollection that were such the case, the limb would admit of being restored to its original position, but that, being dislocated, this cannot be accomplished. The limb thus fixed in a peculiar position, is also elongated, or shortened, as the case may be, while the joint itself, painful and swollen, presents a misshapen outline. With these deformities, the noble proportions of the athlete are exchanged for the crippled distortion of an acrobat or mountebank.

Such is a diagnostic sketch of dislocation, guided by Pathological Anatomy; and should the displacement remain unreduced, we are further led by this guidance to anticipate the nature of those changes which supervene, and to interpret the diagnostic signs *then* present.

If the displaced bone be lodged upon muscle, it gradually burrows for itself a convenient nest, the two surfaces become mutually adapted to each other, and a capsular ligament being formed of condensed cellular tissue, an imperfect joint is established.

But should the bone have found a resting-place on bone, the one loses its periosteum, and the other its cartilage, a receptacle is excavated suitable to the impression of the displaced articular surface, a bony rim or lip is thrown up, by the periosteum, around the margin of this newly-formed cavity, the surrounding cellular texture moreover becomes condensed, so as by forming a capsular ligament, to further provide against any displacement; and thus a far more perfect joint is constructed.

In either case, the muscles which act on the displaced bone, also retain it in its new position, and, becoming permanently shortened, their lines of action get accommodated to the displacement. The natural articular cavity, from whence the bone was dislodged, loses its cartilaginous *facet*, and closes in. It is at length partially obliterated by a dense fibrous deposit. But very slowly, indeed, do these destructive changes proceed; and Nature reluctantly closes the original cavity, and with it, the opportunity for reduction, only when, long disappointed by delay, and wearied by the lapse of time.

These reparative pathological conditions throw out sure signs, during life, by which they can be recognised. The repaired mechanism tells its own tale. The limb being set at liberty by the formation of a new articulation, is now tolerably moveable (and moved by the individual), and resumes more its natural position; but it nevertheless remains unnaturally lengthened or shortened, and the articulation, although perhaps painless, retains its unsightly appearance resulting from displacement with some swelling.

Such is the history of 'simple' dislocation—of dislocation unattended by other injuries than displacement with its immediate accompaniments—and such the all important guidance of Pathological Anatomy to the earliest and most exact diagnosis. The same method of diagnosis will overcome difficulties far greater than these. Pathological Anatomy proclaims during life the nature of those dislocations which are 'complicated' with other injuries.

In sad contrast with those reparative changes by which a new articulation is constructed, and compensation in a measure made for the loss of the natural one, are certain destructive changes,

which peril the joint, even should reduction be attempted in time, and successfully accomplished. I have already hinted at some of these additional injuries. All the parts around the dislocation are liable to be involved in the injury. Certain muscles put upon the stretch —*e. g.*, the pectineus and adductor brevis, by dislocation of the thigh downwards—may be lacerated. Even their unyielding tendons are sometimes ruptured, as the subscapularis tendon, by dislocation into the axilla. Large bloodvessels occasionally share the same fate, accompanied with hæmorrhage and livid swelling; a main nerve also may be torn asunder. Fracture of the head of the bone is another complication. This is more likely to occur in dislocation from direct violence; for example, by a fall on the hip or shoulder, the neck of either bone is perilled; the olecranon may be knocked off; the bulky head of the tibia shattered; or the tarsal end of the tibia bruised and broken.

These surrounding complications of dislocation are Pathologico-anatomical conditions, which can be severally detected and distinguished by their physical signs, or, less certainly, by functional symptoms. Of all encompassing complications, none is more dangerous than a wound extending through the skin and intervening soft parts, *into* the dislocated joint, exposing the articular surfaces, which are not unfrequently protruded by the violence of the injury. This 'compound' dislocation throws open the cavity of the joint, and at once declares the kind of injury; or the escape of synovia is a sign, and the diagnosis can be completed by introducing the finger. As compared with simple dislocation, the compound lesion implies far more extensive laceration of the soft parts, and their exposure to the irritating influence of the air; this additional disorganization being occasioned either by further displacement of the bone and its transit through the soft textures and skin, successively, or by a wound inflicted from without, accompanying the dislocation. In either way, certain joints are more subject to compound dislocation. The hip-joint, very rarely; the shoulder, now and then; the knee and elbow, frequently; and the ankle, constantly.

Compound dislocation is also distinguished by more active

inflammation supervening, followed by suppuration and sloughing; but this, its course and tendency, is due to more extensive laceration of the soft parts, and their exposure to the air. *This*, then, is the essential distinction between 'simple' and 'compound' dislocation, and it refers their diagnosis to pathological anatomy. 'Complicated' dislocation has a similar signification. The history of wounds, fractures, and aneurisms, severally, teaches the same lesson, and extends it into a generalization. Wounds are 'adhering' or 'sloughing' essentially by virtue of their respective pathologico-anatomical conditions. Fractures are, in like manner, 'simple,' 'compound,' or 'complicated.' Aneurisms, again, are 'circumscribed' or 'diffused,' either of which may be *traumatic* or *idiopathic*, by virtue of a diseased condition or otherwise of the aneurismal artery. All these distinctions are open to clinical observation.

Pathological Anatomy, therefore, thus speaks out *during life*, and, by infallible signs, supplies at once the most exact and earliest reliable ground of diagnosis.

CHAPTER V.

CLINICAL PATHOLOGICAL ANATOMY, AND ITS DIAGNOSTIC GUIDANCE.

Pathological Anatomy applied during Life through Physical Signs, to supply the earliest and most exact Method of Diagnosis.—The Value of Physical Diagnosis, illustrated by the Diagnosis of Morbid Products of Nutrition; *e.g.*, Fatty Tumours, Cysts and Cystic Tumours, respectively.

Having established the guidance of Pathological Anatomy as the fundamental Principle of Diagnosis, I now proceed to *estimate* its three aspects in relation thereto; and, firstly, through Physical signs, constituting Physical Diagnosis.

The language in which Pathological Anatomy makes known the various kinds of disease, during life, is spoken in three dialects. The 'physical,' the 'structural,' and the 'chemical' conditions of disease, give utterance, each, by a modulation of that voice, as it were, whereby Pathological Anatomy speaks out at the bedside, and these

expressions are, as 'signs' of disease, represented by as many methods of detecting and distinguishing diseases. Hence, the Physical, the Structural (chiefly by microscopic examination), and the Chemical methods of Diagnosis.

My illustrations of the first of these methods will be drawn from the physical characters of certain tumours, thus applied. Although I have selected *morbid growths* from the wide domain of Pathology, yet the Principle represented by their physical diagnosis admits of more general illustration. Just as the exemplification of each preceding chapter might readily be extended, and, moreover, in support of other Principles than that for the elucidation of which such evidence was adduced, so also the one in question might be gathered from other branches of Pathology, and immediately from the last chapter, of which it is a further illustration; but, in pursuing my plan, I have selected that kind of evidence which seemed best fitted to *specially* exhibit the full import of the particular Principle under consideration.

The physical characters of certain tumours clearly show the *competency* of Pathological Anatomy to supply the earliest and most exact method of diagnosis through 'physical signs;' and perhaps no more familiar, yet conclusive, evidence in support of this position can be adduced, than that which the 'fatty' and 'cystic' tumours which contribute, by virtue of their external characters and properties.

Take, first, the fatty tumour. I do not refer to those cases of local accumulation of fat—fatty hypertrophy of parts—that are occasionally met with. I was lately shown a remarkable case of this kind by my colleague, Dr. Cockle. The breasts of a young woman, aged nineteen, were immensely overloaded with fat, each weighing about eleven pounds; yet these large and ponderous masses, tumour-like in appearance, were *continuous* with the natural fat around. The line of demarcation was inappreciable. A true fatty tumour, not being continuous with the surrounding fat of the part in which it is imbedded, has a circumscribed and well-defined outline. The fat (of the tumour) is enclosed within an investing capsule of cellular tissue, somewhat condensed, yet loosely con-

nected with adjoining tissues. Such a tumour is, therefore, freely moveable as well as circumscribed. But its cellular tunic is not only an isolating barrier; prolongations penetrate far within the mass, and separate its substance into portions or lobes of various sizes and shapes. The fatty tumour, therefore, presents an irregular lobulated surface. This isolated and lobed collection of fat usually attains a large size, and has a proportionate weight. One in the museum of St. Thomas's Hospital, which was removed from the abdomen of a man by Sir A. Cooper,* weighed no less than thirty-seven pounds, ten ounces; but those of heavier weight have been met with. It is difficult to connect this physical character of the fatty tumour with any peculiarity in the nature of the growth itself, further than by remembering that, of all the normal tissues, fat is most readily produced.

Besides these physical characters—defined outline, lobed shape, and free mobility,—certain physical properties are perceptible, such as a mass of fat might be presumed to possess. The tumour is soft and doughy, yet not without some degree of elasticity and resiliency under the touch. To conclude this interpretation of physical signs, the fatty tumour is usually situated where fat itself naturally most abounds subcutaneously, and about the trunk, frequently on its posterior aspect—for example, on the shoulders; and very seldom seen in those parts where fat is scarcely found, and therefore not in the eyelids, scrotum, and so forth. A remarkable instance is recorded† of a fatty tumour situated within the scrotum, behind the testicle and unconnected with it. The mass could be distinctly traced to the abdominal ring, and simulated an omental hernia.

One particular event which not unfrequently occurs in the physical history of fatty tumour, respecting its situation, is worthy of recollection. Owing to the looseness of connexion between the investing capsule and adjoining parts, the mass, more especially if

---

* Med.-Chir. Trans., vol. xi., p. 440.

† Lectures on Pathology and Surgery. B. C. Brodie. Edit. 1846, p. 271.

weighty, may shift from its original locality, and find a new resting-place. In the case just mentioned, the tumour apparently had its origin in the adipose substance connected with the spermatic cord within the abdominal ring; but, as it increased in size, it made its way in the direction of least resistance—namely, into the loose cellular structure of the scrotum.

Lastly, fatty tumour is, generally speaking, solitary, being without companion far or near in the body; and this feature, in what may still be called its physical history, alone remains entirely unexplained at present by Pathology. Otherwise, it is not difficult thus to interpret the ordinary physical signs of an ordinary fatty tumour; nor, moreover, is it difficult to perceive the value of these signs for the purposes of diagnosis, by virtue of their indicating with certainty, and at the earliest period, the pathological condition from which they emanate.

But the physical characters of fatty tumour are not always certain. They are occasionally inconstant. Although always present and perceptible in the case of a tumour purely fatty, save when disguised by the depth of integuments under which the mass lies buried; yet the ordinary physical signs which I have enumerated are occasionally wanting, and nevertheless the tumour is fatty.

The cellular investment is sometimes thick, dense, and fibrous, and adherent to the adjoining tissues. Its prolongations may also become fibrous and firm. The whole substance of such a tumour is therefore hard and comparatively immovable; and although still lobulated and circumscribed, more nearly resembles a fibrous, rather than a fatty tumour. The *lardaceous* variety is of this kind. I once removed from a man's cheek an oval-shaped, hard mass, about the size of a pigeon's egg, and which turned out to be fibro-fatty.* Hard fibrous knots, and even bony nodules, can occasionally be felt within a tumour otherwise fatty. On one occasion I found two or three of these lumps in a large fatty mass situated on the right flank and ilium; and similar cases are mentioned by Paget.†

---

* Lancet, vi., 1856. † Surgical Pathology, vol. ii., p. 99.

On the other hand, the mass of fat may be or become softer than usual. The fat is more oleaginous, and even the fibro-cellular partitions are slighter than usual. The whole substance resembles a bag or cyst of fluid, and gives out the physical signs of a fluid encysted tumour. Moreover, true cysts are mentioned by Paget, as being occasionally developed in a tumour otherwise fatty, and which has the ordinary fatty consistence. Cysts so placed, feel like roundish elastic bags of fluid set in a doughy substance, and are thus readily distinguished. Still more rarely, suppuration occurs within a fatty tumour, and forming a chronic abscess, simulates the character of a cyst.

Whenever a tumour, presumed to be fatty, affects either of these extremes—becoming either more firm and fibrous or softer than usual and cyst-like—then, indeed, it may be impossible to complete the most exact diagnosis through the medium of physical signs. Or, again, if the supposed fatty tumour be deeply buried beneath the integuments, its ordinary lobulated surface, defined outline, and mobility are not perceptible. In one instance within the experience of Sir B. Brodie,* the tumour lay underneath the trapezius muscle; in another case, beneath the breast; "one thought that it was a fungus hæmatodes, another thought that it was something else, and a third could give no opinion at all." A still more doubtful case was this:—a tumour presented below the armpit, and was apparently about half the size of an ordinary orange; however, an incision disclosed an enormous mass, which proceeding from the axilla, extended thence far backwards into the space between the scapula and the ribs. It was found impossible to dissect out the whole of the tumour.

But it should ever be remembered that these are *exceptional* cases, and that, apart from them, the knowledge of Pathological Anatomy, as applied during life by physical signs, fails not to fulfil the most exact diagnosis.

More than probably, as much may be said in favour of the earliest diagnosis, also, by this method. The variations of con-

---

* Op. cit., p. 271.

sistence to which fatty tumour is liable, and which tend to perplex the accuracy of its diagnosis, apparently do not supervene until a somewhat late period of the growth. If examined in an early stage, fatty tumour more uniformly possesses the ordinary physical characters by which it is readily distinguished. For example, if the tumour be firm and unyielding, and with closer attachments than usual to adjoining tissues, it will in all probability have acquired these characters by the fibrous thickening of its investing capsule, just as the tunica vaginalis of an old hydrocele becomes thickened, or an irreducible hernia acquires an unyielding fibrous sac. Probably, also, the hard fibrous knots occasionally found within the substance of a fatty tumour are formed by absorption here and there of the fatty portion, and a corresponding development of the fibrous element by a chronic process bordering on inflammation. Pressure has this effect, and a case in point is mentioned by Mr. Paget,* in which, the patient being a washerwoman troubled with a large fatty tumour, fibrous knots were developed in that portion which rested against the clothes-basket she frequently carried. The oleaginous fluidity and cyst-like condition of fatty tumour is also, I am inclined to believe, not found at an early period or stage of this growth. A central chronic abscess is certainly a subsequent condition.

Taking all these reflections into consideration, we are warranted in concluding that the earliest, no less than the most exact, diagnosis of this kind of growth can be completed, except under occasional circumstances, through 'physical signs' alone, as interpreted by a due knowledge of Pathological Anatomy. Nor is this method deprived of much of its practical value by the circumstance that a fatty tumour may be deeply imbedded rather than subcutaneous, whereby its lobulated surface, circumscribed outline, &c., are imperceptible; for this also is an exceptional condition.

Lastly, we should remember that *exceptional* cases do not impair the *general* value of physical diagnosis, as illustrated by fatty tumour; and that, on the other hand, when the ordinary physical

---

* Op. cit.

signs *are* present, they *exclusively* indicate this kind of growth, and no other morbid condition.

The *purely* fatty tumour invariably gives out the same physical signs, and no other morbid condition gives out these signs—namely, a mass of doughy consistence, with a defined outline, lobed more or less, and freely moveable; usually subcutaneous, probably somewhere about the trunk, posteriorly, and solitary. Moreover, these signs are well pronounced at the earliest period of the growth, and can be recognised during life.

The diagnostic competency of Pathological Anatomy, through the medium of 'physical signs,' thus exemplified by the diagnosis of fatty tumour, is further and fully illustrated by that of cysts and cystic tumours.

A simple or barren cyst, with its fluid contents, necessarily implies a circumscribed and fluctuating tumour, or the resistance only of fluid pressure. Such are the physical characters presented by a *serous* cyst, for example, which may be regarded as the type of fluid (barren) cysts. Similar signs, varying only in degree, are given out by all other cysts whose contents are fluid. Sanguineous cysts; enlarged (synovial) bursæ; adventitious ganglia, so often seen on the back of the hand; mucous cysts—*e. g.*, surcharged Nabothian follicles about the neck of the uterus, or distended Cowper's glands just within the orifice of the vagina; the less solid fatty cysts—*e. g.*, certain wens, and the seminal cysts occasionally found attached to the spermatic cord, of which kind are "encysted hydroceles;" these and other varieties of fluid cysts are known and distinguished from other tumours by their even, circumscribed outline, and more or less fluid resistance to the touch. A collection of such cysts has similar characters. But the cystic growth deviates considerably from this standard, and its physical characters vary accordingly. Indefinite degrees of consistence are found, and correspondingly different degrees of resistance, from that of the most fluid serous cyst to the most solid-feeling, thick-fluid, as grumous blood, synovia, mucus, or the butter-like substance of sebaceous cysts.

Subject to these original deviations, the typical condition of the cystic growth is that of one or more membranous bags, filled with some kind of fluid; and, as such, its physical characters supply a complete diagnosis, both in respect of exactitude and earliness. Other conditions (not deviations) are *exceptional* and *subsequent* productions. The 'proliferous' development of a solid growth within a parent cyst is one of these changes.

Cysts undergo this change most commonly in the mammary gland and thyroid body. Bronchocele occasionally contains proliferous cysts; in sero-cystic disease of the breast, they are more frequent.

The observations of Sir B. Brodie* supply sufficient data from which to gather the general bearing of this (proliferous) change on the question of diagnosis.

First: Membranous cysts, more or less numerous, are generated in the breast, each containing serum. The latter is, in the first instance, of a light yellow colour, and transparent, but afterwards assumes a darker colour, and becomes opaque.

Secondly: Morbid growths or excrescences are generated from the inner surface of one or more of these cysts, projecting into their cavities. These excrescences seem to consist of albumen or fibrin, which after some time (if not immediately) becomes organized.

There is some reason for believing that a similar growth of fibrinous substance may take place from the external surface of the cysts, connecting different cysts with each other.

Thirdly (that which bears most on the present question): Under certain circumstances the cysts become completely filled up with the morbid growths, so that their cavities are obliterated, the tumour being thus converted into *a solid mass*, in which, however, the remains of the cysts are perceptible; and this is the prelude to a still further change, in which the greater part of the cysts have wholly disappeared, a solid mass of an indistinctly laminated texture occupying their place.

Lastly, if one of the membranous cysts be artificially laid open, or if it burst from over-distension with serum, the fibrinous excres-

* Pathology and Surgery, Op. cit., p. 148.

cence from its inner surface, being no longer restrained by the pressure of the skin, increases in size, and protrudes externally in the form of a fungus, giving to the tumour a new and more formidable character.

But, prior to this new phase of the disease, we observe a tumour, originally fluid and fluctuating, converted into an unbroken, solid mass; and the important point to notice is, that this condition is a secondary occurrence, and therefore not calculated to interfere, in the first instance, with the realisation of an early and exact diagnosis, and this through the medium of those physical characters of outline and fluctuation which a cyst usually presents. The globular form of the tumour—observes Sir B. Brodie—and the impression which the fluid within it conveys to the fingers, in general supply the means of an easy diagnosis in the early stage of the disease. Nor, I may add, is the condition which he has described the source of much embarrassment. The change from a fluid to a solid tumour is a gradual process, and during this period Pathology fails not to announce, by certain physical signs, the nature of the tumour and of the changes which it is undergoing. The growths within the cyst, not yet completely filling its cavity, are immersed in serum, and the whole feels a mixed tumour—partly fluid and partly solid. The authority I have quoted mentions such cases. In one cyst, the size of a large walnut, a fourth of its cavity was occupied by an irregularly shaped excrescence attached to one portion of its internal surface. A second cyst contained serum, but about one-third part of its cavity was occupied by an excrescence.

Another exceptional and secondary condition tending to obscure a physical diagnosis, is considerable *thickening* of the cyst.

This change, which resembles the thickening of the tunica vaginalis under the unremitting pressure of fluid in an old hydrocele, is only an illustration of a law long since observed by John Bell, and by which he endeavoured to explain the origin and unlimited growth of tumours in general—that it is the property of living matter to be thickened by use. Almost daily experience brings

with it proof to what extent the walls of a cyst can thus become thickened. In cutting through a common wen of the scalp, in order to turn out either half, I have found each portion equal in thickness to the shuck of a horse-chestnut.

A thickened cyst simulates the characters of other tumours. It resembles a chronic abscess. But, to justify this comparison, the abscess must be in that quiet condition which John Hunter described as being a "collection of matter without inflammation," and therefore without the pain, heat, and redness of inflammation, only swelling, with fluctuation. The interior of such an abscess is a pus-secreting cyst of some thickness, and its nature is so far questionable; but the pus-cyst is more blended with surrounding tissues, it is less circumscribed than a merely thickened cyst.

The physical diagnosis is more equivocal, when an ordinary cyst has actually become *converted* into a painless chronic abscess. A case in point is now under my care at the Royal Free Hospital. My notes tell me that "J. J., æt. 28, was admitted December 28th, 1859, with a tumour on the outer and anterior aspect of his right thigh. It is situated just below the anterior and superior spinous process of the ilium. The tumour was first noticed about eighteen months ago, and at that time resembled an egg in size and shape, but was soft and fluctuating. It had hitherto occasioned no pain or inconvenience, but as the lump gradually increased it caused considerable pain, extending downwards to the knee, at the same time affecting the action of the muscles in its neighbourhood, and consequently impeding progression. The swelling has now a well-defined outline, of the size and shape of a cocoa-nut. What is it? A circumscribed lobulated mass, freely moveable, soft, somewhat elastic, like a bag distended with some fluid, but from which, on puncture with a large-sized trocar and canula, no kind of fluid flowed, save a drop or two of blood;—an apparently solid tumour. Suffice it to say, that after a careful manipulation the swelling gave to my fingers the impression of being a fluid tumour, and as of a cyst deeply buried. My colleague, Dr. Cockle, whose judgment I sought, inclined even more decidedly

to this view. His opinion concurring with mine, I removed the tumour. A vertical incision eight inches long exposed the tensor vaginæ muscle and the fascia lata. The next incision brought me down upon a large cyst, and as by this cut the knife had slightly entered its lower end, there rolled out a quantity of thick sulphur-coloured fluid, apparently pus. This portion of the cyst immediately collapsed, but the upper and larger portion still remained quite full and tense. Further dissection showed that the cyst lay deeply underneath the tensor fasciæ and sartorius muscles, and on the vastus externus and gluteus medius muscles, from which, with some difficulty, I succeeded in removing it.

"The cyst turned out to be a bursa, much enlarged, and which, as it enlarged, had slowly suppurated and become converted into a sort of chronic abscess; but its interior did not present the pulpy mammillated pyogenic membrane, which characterizes an ordinary chronic abscess. Although the cyst-wall was of considerable thickness, its inner surface was smooth and shining like a serous membrane, with a little coagulated fibrin on it here and there, which readily crumbled under the fingers. A septum ran across the cyst, dividing its cavity into two compartments—an upper and a lower, of unequal size; and in this partition was seen a black, sloughy-looking mark corresponding with the aperture previously made by the trocar, and which having thence entered the substance of the partition, thus accounted for the anomalous fact, that from a fluid tumour no fluid should have issued when it was punctured."

The obvious inference from a full consideration of this case, and which bears immediately upon the present question of physical diagnosis, is this: *had* I discovered by puncture of the tumour, before the operation, that it contained pus, as if a chronic abscess, I could not have concluded by virtue of the physical characters of the tumour that it was a large *bursa* which had suppurated. The same inference was true also as regards a suppurating cyst which I lately removed from the face of a patient in the hospital. This cyst was the size of a large marble, and situated just below the margin of the right orbit, upon the infra-orbital foramen. The

man had been applying cobbler's wax, which had induced inflammation, suppuration, and thickening of the cyst—thus tending to obscure the diagnosis.

But, as touching the competency of physical diagnosis, it should be remembered that these suppurating cysts are *exceptions* to the general rule. A true cyst is rarely so thickened as to resemble a chronic abscess, and moreover, this suppurating condition is not the condition of a cyst at an early period of its existence. These two considerations redeem the value of physical characters in relation to the earliest and most exact method of diagnosis.

The same argument applies to other occasional cases in which the cyst having become thickened, and its contents perhaps being unusually consistent, the swelling assumes a general resemblance to some other kind of 'tumour' properly so called. The large bursa on the thigh, which I have mentioned, felt and appeared through the integuments not unlike a fatty tumour. I am sure that every surgeon of experience must have met, now and then, with a similar case, and where it has been somewhat difficult to determine the kind of tumour by its external characters alone. I have occasionally met with cysts which were so far thickened and lobulated as to have resembled a fibrous tumour.

But if the thickening and induration of a cyst sometimes misleads: its physical diagnosis becomes more obscure, should it, or indeed any cyst, lie buried within the substance of a solid tumour, the particular kind of tumour will of course make the difficulty of detection more or less; but in every case the intervening substance cannot fail to embarrass. Cysts are sometimes found set in the substance of a fatty, fibrous, or cartilaginous tumour, and perhaps no kind of tumour is exempt.

Lastly, the depth of integuments, underneath which a cyst may be situated, will more completely conceal its true character. The tumour which I removed from the thigh was beneath the fascia lata, the tensor fasciæ, and sartorius muscles; and these circumstances helped to obscure its diagnosis. I remember examining a tumour in the left popliteal space of an out-patient under the care of

Dr. O'Connor, at the Royal Free Hospital. It was deeply seated between the hamstring muscles, and pulsated with the artery, as if an aneurism; but it did not subside under pressure, nor when the artery was compressed in the thigh. On the other hand, this tumour was tense and elastic, like a bag of fluid would be if resisted on all sides and examined through a fascia such as closes in the popliteal space. These and other considerations led Dr. O'Connor and myself to concur in our opinion that it was a cyst. Accordingly, I punctured it as such, and, sure enough, out poured more than three ounces of a thick, glairy fluid. I am not aware whether the cyst filled again. In another case, an out-patient of mine at the hospital had a lump about the size and shape of a hen's egg, situated deeply in the muscles of the calf. It was scarcely moved by the action of the gastrocnemius muscle, and I concluded that it was *under* that muscle, and perhaps also the soleus. I fixed the supposed cyst with one hand, and sent in a trocar and canula posteriorly; there issued about two ounces of the same kind of fluid that flowed from the popliteal cyst. I could mention similar instances of deeply-buried cysts, but these will suggest their general diagnostic import.

The frequency of deeply-seated cysts, as compared with the superficial, is an open question.

All other conditions, tending to obscure the diagnosis of cysts (and cystic tumours), by virtue of their physical characters, are exceptional and secondary.

Apart from such cases, the physical characters of cysts are—as those of fatty tumour—constant. A circumscribed outline, and fluid resistance in some degree to the touch, invariably accompany and announce the presence of a cyst or collection of cysts. These characters are also peculiar; they point to no other kind of tumour or swelling apart from the cases adverted to. Moreover, they are readily perceived during life.

Thus do Physical Signs, interpreted as I have shown by Pathological Anatomy, at the bedside, supply the earliest and most exact method of Diagnosis. Such is the Principle of Physical

Diagnosis. I need not pursue its application. This has been done in practical works specially devoted thereto, and more particularly with reference to diseases of the thoracic and abdominal organs. The selection I have made of certain tumours will be conveniently followed by other morbid growths, considered in relation to Physical Diagnosis. This will lead to another Principle, the opposite to that which has been laid down.

The *insufficiency* of Physical Signs as a guide to the earliest and most exact Diagnosis.

Physical Properties are most readily recognised during life at the earliest period of disease (or injury), and therefore, it may be, are recognised at a time when the case is least complicated, and most remediable by the simplest measures: but such properties are nevertheless most equivocal (or inexact) signs of disease. Unaided Physical Diagnosis therefore fails in the practice of Medicine and Surgery.

This general Principle illustrated by the diagnosis of Morbid Products of Nutrition, as, quasi-malignant varieties of non-malignant Growths—*e. g.*, recurring fibroid and fibro-nucleated Tumours, respectively.

The Physical properties of morbid Structural conditions are indeed their most obvious attributes; but the co-existence of these properties with certain known conditions of structure is not invariable. This inconstancy falsifies, or at least impairs, the diagnostic value of Physical characters. Their *insufficiency* becomes more apparent when the evidence they afford is contrasted with that supplied by Structural characters.

To demonstrate this proposition, I shall select certain quasi-malignant or cancroid varieties of growth, the typical condition of which is undoubtedly non-malignant. Such are the *fibro-nucleated* (Hughes Bennett), and *recurring fibroid* (Paget), varieties of 'fibrous' tumour. The structural affinities of these growths will at once be seen in the following tabular view of their essential elements.

Fibrous tumour—Fibres, white } with nuclei.
Ditto elastic }

Fibro-nucleated—Fibres white, with oval nuclei.

Recurring fibroid—Fibre-cells; *i. e.*, elongated and caudate cells with nuclei.

What are the physical characters of these growths? The typical form of fibrous tumour is very hard, elastic, and mostly of a greyish colour on section, interlaced with white opaque bands variously arranged. Its minute structure is seen by the table to be that of ordinary fibrous tissue. On the other hand, the fibro-nucleated tumour only represents a rudimentary structural condition of fibrous tissue—viz., fine filaments infiltrated with many well-defined oval nuclei; yet their physical characters very much resemble those of ordinary fibrous tumours. These two conditions of structure cannot therefore be distinguished by any visible physical characters. Their diagnosis, however, is of great surgical importance; for the fibro-nucleated variety is very liable to recur again and again when removed, and we may be unable to determine our diagnosis until *after* an operation, the result of which may be only to induce the speedy return of a growth we had thus hoped to have effectually eradicated.

So also the recurring fibroid variety affords an equally appropriate exemplification of this diagnostic difficulty. Mr. Paget mentions one such tumour, situated over the upper and outer part of the leg, close to the tibia. Its size was that of a filbert, and it was considered fibrous when first removed in 1846. Some months after, another, as large as a walnut and decidedly fibrous, was removed from the same spot. In October, 1847, a third tumour, the size of a patella, was again removed. This was so like a fibrous tumour that, without the microscope, Mr. Paget would certainly have regarded it as such. Yet it consisted of nucleated fibre-cells—*i. e.*, nucleated cells elongated into imperfect fibres having a caudate or spindle shape—together with free nuclei, but very little filamentous tissue. Here then we observe cells developing into fibres—in fact, another rudimentary condition of fibrous tissue. A fifth tumour returned in the same locality, and was removed June, 1848. This also possessed the same fibrous appearance and the same minute structure as the preceding.

But, some months after, there sprang up in the same locality a large, soft, bleeding protrusion, which after a fatal

amputation of the limb (November, 1850) was found to have a milk-white colour, except where tinged with effused blood, and the exposed portion of this protrusion was soft, pulpy, and grumous. Mr. Paget adds :* "One would certainly, judging by its general aspects, have called it a brain-like medullary cancer, and yet it had essentially the same microscopic character as the tumour I first examined from the same patient." Similar cases are cited by this authority as having come under the notice of himself and other pathologists. My own observations concur.

Here then are tumours—the fibrous, and recurring fibroid in its first stage, which possess the *same* physical characters, but conjoined with a *different* structure ; and conversely, recurring fibroid tumours in their first and second stages, presenting *dissimilar* physical characters associated with the *same* essential structure.

Yet the progress of tumours thus physically related is of widely different therapeutical importance. Indeed, they differ more in this respect than the varieties of cancer, which vary only in the degree of their local and constitutional influence. Recurring fibroid, unlike ordinary fibrous tumour, not only returns (as its name implies) after complete removal, and in this particular resembles cancer-growth, but it also recurs at shorter intervals after each operation, and successively attains more speedily a larger size, invading also and destroying surrounding tissues. The earliest and most exact diagnosis of these tumours is therefore a question of the highest therapeutical interest, but one which cannot be decided by physical characters alone. I am of course aware that their diagnosis may be guided, if not determined, by the fact of local recurrence —in the case of a second, third, or even fourth tumour reappearing in the same situation ; but the question is the *primary* diagnosis of recurring fibroid and ordinary fibrous growths—tumours which closely resemble each other, except in their minute structure and pathological tendencies. Can we, by an external examination of their colour, consistence, size, shape, and situation, before an

---

* Surgical Pathology.

operation, foretell their relative liability to return if completely extirpated? Certainly not; the history of fibrous tumours, and their recurring fibroid as well as fibro-nucleated varieties, justifies this conclusion.

Physical diagnosis fails in these instances, and may fail in others, again and again, to determine what are the co-existing changes of structure. The *inconstancy* of physical characters in connexion therewith, falsifies, or at least impairs, the diagnostic value of such characters, as signs.

This explains how it is that certain well-known classifications of tumours, and corresponding diagnosis, have not stood the test of more extended experience.

We must ever cherish the name of Matthew Baillie; for he, more than any man of his generation, sought to connect Pathological Anatomy with Clinical Medicine; yet why should I hesitate to criticize the method of one who, if he were now alive, would, I am sure, soon discover and acknowledge its imperfection? Errors of the past are guides for the future; they are but ripples in the stream of human progress.

The special purpose of Baillie's great work is announced in his preface to the first edition.* It was "to explain more minutely than has hitherto been done the changes of *structure* arising from morbid actions in some of the most important parts of the human body." By more accurately describing structural diseases, Baillie hoped to be enabled "to distinguish between changes (of structure) which may have some considerable resemblance to each other, and which have been generally confounded." Thus would be laid the foundation of a more exact and rational diagnosis.

With this view, certain tumours of the liver, for example, are described and distinguished, according to their 'physical' appearances: *i. e.*, the common tubercle; the *large white* tubercle; the *soft brown*, and the scrofulous, tubercles; the *reddish* tumour; and lastly, the *soft*, and the *hard*, liver. The distinctive characters

---

* Morbid Anatomy, 1793.

of these tumours are then further specified in the following terms. The common tubercles occupy "generally the whole mass of the liver, are placed very near each other, and are of a rounded shape. They give an appearance everywhere of irregularity to its surface. When cut into, they are found to consist of a brownish or yellowish white solid matter. They are sometimes of a very small size, not larger than the heads of large pins, but more frequently they are as large as small hazel-nuts, and many of them are sometimes larger."

The large white tubercles are "hard white masses"—"often as large as a chestnut"—more abundant near the surface of the liver than in its interior, and isolated by considerable portions of healthy liver between each cluster. "They consist of a firm, opaque, white substance, and are generally somewhat depressed or hollow upon their outward surface." The whole liver is frequently much enlarged.

The soft brown tubercles are also principally situated at the surface of the liver, are "about the size of a walnut," and consist of a smooth, soft, brownish matter.

The scrofulous tubercles are like tubercles of the lungs, and "have the same size, the same structure, and the same feeling to the touch, but are a little browner in their colour."

The liver flaccid with reddish tumours. These are described as soft, of considerable size, and interspersed through the liver, which contains a thick sort of pus.

Lastly, the very soft liver is commonly of a leaden colour; whilst the liver very hard in its substance, offers no peculiar appearances on section.

Such was the foundation whereon Baillie endeavoured to raise a more exact diagnosis of these and other structural diseases. But Physical characters proved insufficient for this purpose; associating, as they did, diseases the most dissimilar in point of structure and pathological career, and dissociating those which were essentially the same and of similar import.

Abernethy perceived this fundamental error, and expressly

proposed to rectify it, by his description and diagnosis of tumours (1804).* Accordingly we read that "the *structure* of tumours is also a part of morbid anatomy which deserves to be examined; since (as it did not come within the scope of the undertaking) it has not been fully discussed by Dr. Baillie in his very valuable treatise on that subject."

In fulfilment of this proposal, Abernethy proceeds to classify tumours as new formations; first, sarcoma, so called from having a "*firm* and *fleshy* feel," including many varieties. This group, associated as the species are by their physical characters, includes growths of the most varied structure and pathological import. The wide-spreading and malignant constitutional cancer is coupled with the merely local and comparatively harmless fatty tumour.

This purely Physical method bore its fruit in due season. We may trace its evil results in the records of subsequent pathologists, no less than in the observations of Abernethy. Far be it from me to undervalue the merits of that distinguished surgeon, for we must all respect the memory of a man who had the courage to leave a beaten path, and to think for himself. The freshness of originality pervades the whole of Abernethy's writings. But the purely Physical method of Baillie and Abernethy gave a superficial aspect to Pathology, which long afterwards prevailed, and the vestiges of which can still be traced in works that I need not specify. Hence the descriptive terms—chalky, curdy, cheesy, &c., commonly used by some authors, as sufficiently distinctive of morbid products; characters that have no constant connexion with the same alterations of structure (and chemical composition), each to each, and therefore convey no information whatever respecting these more essential conditions of disease.

Nor is this inconstancy surprising; for, in respect of simple inorganic products, very different physical characters may be connected with the same substance. Carbonate of lime may exist either as chalk or marble; and other chemical compounds have

---

* Surgical Observations—Tumours.

physical properties so variable as to elude detection and identification thereby. Nor is this law true only of compound substances. It extends also to many chemical elements. Carbon, as the diamond, is very hard, transparent, colourless (mostly), and in the form of a cube or octahedron; while, as blacklead, it is soft, opaque, black, and generally shapeless. Who could have predicted, by their physical characters, that the diamond and blacklead were alike varieties of carbon? Sulphur may assume different characters, disguising its identity. So also may phosphorus.

If then, by physical characters, we cannot recognise chemical compounds, or even their elements when uncombined, how far less likely are we thus to identify conditions of structure superadded. Misguided by their physical characters, we should inevitably associate diseases, having the most dissimilar structural conditions; pathological course, tendency, and significance.

When, therefore, we read or hear, in Clinical Medicine or Surgery, of chalky, curdy, and cheesy substances, &c.; to what pathological conditions of structure and composition do such descriptive terms refer? Is the cheesy substance a tuberculous deposit, a variety of fatty growth, or the result of degeneration? Or, is it *cheese* after all? By Physical characters alone we cannot tell.

CHAPTER VI.

THE DIAGNOSTIC GUIDANCE OF CLINICAL PATHOLOGICAL ANATOMY (CONTINUED).

The application of Pathological Anatomy during life (continued) by Structural characters—and the *superior* value of their guidance to supply the earliest and most exact Diagnosis.

Changes of minute Structure can be detected at the earliest period, during life, by puncturing Growths and Deposits (situated near the surface of

the body), and by examining with the microscope these or other Morbid Products, found in Discharges and Secretions coming from internal parts and organs.

Moreover, the Structural alterations thus detected are more constant in any given case, than alterations of Physical properties, and are therefore more exact signs of the morbid condition; and being also discoverable at the earliest period, before the supervention of complications, are thereby best adapted to supply the chief method of Diagnosis whenever such minute Structural alterations are available.

The value of this general Principle illustrated by the earliest and most exact Diagnosis of Morbid Products of Nutrition, as Growths and their respective varieties: *e.g.* Fibrous Tumours—including the *fibro-nucleated* and *recurring-fibroid varieties:* Cartilaginous Tumours—including the *myeloid* and *recurring varieties;* Cancer—*encephaloid, scirrhus,* and *colloid.*

The Law of Structural Retrogression applied.

The Principle advanced in this chapter was indirectly demonstrated in the latter part of the last by the insufficiency of physical signs, "as *contrasted* with the (superior) evidence supplied by structural characters."* By the diagnosis of 'fibrous tumour,' and its *recurring* varieties, both these Principles are illustrated concurrently. For the pathologico-anatomical dissimilarity of the fibrous tumour and its recurring *fibro-nucleated* variety—apparently identical by their physical characters—can alone be determined by virtue of their structural characters as declared by microscopic examination; thus illustrating the superior value of this method of diagnosis. In like manner, as regards the fibrous tumour, and its *recurring fibroid* variety, 1st stage. Lastly, the pathologico-anatomical similarity of recurring fibroid tumours, in an *early* stage, and in *advanced* stages—apparently non-identical growths, by their physical characters—can alone be determined by virtue of their structural characters; thus again illustrating the superior value of this method of diagnosis.

But the diagnostic value of structural characters, as well as that of physical characters, is obviously regulated by the constancy and exclusiveness of their mutual connexion, in respect of each particular species of disease. This implies the *identity,* and *identi-*

---

* P. 237.

fication, of the particular conditions in question; that of structural characters, for example. The *same* structural characters must invariably and exclusively coexist with the same physical characters (and chemical composition) to constitute the same disease. If therefore, in the clinical study of disease, either order of characters be *variable*, we should hesitate to accept such *indefinite* characters in diagnostic evidence of the disease in question.

Now, on behalf of structural characters, *their* variability may be, and probably often is, *more apparent* than *real*. This is illustrated by the diagnosis of certain morbid products of quasi-variable structure, and whose types are well defined; such is 'cancer-growth,' and its varieties.

All cancers present the same cell. This, at first colourless, pellucid, and consisting of a delicate envelope, contains a large clear nucleus or two, sometimes more, never less, within each of which is imbedded one or two nucleoli, also large and clear. Such is the *cancer*-cell (Bennett). It assumes various shapes, being either round or more commonly caudate, fusiform, &c., from outgrowths in one or more directions. These cells are found deposited in a filamentous stroma or meshwork, having a variable locular arrangement and closeness of texture. This intercellular substance is probably, for the most part, nothing more than the fibrous tissue of the part in which the cells are deposited. Another intercellular substance, gelatinous, translucent, and amber-coloured, may be present in more or less abundance, and would appear to be peculiar to cancer.

As the proportion of cells, or of either intercellular matter prevails, so do we recognise encephaloid,—abounding with cells, and therefore soft, opaque, and of a dead white colour, or red from hemorrhage; scirrhus—fibrous, and therefore hard, semi-transparent, and of a bluish white or fawn colour; and colloid—gelatinous, transparent, and yellowish green.

These leading varieties of cancer are further allied by possessing a similar chemical basis: viz., chiefly albumen associated with fibrin, gelatine, osmazone, fat, salts—*e. g.*, phosphates and carbo-

nates of lime, with carbonates of soda and magnesia, oxide of iron, and water.

Now, do the *same* conditions of structure invariably and exclusively coexist with these physical characters, and this less certain chemical composition, of cancer? This question bears directly on its exact diagnosis and therapeutical treatment. For the evil tendencies of scirrhus, encephaloid, and colloid are of unequal importance. The first two are more prone to recurrence after operations for their removal, and contaminate the system more fatally; the latter is less liable to reappear, and is moreover comparatively localized. Can we then exactly determine our diagnosis of each variety, and this at the earliest period, by any corresponding structural characters which may be disclosed, either by examining the discharges (from cancer) with the microscope, during life, or by a more complete examination of the growth when removed by operation? I think not. No sufficiently exact 'structural' differences have as yet been detected, by which to identify these varieties of cancer-growth, and we are therefore compelled at present to rely solely on their 'physical' characters to solve the question of their (differential) diagnosis.

This conclusion refers only to the *comparative* value of structural characters; for many other circumstances associated with cancer—its seat, course, and duration, its influence on surrounding parts, the general symptom of cachexia, and the peculiarities of age, sex, previous diseases of the individual, and hereditary taint—combine to regulate our diagnosis.

But respecting the value of anatomical characters *alone*—Vogel truly remarks, that the anatomical and histological relations of carcinomatous tumours exhibit the greatest variety; indeed, even in the same tumour, different parts often present very different characters, and which also *vary with their stage of development.* The italics are my own.

Pressing the question further,—is the 'cancer-growth,' in any form, a special kind of growth, structurally distinct from all others?

Opinions are pretty evenly divided respecting any supposed

specific characters of the cancer-cell. Lebert and Robin decide affirmatively; while Vogel and Virchow deny its individuality. Forster even maintains that similar cells, having the same size and shape, are met with as often in healthy as in diseased tissues; and, although Bennett accords that peculiarity of structure to the cancer-cell which I have mentioned, yet he is disposed to differ from Lebert, and rather to agree with Müller in believing that *no single element* is diagnostic. Bennett maintains that cancer is indeed a special kind of growth, but to be recognised only by a *peculiar association* of structural elements not themselves specific. Paget would seem disposed to qualify even this restricted definition. He asks, are there any cancers not formed of cancer-cells? The answer according to this authority is affirmative; for there are *rare* tumours which present the whole clinical history of cancers, and which should therefore be called by the same name, though they have not these (so-called) peculiar cancer-structures, or have them in very subordinate quantity. Mr. Paget does not here refer to cancers of which all the structures are imperfect, or degenerate, or diseased; but to such as the fibrous cancers, the osteoid, and certain varieties of the medullary. They all deviate from the assumed specific cancer-structures; and two of them, the fibrous and the osteoid, approximate to the characters of natural tissues.*

Guided by these observations respecting the structural individuality of cancer, as a distinct species of growth, we must conclude:—1stly. That, occasionally, the structural elements of cancer are absent in tumours, otherwise cancerous; and therefore, that such elements are inconstant. 2ndly. That these elements may be found (and often, according to Forster,) in healthy as in diseased tissues, and are, therefore, not peculiar to cancer.

These two propositions, taken together, *apparently* negative the value of the *cancer*-cell, as the most exact method of diagnosis.

If, then, we can rely with confidence only on structural cha-

---

* Lectures on Surgical Pathology, vol. ii., p. 565.

racters for the exact diagnosis of certain growths—*e. g.*, fibrous tumours and their recurring fibroid and fibro-nucleated varieties; while in respect to other species of growth—*e. g.*, cancer—we cannot depend on the structural individuality of the cancer-cell; it becomes a question of the deepest practical interest, whether its variable conditions can be explained. Are they so many *gradations* of the *same* structural development? If so, its variability may thus, perhaps, be reconciled, and the diagnostic value of structural characters fully confirmed.

It should be remembered that tumours which run the course of cancer, but which have not its minute structure, are quite *exceptional;* and therefore, the question of diagnosis by virtue of structural characters is reduced to this:—Why is the structural element of cancer not peculiar to this growth, but so nearly resembles that of certain other growths, and even healthy tissues? and can the structural conditions of the varieties of cancer be reconciled and referred to a common type?

Now, we have already seen that the structural peculiarities of fibro-nucleated and recurring fibroid tumours merely represent rudimentary (or developmental) stages of the ordinary 'fibrous tumour;' and further, that there is a certain resemblance between the permanent structure of fibrous tumour and the transitory conditions of development of its analogous healthy (fibrous) tissue.

Passing on to 'cancer-growth,' its essential structure represents *conditions of healthy structural elements.* Neither the fibrous stroma nor the cells of cancer are specific. The former, with perhaps rare exceptions, is the natural fibrous tissue of the part wherein the cells are infiltrated; and they resemble the cells of cartilage, and certain epidermic cells. For this reason, apparently, Bennett limits his definition of the *cancer*-cell to localities which are unconnected with a mucous or an epidermic surface, and nowhere surrounded by a hyaline or fibro-hyaline substance—the intercellular basis of cartilage.

The qualifying terms of this definition at once suggest a true and sufficient explanation of the anomalous fact that the cancer-

cell is not peculiar to cancer-growth; so that by limiting the definition of cancer (in virtue of its structural elements) to those parts of the body where the otherwise peculiar cells of cancer are normally found, in connexion with healthy tissues, this structural element becomes available for the diagnosis of cancer.

Excluding those exceptional forms of growth which simulate cancer in their clinical history, but which do not present the cells of cancer, and also excluding those healthy tissues with which similar cells are found, then this structural element, when present, is the most unequivocal sign of the presence of cancer, and the value of structural characters is fully confirmed, as the earliest and most exact method of diagnosis. In like manner, the different varieties of cancer, so far as they can be distinguished by structural characters, chiefly correspond to rudimentary stages of analogous cartilage-cells.

The cells of encephaloid—very numerous, and diffused amid an abundant soft intercellular substance—grow most rapidly. They, therefore, often reach their highest stage of development—that of cells enclosing others; and being found, moreover, in every rudimentary stage, and perhaps also throwing out their most fantastic forms, present the most varied appearance. The cells of scirrhus—less numerous and more closely infiltrated within the meshes of a dense fibrous tissue, or other tissue of the part affected—grow less rapidly and variably; while the cells of colloid, well nigh superseded by the abundant gelatinous matter in which they are suspended, have a variable shape and development.

Thus, these *apparent* differences of structure of the *cancer*-cell can readily be reconciled, and its deviations referred to a common type by the law of structural retrogression—*i. e.*, that morbid growths ('cancer,' for example,) represent only structural reversions to various rudimentary conditions of analogous healthy tissues, by the as variously arrested development of their structural elements. Guided by this law, the structural individuality of other growths, as the 'cartilaginous' and 'fibrous' tumours, is established, and the varieties of each of these types explained.

When, therefore, we fail to discover the uniform coexistence of the same structural conditions with physical characters (and chemical composition), we may perhaps reconcile this variability by a due knowledge of healthy developmental histology, and extend thereby the value of anatomical characters on behalf of the most exact and earliest reliable diagnosis of Morbid Growths: for their varieties are deviations only from so many typical conditions, they are but *phases* in the existence of certain known structural elements; the history of their formation, development, and maturity, their diseases, degeneration, and death.

CHAPTER VII.

THE DIAGNOSTIC GUIDANCE OF CLINICAL PATHOLOGICAL ANATOMY (CONCLUDED).

The application of Pathological Anatomy during life (concluded) by Chemical conditions.

The *potential* value of Chemical Pathology, to supply the earliest and most exact Diagnosis.

Alterations of Chemical Composition are, indeed, the most essential condition of Disease, but the least readily detected and identified. This Principle illustrated by Deposits of lymph,—forming false tissues, pus, tuberculous or scrofulous matter, and by the present state of Pathological Chemistry in general.

THE value of 'chemical' conditions, as touching the earliest and most exact method of Diagnosis, is to be estimated, like that of either physical or structural characters, by the constancy and exclusiveness with which certain known chemical conditions pertain to each particular disease. And this implies the *identity* and *identification* of these conditions.

This method of Diagnosis is found to be incompetent, not by any inherent variability of the chemical conditions of disease, but by reason of the inherent difficulty of detecting and identifying them. At the same time, their *essential* importance is easily demonstrated.

Deposits of lymph—forming false tissues, pus, and tuberculous

or scrofulous matter—are an appropriate illustration of this position. The different structural conditions of these, no less than of other morbid products, point to equally different chemical conditions of lymph as their *immediate* origin.

False membranes are distinguishable from deposits of miliary tubercle and pus, by well-defined 'structural' characters. The structural elements are themselves distinctive. Fibres—those of cellular tissue—abound chiefly in false membranes. Pus consists of corpuscles, and miliary tubercle is an intermediate admixture of both cells and fibres. Susceptibilities of similar gradations of organization are denominated by Dr. C. J. B. Williams, the euplastic, the aplastic, and the cacoplastic varieties of lymph. Each of them possesses equally distinctive 'physical' properties. Euplastic, or healthy, lymph—transparent, nearly colourless, and tenacious—forming the connective tissue of wounds which unite by adhesion. Aplastic lymph, or healthy pus, contrasts remarkably, being opaque, yellow, and diffluent. Cacoplastic lymph—the curdy, flaky, purulent discharge from indolent scrofulous ulcers—is opaquish and yellowish, but far less tenacious than lymph of the euplastic type. The contrast of these and other distinctive characters, physical and structural, is more clearly perceived when viewed in juxtaposition.

LYMPH.

	Fibrinous.	Intermediate Condition.	Corpuscular.
Physical properties.	Transparent.	Opaquish.	Opaque.
	Colourless.	Yellowish.	Yellow.
	Tenacious.	Tenacity less.	Diffluent.
Structure.	*Fibres.*	Cells.	*Cells.*
	Cells.	Fibres.	Granules.
	Blood-vessels.	Blood-vessels?	Blood-vessels, none.
	Ex. Connective Tissue.	Ex. Scrofulous Matter.	Ex. Pus.

The distinctive characters of lymph are certainly most obvious when it is effused from simple membranes, as the serous in pleuritic effusions; while the more complex structure of skin and

mucous membrane retard the separation of lymph, and modify its properties, by admixture with the peculiar secretions of these tissues. Hence the varied products of skin diseases, and the heterogeneous appearances of many discharges from the gastro-intestinal canal, and from the urinary and pulmonary passages. But the distinctions I have drawn are fully confirmed by the observations of Mr. Paget, who carefully examined the materials exuded in thirty cases of blister from cantharides.

What, then, is the 'chemical' constitution of these varieties of lymph-deposit, whose physico-structural characters are always different? Products having this well-defined individuality cannot be evolved from lymph having the same chemical composition. Such an hypothesis would be incompatible with both analogy and fact. By physiological analogy, and apart from pathological observation, we should infer that the varieties of any product indicate corresponding differences of chemical composition, which determine the particular condition of structure, and the physical properties, that each variety eventually assumes. This would appear to be the local and *immediate* origin of *all* diseases of nutrition. An intimate relation subsists between the chemical composition of blood and the various structural conditions of tissues and organs—*i. e.*, the products of *healthy* nutrition. By analogy, therefore, we are led to anticipate a similar pathological parallelism between the chemical condition of this fluid and the structural peculiarities of diseased normal tissues and new products. Paget refers to his own observations and those of Rokitansky, in corroboration of this position (suggested by analogy), and concludes, respecting the products of inflammation, that, "if it should appear that the same tissue, inflamed by the same stimulus, will in different persons yield different forms of lymph, we shall come near to certainty that the character of the blood is that which chiefly *determines* the character of an inflammation."*

But hitherto analysis has altogether failed to discover the

* Op. cit., vol. i., p. 327.

chemical individuality of lymph-deposits, not to mention the different blood-conditions they indicate. Their chemical composition remains, and is likely long to remain, an open question in pathology. We cannot *thus* identify these products, and refer them to known chemical conditions of the blood, from which they are derived. Herein consists their *essential* distinction; yet their diagnosis, the earliest and most exact, cannot be determined by chemical analysis.

Nearly the whole of Pathological Chemistry is in like manner defective. Until recently, this defect was overlooked. Organic analysis had been applied to Pathological Anatomy, chiefly with the view of discovering the "proximate constituents" of morbid organic compounds, rather than applied to the *actual products* of disease. The former method of investigation may be philosophical, but is not competent to meet the requirements of diagnosis. At the bedside various morbid products are presented to the surgeon for his discrimination; such are the materials of tumours, derived therefrom by ulceration or puncture, and discharges from the mucous passages. These discharges afford *direct* evidence of the condition of those internal parts from whence they come, just as the condition of the kidneys may be inferred from the chemical nature of the urine; and if chemical analysis is to determine our diagnosis, it can only be by supplying the most exact, as well as the earliest, knowledge respecting, at least, the qualitative composition of the various morbid products in question. Yet, what says clinical experience? That we know little or nothing, with certainty, of morbid products, viewed in this light.

While, therefore, the theory of "proximate constituents" may have done much for the philosophy of organic chemistry, it has yielded but little, through chemical pathology, towards the most exact method of diagnosis at the bedside; and the captivation of this theory has doubtless seduced many observers from the more practical inquiry respecting the composition of the *actual products* of disease. This misdirection of labour, and consequent defect in chemical pathology with reference to diagnosis, is more particu-

larly noticed by Becquerel and Rodier;* its reparation being commended to the attention of young and aspiring chemists. The work referred to is specially devoted to careful and repeated analyses of the *actual products* of disease, and these investigations are the most complete and recent expression of the present state of pathological chemistry. The following summary, however, shows that the results of chemical analysis are not yet sufficiently exact to fulfil the requirements of such diagnosis.

The chemical pathology of "diseased normal tissues" is regarded by Becquerel and Rodier as unfinished. Thus, of cartilage, they observe, we know of no researches hitherto published respecting this tissue, when diseased. Of the brain, spinal cord, and nerves, the teeth and hair, analyses have been made only in the healthy condition of these structures, or at least when their state has been but ill-defined. The chemical composition of the crystalline lens, in health and disease, is unknown.

Concerning "organic products of new formation," analyses are given of *certain* 'deposits,' and of *one* kind of 'growth.' Cancer-growth only is considered; and the analyses by M. Foy of encephaloid mixed with scirrhus, of pure encephaloid by M. Baudimont, and of scirrhus by Collard de Martigny, by Hecht and by L'Heritier, are passed over with this comment:—we shall merely say that these analyses having been made by different observers, and upon different principles, can scarcely be of any use, inasmuch as their results are, so to speak, analogous to those afforded by the analysis of many other organic changes. Moreover, many chemists have asserted that some of these analyses are similar to those of muscular tissue.

Of 'deposits,' the analyses of pus by Guiterbock, Valentin, Golding Bird, and Wood, yield discordant results. Our authors inquire, whence these discrepancies? How is it that no two analyses are the same? It would be (they add) very desirable that numerous analyses of each variety of pus should be made, for we

---

* Pathological Chemistry, 1853. Translated by S. T. Speer, M.D., 1857.

believe that many useful and valuable conclusions might then be obtained.

Lastly, the chemical pathology of the *blood* and *other fluids* is also pronounced by Becquerel and Rodier to be very imperfect. Indeed, they regard the analysis of even healthy blood as being at present an unfinished inquiry, especially respecting the nature of its extractive and fatty matters, the composition of its salts, organic and inorganic, the changes produced by respiration and nutrition, and "the presence of those immediate principles of which its more important constituents are composed." Certain chemical changes which the blood undergoes in a *few* diseases are investigated by Becquerel and Rodier, but they confess that "many morbid conditions of this vital fluid are as yet unknown, and that many acute and chronic diseases remain in which the changes taking place in the blood are still a mystery."

All these repeated failures of analysis to detect and identify the chemical conditions of disease lead to one and the same conclusion:—That, guided by this, the chemical method of diagnosis, we should inevitably associate diseases essentially different, and dissociate those which are possibly identical.

The *practical* incompetency of Chemical Analysis to supply the earliest and most exact Diagnosis; illustrated by the Diagnosis of many Blood-diseases, *e.g.*, scurvy, symmetrical diseases, and diseases having seats of election.

The diagnosis of scurvy is a singularly conclusive illustration of the *practical* incompetency of chemical analysis, at the present time, to detect and identify the essential condition of disease.

I need scarcely raise the question whether scurvy is essentially a disease of the blood, for all pathologists are agreed on this point. The imperfection of the blood, observes an authority (Dr. Budd), is the source of all the symptoms, and the cause of all the morbid anatomical changes which are observed in scurvy.

Now, the physical appearances of scorbutic blood, and its structural characters when examined with the microscope, are well known.

The blood has undergone remarkable and significant changes of colour; from the florid red of health, it has assumed a dark brown or green tint; it appears, also, only half coagulated, the supernatant serum being of a livid colour. Again, the red corpuscles are observed by Drs. Ritchie and Buchanan* to have become irregular in their outline, their disks more flattened, and more disposed to cohere together and aggregate into large insulated masses, than the corpuscles of healthy blood. These peculiar appearances are denied by other observers, who regard them either as inconstant or altogether absent. Dr. Garrod, for instance, affirms, that recent examinations have shown the blood not to be in a dissolved state, as was formerly supposed, but that the globules are normal in appearance, the clot firm and frequently buffed and cupped.†

The balance of evidence, however, preponderates in favour of the characteristic colour and fluidity of the blood, and the collapse of the red corpuscles.

I would therefore ask,—what changes of chemical composition predetermine and accompany these peculiar physical appearances, and broken-down condition of structure? That scurvy consists essentially in a *chemical* alteration of the blood, will be evident on reviewing its causes and the methods of treatment which have been proposed. All the known causes of scurvy are referrible to some defect of diet, and its successful treatment resides in the restoration of this deficiency. But opinions differ, or are even opposed, respecting its nature.

Dr. Budd arrived at the following conclusions:—1stly. Anti-scorbutic properties reside exclusively in substances of vegetable origin. 2ndly. That these properties are possessed in very different degrees by different families of plants; least so by the farinaceous, as wheat, oats, and barley; most so by the succulent, as the aurantiæ, comprising oranges and lemons; lastly, by potatoes (a solanaceous plant). 3rdly. The anti-scorbutic property is impaired by the action of strong heat, nevertheless

* Edinburgh Monthly Journal, July, 1847. † Ibid., January, 1848.

boiled potatoes are anti-scorbutic (Baly); impaired, also, by vinous fermentation, but improved, probably, by acetous fermentation.

Dr. Christison examined the diet of certain railway labourers who were admitted with scurvy into the Royal Infirmary of Edinburgh during the spring of 1847. The general conclusion of his inquiry was, that no doubt can exist of a tendency to scurvy being engendered by a diet too purely farinaceous or saccharo-farinaceous and fatty; that this tendency cannot be counteracted by even a superabundance of the vegetable nitrogenous principle, gluten; but that it may be effectually counteracted by milk, and probably, also, by other azotized articles of food from the animal world. This authority adverts to the influence of other forms of diet as productive of scurvy, and amongst them he includes the deprivation of succulent vegetables; but, for the most part, the conclusions of Dr. Christison are *opposed* to those of Dr. Budd. The former regards animal casein, or at least vegetable albumen, as essential to the anti-scorbutic diet, and believes that it is to the latter principle, and not, as some suppose, to small proportions of salts with vegetable acids, that the ordinary succulent vegetables owe their undoubted anti-scorbutic properties. Dr. Ritchie inspected the diet of those who were admitted with scurvy into the Glasgow Infirmary during the winter of 1846-7, and states,* that the general fact in regard to the food of all was, it failed in variety and in the quantity of its animal constituents; and that in all but a fraction of the cases in which they had been very deficient, the patients had been subject for months to a total deprivation of fresh succulent vegetables. Dr. Lonsdale, of the Cumberland Infirmary, enumerates among the results of his experience, during the year 1847, of scurvy in Cumberland and the southern parts of Scotland, that a deficiency of potatoes constituted the chief error of diet, and was the main cause of that epidemic; whilst the want of variety, and the deficient quantity of food, hastened the development of scurvy.

The researches of Dr. Garrod† appear to me more critical and

---

* Edinburgh Monthly Journal, July, 1847.

† Ibid. 1848.

exact, having been conducted with the special object in view of determining the precise chemical constituent absent or deficient in scorbutic blood and food, and present in the anti-scorbutic condition. Dr. Garrod's analyses tend to prove that potash is deficient in scorbutic blood, and that the quantity of that alkali thrown off by the kidneys is also less than usual; furthermore that all anti-scorbutic diet and medicine abound in potash, which, again, is proportionally deficient in scorbutic food. On the other hand, Becquerel and Rodier are inclined to attribute the symptoms of scurvy to the presence of an undue proportion of soda in the blood, more especially in the chronic form of the disease, and that the excess of this alkali is accompanied by a diminished proportion of fibrin. They thus explain the efficacy of a vegetable acid regimen, unlike the alkaline treatment recommended by Dr. Garrod.

From all this evidence, one conclusion only can be drawn; that although the physical appearances and structural characters of scorbutic blood are well known, its 'chemical composition' is still an open question. Under the conflicting chemical opinions to which I have adverted, a man presents himself with the symptoms of scurvy. For some time he has gradually lost strength and colour; he now looks sallow and dejected. By-and-bye, red or livid spots appear, principally on his legs, which, moreover, seem bruised, of a yellowish green colour, swollen, and hard. Our patient may cough up or vomit blood, lose it by the bowels, or occasionally pass it with his urine. His gums are turgid, spongy, and rotten; they bleed on the slightest pressure. These hemorrhagic tendencies are evidently due to some altered condition of the blood. In other words, scurvy is essentially a blood-disease. But how can we reconcile the discordant opinions respecting its pathology? The *chemical* change is the point at issue. In vain we examine the blood vomited or otherwise lost in this terrible malady. Is it reduced below, or raised above, its healthy point of alkalinity? Does the blood lack potash, or abound in soda? or do neither of these defects prevail? and is it deprived of its healthy proportion of azotized constituents?

Are there, in fact, two or more chemical conditions of the blood, and these of almost opposite nature, but associated with the same physico-structural characters, and ecchymoses, &c., during life? or do these characters and symptoms indicate one and the same chemical change, as yet undiscovered? In respect of treatment, curative and preventive, are we to administer potash, or acids, or milk, or potatoes, or what?

The truth is, analysis has hitherto failed to detect and identify the chemical constitution of the blood in scurvy. Otherwise, it would then be quite practicable to apply this method of Diagnosis so as to fulfil the earliest, no less than the most exact, detection of the essential condition of this disease.

This twofold position is further illustrated by the diagnosis of certain other blood-diseases, affecting the skin or other textures. I allude to diseases which have particular 'seats of election,' and 'symmetrical diseases.'

Scrofula is an example, and especially of the latter kind. As manifested by the skin, I once met with a remarkable instance of *symmetrically* distributed scrofulous disease on either side of the neck. My notes are descriptive of what I saw. "In the middle line, a vertical scar is seen, extending downwards to the sterno-clavicular articulation, where it terminates in a kind of root, and upwards as far as the os hyoides. From thence a branch scar on either side passes upwards and backwards to the angle of the jaw; and from this branch again, on either side, another seam extends upwards to the mastoid process, and downwards on the sterno-mastoid muscle. Either axilla is the seat of an horizontal seam, from which there flows a small quantity of scrofulous matter." Syphilitic deposit—secondary syphilis—is equally shared by the *textures* it affects; thus, the eruptions of lepra and psoriasis are disposed tolerably *symmetrically* on either half of the skin, as *the* texture elected.

Then, again, some diseases select chiefly certain *portions* only of this or that texture; psoriasis, for example, claiming the flexures of joints. Is there any disease limited to only *a* portion of any given texture?

'Symmetrical distribution' in corresponding portions of texture in either half of the body is the rule. And this law obtains, whether certain textures only, one only, or portions only of that one, be the 'seat of election.' This election, and this symmetrical distribution in portions of texture apparently identical throughout its whole extent, alike imply an intimate chemical relation between the *blood* and *tissues*. Treviranus indicated the Physiological aspect of this relation by observing that "each single part of the body, in respect of its nutrition, stands to the whole body in the relation of an excreted substance." In other words, each part of the body stands related to the blood in the capacity of an excreting organ; the osseous texture electing and withdrawing certain constituents therefrom, the skin electing and withdrawing certain others, and so forth; the textures severally re-adjusting, and collectively maintaining, the chemical constitution of the blood in a healthy state. A similar Pathological relation exists. When, therefore, any noxious material or poison is present in the circulation, it may be selected and withdrawn by its appropriate excreting tissues or tissue, and left untouched by the rest. If the *skin* and *mucous membrane* be the appropriate emunctories, then these textures excrete the *materies morbi*, and a disease of the skin and mucous membrane, conjointly, is produced—*e. g.*, most of the exanthematous eruptions. If the *skin* alone be the appropriate emunctory, then that texture alone excretes, and a disease of the skin only is produced—*e. g.*, lepra. If *certain portions* only of this texture be the appropriate emunctory, then those portions only excrete, and a more localized disease of the skin is produced—*e. g.*, psoriasis. Lastly, if, as the rule, the *counter portions* of skin be also the appropriate emunctory, then they also excrete, and the disease of the skin is symmetrical—*e. g.*, lepra and psoriasis. In like manner, other tissues eliminate blood-poisons.

Chemical analysis has hitherto failed entirely to demonstrate the presence of such poisons, albeit circulating in the blood of thousands of people, and their manifestations daily witnessed.

But the textures severally detect the presence of these poisonous matters in the blood, and they being the *essential* morbid conditions in a large class of diseases, we must acknowledge the practical incompetency of chemical analysis to fulfil the earliest and most exact diagnosis.

It is worthy of note that this marvellously delicate analytical power of the textures severally, respecting diseases of the blood—indicating, as it does, an intimate chemical relationship between the blood and the component textures of the body in health—signifies the discovery of a physiological relationship by pathological observation. Similar contributions from this source confirm the reasonable assurance that Pathology, advancing, will yet more and more illumine the darkest recesses of Physiology—a department of natural science inaccessible, it may be, by experiments on animals, and necessarily, for the most part, inferential, and therefore inconclusive, as regards the human species. In this dilemma, morbid conditions are equivalent to so many experiments which indicate the conditions of health. Pathological observation becomes Physiological discovery!

The *present* value of Chemical Analysis to supply the earliest and most exact Diagnosis, illustrated by the diagnosis of certain Blood-diseases,—*e.g.*, cholera, gout, and rheumatism.

Having shown how little chemical analysis has hitherto accomplished for Diagnosis, and least of all respecting blood-diseases, it is gratifying to gather some of the first-fruits of this method of detecting and distinguishing diseases.

The most exact and earliest reliable diagnosis of cholera, and certain forms of diarrhœa, can alone be determined by the results of chemical analysis. The composition of the blood, as *contrasted* with that of the rice-water evacuations in true cholera, is the *essential* distinction between this disease and advanced stages of serous, mucous, and bilious diarrhœa.

A man suffers for some days from copious bilious diarrhœa. At length, though perhaps gradually, his skin feels cold and clammy

(sometimes dry), loose also, and shrivelled; while its usual pink colour shades off to a dusky blue tint, particularly about thin prominent parts, as the fingers, ears, nose, lips, and around the eyes, which, moreover, appear suffused and sunken. So much for outward appearances. Meanwhile, the pulse becomes feeble, and at last scarcely perceptible; for, although the heart beats forcibly, yet the blood is now too viscid, from prolonged evacuations, to be adequately propelled. The respiration is therefore hurried and oppressed, while the cerebral functions are blunted and lethargic. These symptoms are alike the consequences of an enfeebled circulation—the symptoms of collapse. But they are also the symptoms of true cholera, and *thus* far it is impossible to distinguish *this* disease from *severe* diarrhœa.

With the general symptoms of ordinary severe diarrhœa, a *whitish* flocculent fluid, resembling rice-water, may be expelled from the bowels. Pint after pint, and quart after quart, is discharged at short intervals, and apparently quite involuntarily, yet suddenly, and often with great violence, as from a tap; at the same time a stream of white or greenish fluid is copiously spirted from the mouth, and thrown to some distance, without, however, any straining effort. How much must the volume of the blood be thereby reduced! No discharge of bile could be attended with such *utter* collapse. Hence the more enfeebled circulation, which the heart labours in vain to restore; hence the livid, cold, and shrunken surface, the oppressed breathing, and fatal lethargy of Asiatic cholera. The poor victim, around whom the shades of death are closing, is for a time restless and disturbed, but soon an apathetic indifference steals over him, from which he is roused only by cramps of the legs, thighs, and belly. With these, he cries out in a hollow and sepulchral voice for drink. A ferocious thirst instinctively prompts his constant entreaties for water, wherewith to supply the reduced volume of blood—apt proof of a natural restorative tendency. But water is not blood, only *one* of its components. The evil lies deeper. Analyse the rice-water evacuations, and we at once discover those materials which the blood has lost;

analyse the blood itself, and we further discover the absence of those constituents, and the presence of those secretions—bile and urine—which ought to have been discharged, but are now retained.

In other words, the composition of cholera evacuations (when contrasted with that of the blood in this disease) affords the *surest* evidence for the most exact diagnostic distinction of *collapse* from Asiatic cholera and that arising from bilious diarrhœa.

The history of symptoms conducts to this conclusion :—that the rice-water evacuation is the only symptom *constant* in, and *peculiar to*, the collapsed stage of Asiatic cholera. True it is, that a similar evacuation occurs occasionally in phthisis, and even in chronic dysentery; but these diseases differ from Asiatic cholera in other and more important particulars. The diagnosis is not, *then*, restricted to chemical evidence afforded by analysis of the evacuations. It is between choleraic and other allied forms of diarrhœa that the 'chemical' nature of the evacuations yields *conclusive* evidence of their identity or difference. I do not mean to assert that a chemical examination of the discharges is always necessary to determine whether the disease be cholera or not; for the rice-water *colour* is tolerably characteristic, and bare inspection, therefore, of the evacuations may suffice to identify the disease; but I do mean to affirm that, for the *most* exact and earliest *reliable* diagnosis, the only method of examination is by 'chemical analysis.'

What, then, is the *relative composition* of the rice-water fluid and of the blood in cholera? Elaborate analyses, by Dr. Dundas Thomson, prove—Firstly, that the said fluid, whether alvine or vomited, consists chiefly of water and common salt, with a small proportion of albumen. The composition of this fluid is, in fact, precisely similar to that secreted by serous membranes in hydrocele, hydrocephalus, and other forms of dropsy. Secondly, respecting the blood; its water is reduced from 7 to 13 per cent. The proportion of solids to water contrasts thus :—

Health	1 to 3·91
Cholera	1 to 2·62

This relative excess of solid matter is due, I presume, to the large amount of water withdrawn. Accordingly, while the total amount of salts present may rise as high as 12·56 in 1000 (Thomson) above the average of 7·50 in 1000, yet the soluble salts remaining in the blood are proportionately less than the insoluble. These results are fully confirmed by the more recent, though limited, observations of Becquerel and Rodier.

The balance of the blood's constituents is thus seen to be disturbed, and the results of chemical analysis employed after death fully corroborate the value of this method of diagnosis during life, and that it supplies the critical distinction between true cholera and allied forms of diarrhœa. This conclusion is confirmed by the comparative results of different modes of treatment. The combination of salts, first suggested by the late Dr. Stevens, was tried on a large scale by Dr. Marsden at the Royal Free Hospital, and with considerable success. Compared with other modes of treating the collapsed stage of cholera, it afforded the happiest results. Of eighty-one cases, only seven died, and seventy-four recovered! The combination of salts used was—common salt, two drachms; carbonate of soda, one scruple; chlorate of potash, seven grains. But this formula nearly represents the salts discharged from the blood, and discovered in the rice-water evacuations. Chloride of sodium is the prevailing ingredient removed from, and restored to, the blood.

Here, then, the results of therapeutic measures—themselves suggested by chemical analysis—confirm the value of this method of Diagnosis.

Gout and rheumatism bear similar testimony, in so far as the *essential* conditions of both these diseases can severally be detected and identified by chemical analysis of the blood and urine. But with reference to all morbid states of the blood, as manifested by deposits in the urine, microscopic examination is the more conclusive method of Diagnosis. Both together, illustrating the combined application of the Principles established in this and the previous chapter, will be fully discussed in *that* portion of

Etiology, and its bearing on the Prevention of disease, which is set forth in Chapter X.

In conclusion, respecting the general application of Chemical Diagnosis; can the apparent *variability* of chemical conditions be explained, so as to reconcile the discrepancies of chemical analysis, and thereby extend the value of this method of diagnosis? For example, Rokitansky* refers to the constant connexion of certain different conditions of fibrinous structure with Mulder's gradations of protein oxidation.

Now this connexion assumes the *individuality* of protein, regarded as the basis of a series of fibrin products; and moreover, affirms that its *successive stages* of oxidation constantly coexist with certain different physico-structural conditions of fibrin. I would name this and other such-like serial evolutions, the law of 'chemical generation;' a very important one if true, for it introduces the general application of organic radicals to Chemical Pathology. By this law we might trace the successive evolution or chemical generation of morbid products from certain, possibly only a few, proximate constituents. We might thus refer the apparent chemical variability of morbid products to their composition. This law would be analogous to that of 'structural retrogression;' and as I have thus endeavoured to reconcile the variability of anatomical characters, and to extend thereby their value in the diagnosis of structural disease, so also the law of chemical generation might similarly extend the value of Chemical Diagnosis.

The rise and fall of one organic radical and theory of chemical generation—as applied to Physiology—may be told in a few words. A distinguished chemist, Mulder, thought he had isolated a substance consisting of $C_{40}H_{31}N_5O_{12}$. He conceived that this compound, acting as an *element*, entered into combination with different proportions of sulphur and phosphorus, and thus gave rise to certain animal and vegetable substances—albumen,

* Pathological Anatomy, Syd. Soc. Trans., vol. i. p. 96.

fibrin, casein. He therefore named the new substance Protein, for it seemed to be the proximate element or basis of a whole series of compounds. This series was accordingly designated the protein, or albuminoid series of compounds, from albumen being regarded as their type. These compounds, moreover, directly form blood, and were therefore appropriately named sanguigenous; while the various organized tissues being produced from blood, the protein compounds were also named—plastic elements of nutrition. Here, then, was a most valuable discovery, no less than a clue to the whole chemical history of nutrition, and indirectly to that of certain morbid products. Unfortunately, however, subsequent observations proved (amongst other objections) that no such substance as Mulder's protein exists without sulphur, and that protein itself is only one of the first products in the decomposition of albumen, fibrin, or casein, when heated with potash or other strong caustic alkali. Accordingly the protein theory of chemical generation subsided.

Nor are the discordant results of chemical analysis, and therefore the *practical* incompetence of this method of Diagnosis, at all surprising. For when bodies enter into 'chemical' combination, their individual properties are lost, or at least become altogether *imperceptible,* and nothing then remains to indicate their presence in the resulting compound. When substances are again liberated by decomposition, they recover their peculiar properties, and again become perceptible to our senses. Meanwhile, in combination, substances are temporarily withdrawn from our sight and from the cognizance of our other senses. Sulphur and mercury, for instance, are very readily recognised, each by its own physical properties; they combine, and together form cinnabar, a substance possessing the properties of neither of its constituents. Who would even suspect the presence of either sulphur or mercury? Yet the resulting compound, when heated, yields up these elements again. But if the simple union of only *two* bodies temporarily obscures their individual presence, how far less likely are we to detect the numerous constituents of *mixed* organic products!

Observe the varieties of pus;—what is the essential composition of this fluid, and what are the different conditions of blood which its varieties indicate?

To meet the requirements of practical Medicine and Surgery, we must begin with morbid products as they are presented during life at the bedside. Yet herein lies our difficulty. The phenomena of Physical science are more open to observation than those of Chemical Pathology; for, the very term, *composition* or combination, implies the withdrawal of bodies beyond the reach of vision. An appeal to vital properties may occasionally be a test of composition, more obvious than the evidence afforded by chemical re-agents. For example, the vital contractility of a piece of muscle, when dipped in a solution of strychnine, responded to the presence of only $\frac{1}{3000}$ part of a grain of this substance (one of the last observations of Marshall Hall). Even vision, aided by the microscope, can detect substances, the presence of which chemical analysis fails to disclose; hence, many of the adulterations of food and medicines, brought to light through microscopic examination by Dr. Hassall.

Chemistry possesses not this resource, but in fact owes its existence to the impossibility of thus detecting the presence of substances in combination. It is, essentially, the science of the short-sighted, the art of the blind.

THE PRINCIPLES OF ETIOLOGY,

OR

OF THE *EARLIEST* AND *MOST EXACT* DETECTION AND DISCRIMINATION DURING LIFE OF THE CAUSES OF DISEASES AND INJURIES, INDIVIDUALLY, AND OF THE OPERATION OF SUCH CAUSES.

PRELUDE.

The causes of diseases and injuries are 'external,' or 'internal;' and either may be predisposing or immediate in their operation. Internal causes are 'local,' or 'constitutional,' morbid conditions.

The earliest and most exact Etiological knowledge respecting the *operation* of internal causes can be concluded only by special Clinical observation of their Functional manifestations; but such knowledge implies the previous detection and identification of these causes, and a prior Diagnosis therefore of similar quality. Hence the guidance of Pathological Anatomy to Etiology in respect of 'internal' causes.

THE word 'cause' implies a relation—the relation of antecedence and consequence. The antecedent is the cause, the consequent the effect. But the antecedent must be immediate, otherwise the relation of causation is not suggested. A fracture may follow a fall, but if this injury does not supervene immediately afterwards, say not until a few minutes or perhaps moments only have elapsed, then some other act of violence may have intervened, and the fall cannot be regarded as the cause of the fracture; thus clearly showing that a cause signifies an *immediate* antecedent. Then, again, it must be an *invariable* antecedent, or at least in proportion as it is so, by so much does it fulfil the idea of being a cause. A fracture is followed by inflammation, the more severe if it be compound; but if a compound fracture were followed by inflammation once only in, say, a

hundred cases, then this form of injury would not be regarded as a cause of inflammation. The relation would be one merely of casual coincidence.

A cause is, therefore, not only an immediate, but also an invariable antecedent of that which is its effect, and the relation of causation implies a strict fulfilment of these two conditions by any antecedent regarded as such. I need scarcely add, that in so far as it is the *only* antecedent having these two qualifications, by so much is it the *only* cause, *i. e. the* cause.

But the regular order of succession which causation implies is scarcely ever observed, excepting by the phenomena of Physics and Chemistry. A tree falls at once and invariably under blows from a hatchet. Gunpowder explodes immediately and invariably by a spark of fire, and the resulting chemical compounds are at once produced, and are invariably the same.

Less regular are the phenomena which characterize 'living' beings in their conditions of health and disease, respectively. The phenomena of Physiology and Pathology do not obey a regular order of succession—the same antecedents are not immediately and invariably followed by the *same* consequents—the relationship of cause and effect is broken. Why this should be will presently appear. An organism circumstanced as the human body is, receives the influence of many external agents—food, air, temperature, &c.; and these agents operate either to evoke and maintain life and health, or to induce disease and death.

Many 'external' agents, therefore, may shake the fortress of health. So numerous, indeed, and varied are the accidental causes of disease and injury by which the body is encompassed, that in one of Addison's admirable essays Death is figuratively represented as a person who metamorphoses himself like another Proteus into innumerable shapes and figures. To represent the fatality of fevers and agues, with many other distempers and accidents that destroy the life of man, Death is supposed to enter, "first of all, in a body of fire; a little after he appears like a man of snow, then rolls about the room like a cannon-ball, then lies on the table like a

gilded pill; after this he transforms himself of a sudden into a sword, then dwindles successively to a dagger, to a bodkin, to a crooked pin, to a needle, to a hair."

This diversity of character precludes the possibility of classifying external causes with much precision of distinction. In a general way, they may be considered either *mechanical* or *chemical* agents, or, lastly, *irritants*, which influence the vital properties and powers of the body. The most practical view of external causes is, to associate them with the *habits of society* and *class occupation*. This aspect of Etiology is certainly the one most important to the medical practitioner; and it is also, as I have elsewhere remarked,* "a language that everybody should learn, nor disdain to have its alphabet perpetually in their hands."

The habits of society and class occupation variously regulate and modify the hygienic requirements of regimen, ventilation, temperature and clothing, exercise, daily occupation, sleep, and the estate of marriage. But all these conditions are concerned in the preservation of health and the production of disease; the former result, therefore, indicates the public importance of social habits and class occupation in connexion therewith, and the latter issue shows the therapeutic interest of this aspect of Etiology.

External causes which have this habitual character may operate again and again, and yet predispose only to some disease or injury. On the other hand, any adverse external condition to which the body is suddenly subjected may as immediately produce its effect. External causes are therefore said to be either *immediate* or *predisposing* only, in relation to the effects of their operation.

Many causes of disease and injury operate within the body, and these are called 'internal' causes. Their operation is referrible to the Physiological law of balance between the functions of the various parts of the body, whereby all are mutually associated. Taking the blood's circulation as our starting-point, and as the

---

* The Irritable Bladder—its Causes and Curative Treatment, 1859.

link adjoining nutrition, then respiration, digestion, and excretion are accessory functions, and collectively have a direct relation to those of the nervous system. These, again, are in their turn dependent on the former for their support. No link in the chain of this circle can be broken by the Pathological excess, deficiency, or perversion of any one function, without disturbing, more or less, the harmony of all the rest.

Aberrations of functions are probably always connected with, and dependent on, structural alterations of their respective organs or tissues (while, conversely, diseases of structure surely produce alterations of function); for although the earliest alterations of structure are minute, and therefore likely to escape detection, or, relating only to the vascular condition of the organ or tissue affected, are almost or altogether effaced before post-mortem examination (in the event of a fatal termination), yet instances of functional disturbance *alone* are very doubtful, and, one by one, are being referred to altered structure. The pathological relation which associates functional diseases, so called, is thus transferred with all its significance to diseased alterations of structure. *They* mutually bear the relation of cause and effect, and pathologico-anatomical conditions are, indirectly, the (internal) causes of each other.

The operation of internal causes implies the failure of the vital powers—assimilation, excretion, sensibility, and contractility—by the due operation of which the body is preserved in a state of health. These attributes of living matter together represent the "vis medicatrix naturæ" of Cullen (probably also the "archæus" of Van Helmont, and the "anima" of Stahl). "We witness the operation of the vis medicatrix," observes Dr. Williams, "in the careful manner in which noxious products of the body and useless constituents of food are ejected from the system; in the flow of tears which washes a grain of dust from the eye; in the acts of sneezing and coughing, which remove irritating matters from the air-passages; and in the slower, more complicated, but not less apt, example of inflammation, effusion of lymph, and those suppurative

actions by which a thorn, or any other extraneous object, is removed from the flesh."*

This innate power of *resisting* the supervention of morbid conditions further and fully *explains* why the same cause does not invariably produce its reputed effect in different individuals, or in the same individual at different periods of life; this uncertainty being due to different degrees of resisting power.

Diseased conditions of structure—internal causes—are (like external causes) either *predisposing* only, or *immediate*, in their operation; more commonly they have the former character. Thus, disorganization of a joint by disease is only predisposing to dislocation; so, also, softening of bone as regards fracture.

While, however, either kind of cause, without or within the body, may be self-sufficient, both kinds may *co-operate* or operate in *succession*—the internal cause more commonly predisposing—the external, when sufficiently aided thereby, immediately inducing this or that disease. Thus, a fall which does not produce a hernia in one case, immediately does so in another, owing to weakness of the abdominal wall at the seat of rupture, being the structural predisposition to this lesion; and a posture which does not cause an apoplectic seizure in one case, immediately does so in another, owing to the blood-vessels of the brain having previously undergone calcareous degeneration.

Another aspect of internal causes is, they are either 'local' or 'constitutional.' The former term requires no explanation; the latter has not, as I conceive, hitherto been clearly and correctly defined.

John Hunter was the first pathologist who endeavoured to attach a precise meaning to the expression, constitutional disease. These are his words:—"Perhaps there is no term so vague or undetermined in the mind as the term, constitutional. Universal action of every kind may be called constitutional, even when arising from some local cause, which I have called one of the mixed; or

* Principles of Medicine, 1856, p. 7.

one capable of producing local effects, which we have also called one of the mixed. But a true constitutional disease is one arising, as it were, spontaneously in the constitution, partaking of the nature of the constitution itself."*

The last sentence of this definition is unintelligible—" a constitutional disease is one arising in the constitution ;" and scarcely intelligible is the opening expression, " universal action,"—nor does it become more so by the additional words, " of every kind." Abernethy's essay " On the Constitutional Origin and Treatment of Local Diseases," left the question unanswered. The expressions, 'constitutional' disease and 'constitutional cause' remain unexplained.

It may be alleged that common experience has already affixed something approaching to a definite and correct meaning to these expressions ; and, indeed, if common experience be *analysed,* it affords the elements necessary for this purpose. For example, itch is a local disease ; scrofula, however, is said to be a constitutional one. How so? It is essentially a disease of the blood, arising probably from mal-assimilation. Now, the blood is a 'tissue,' *distributed throughout* the body, and also one, in point of *functional* importance, *most essential to life.* These, then, are the two elements comprehended in, and therefore implied by, the expression, constitutional disease—as exemplified by scrofula. In like manner, diseases of the nervous system, no less than those of the blood, are constitutional, in so far as they are prevalent throughout the body, and functionally predominant. These two elements will be observed in the course of the next chapter to characterize and distinguish constitutional diseases.

Is either element more *characteristic* than the other? Functional predominance is *most* so. A tissue extensively distributed only, but not functionally controlling by its influence, cannot be the *seat* of constitutional disease. For example, cellular tissue exists throughout the body, yet its function is mechanical—to pack and

* Lectures on the Principles of Surgery, ch. xi., Local and Constitutional Diseases.

connect together all other parts; it does not control their formation and maintenance. The cellular tissue may, therefore, be that tissue whereby a constitutional disease is chiefly *manifested*, but it cannot be the seat of any such disease. Scrofulous matter is deposited and abscesses form in the cellular tissue, but scrofula itself is a disease of the blood.

On the other hand, a structure which is functionally predominant, but not extensively distributed, is an organ, essential indeed to life, and, as such, subject to disease of like functional import—for example, disease of the heart; but this is 'organic,' not 'constitutional,' disease.

The 'tissues' are the structures extensively distributed,* and *some* of these structural constituents of the body are also functionally predominant throughout. Consequently, the functionally predominant tissues are the seats, other tissues and parts being only subject to the manifestations, of constitutional disease.

This view of a large class of internal causes represents a general pathological relation of all the tissues. That some of these structural constituents functionally predominate in the compound functions of digestion, circulation, respiration, secretion (including excretion), and those of the nervous system. Such tissues may severally be the seat of disease—constitutional. All other tissues functionally subordinate, although generally distributed, more or less, throughout the body, are only secondarily involved, and manifest, through mal-nutrition, local nervous affections, or in both ways evince the character of the primary constitutional disease.

This is my definition of 'constitutional' diseases and their relation as 'constitutional causes.' Their *operation*, and that of local internal causes, can be discovered only by special Clinical observation of their Functional manifestations; but this implies the previous detection and identification of the pathologico-anatomical conditions they represent, and, therefore, prior Diagnosis.

---

* See definition, Elements of Anatomy. Quain and Sharpey, 1848, p. 19.

The earliest and most exact Etiological knowledge of the operation of internal causes further implies a prior diagnosis of similar quality. Hence the guidance of Pathological Anatomy to this standard of Etiology, in respect of internal causes.

Pathological Anatomy being the guide to the earliest and most exact detection and discrimination of internal causes during life, suggests the Principle of Prevention in the Practice of Medicine and Surgery.

CHAPTER VIII.

LOCAL MAY PROCEED FROM CONSTITUTIONAL MORBID CONDITIONS.

This general Principle of Etiology illustrated by the manifestations of Blood-diseases in their appropriate 'textures.'

1. In many textures.—Pervading Blood-diseases—Syphilis, Scrofula, Scurvy, Purpura. Growths—Malignant. Degenerations.
2. In special textures.

Skin, Mucous membranes, or both.

Fevers—Non-infectious: inflammatory, hectic, gangrenous typhoid.
Infectious: eruptive (exanthematous)—typhus, typhoid, relapsing, measles, scarlatina, small-pox, chicken-pox, erysipelas, plague.

Mucous membranes.

Diseases, infectious—Hooping-cough, influenza, cholera (Asiatic).

Cellular texture—Subcutaneous.

Cellulitis, Carbuncle, Boil.

Fibrous textures and Synovial membranes.

Rheumatism, Gout.

The Pathological Principle which guides the Preventive View of these Diseases.

Chemical Pathology applied to determine how far it is possible, in the present state of knowledge, to more early and exactly detect and discriminate these Blood-diseases, so as to prevent their local manifestations.

Pathological Anatomy of these local manifestations applied to more early and exactly detect and discriminate the Blood-diseases from which they respectively proceed, so as to prevent those of *infectious* origin being propagated.

Constitutional is not necessarily blood-disease; but all diseases of the blood are eminently constitutional, and best illus-

trate the operation of constitutional diseases as internal causes of other morbid conditions. These secondary conditions are always local, although all local diseases are not manifestations of constitutional disease. Hence the Etiological Principle advanced in this chapter. What, then, are the local manifestations of 'blood-disease'? Certain generic characters denote blood-origin, whatever may be the particular species of blood-disease.

Firstly. Blood-diseases manifest themselves locally by mal-nutrition, and in many textures simultaneously, *e. g.* syphilis.

Secondly. Blood-diseases are migratory or metastatic; they manifest themselves locally in many textures, consecutively—shifting from texture to texture: *e. g.* syphilis, scrofula.

Thirdly. Blood-diseases are symmetrical, not unfrequently, in their local manifestations;—exactly corresponding portions of the same texture, skin for example, may present the same form of eruption, disposed in the same pattern, and to the same extent, on either side of the body, *e. g.* psoriasis.

Fourthly. Blood-diseases are serpiginous;—their local manifestations are extended continuously by a creeping movement, as it were, the course of such extension being apparently guided by the texture itself: *e. g.* erysipelas.

Besides these manifestations of blood-disease by various forms of mal-nutrition, certain antecedent circumstances respecting the production and propagation of the disease are equally conclusive evidence of blood-origin.

Firstly. All diseases caught by infection, *i. e.* inhalation, are blood-diseases: *e. g.* exanthematous or eruptive fevers.

Secondly. All diseases produced by inoculation with the blood, or lymph effused from the blood, of one of the human species, or that of an animal, are blood-diseases: *e. g.* small-pox and cow-pox.

Lastly. Detection of poison in the blood, by examination—chemical, microscopical, or both—would be the most direct evidence of blood-disease; but this has scarcely yet been realized.

Every species of blood-disease does not exhibit *all* these characters, or each in the *same* degree. The local manifestations may

not be pervading *and* migratory, *and* symmetrical, *and* serpiginous, *and* infectious, *and* inoculable; or, if all these characters be present, they may not be *uniformly* so. Some may be absent in this case, or predominate in that; but taken *collectively*, these characters are constant, and peculiar to, diseases of blood-origin. Accordingly such characters are distinctive, and conclusive evidence, short of actual demonstration, of the presence and operation of some morbid condition of the 'blood.'

On the other hand, the two characters of symmetry and serpiginous course indicate some determining power in the 'textures' themselves, respecting the texture or portion of texture, as the particular locality in which a particular blood-disease shall manifest itself. For, if from the same mass of blood a given form of mal-nutrition is presented by *one* particular texture, or by *symmetrical portions only* of that texture; or, if not remaining thus localized, this mal-nutrition extends *continuously* in the original texture only, and not beyond to any adjoining texture; then, in either case, these phenomena indicate some elective power possessed and exercised by the texture itself, whereby it withdraws the blood-poison, and separates the *materies morbi* from the general mass of circulating blood. Moreover, this poison is of various *kinds;* for if a particular texture selects that which all others refuse, then that something must differ in its nature, according to the texture which selects it; and blood-poisons must be as numerous as there are electing textures—each to each—as numerous as there are diseases having "seats of election."

Such are the distinctive characters of 'blood-diseases,' and such the relationship they indicate between the blood and the various component textures of the body. How significant, then, does the study of Histology become, when viewed in this light; how important in its bearing on the practice of Medicine is that "General Anatomy," which Bichat, of immortal memory, originated! Textural Anatomy becomes a shaded map or chart, on which all diseases of blood-origin are registered. I have, therefore, ventured to classify these diseases, according to the *various textures*

in which their local manifestations are declared by the distinctive characters I have already enumerated.

Of diseases thus defined, the chief will now be reviewed agreeably to the foregoing preliminary observations. Perhaps no single local manifestation will be found characteristic, but a concurrence—cotemporaneously or consecutively, of such symptoms.

Secondary or Constitutional Syphilis.—Every out-patients' room, at a large hospital, I should say, and certainly that of the Royal Free Hospital, affords abundant opportunities for comparing secondary syphilis, in all its various forms, with diseases arising from other causes; and in my experience, while their resemblance is often remarkable, their distinctive appearances are rather in degree than kind.

Take firstly—'skin-diseases.' Similar eruptions—exanthematous, papular, squamous (vesicular), pustular and tubercular—arise alike, as manifestations of constitutional syphilis, and under other circumstances. Much stress has been laid on the *copper colour* of syphilitic eruptions, but this is not peculiar to syphilis.

In describing the copper colour, and its various shades, Erasmus Wilson* judiciously corrects the prevalent error of relying thereon as evidence of secondary eruptions; that these colours are by no means *pathognomonic;* that they are commonly met with in chronic eruptions of other kinds,—for example, in acne; and that he has often seen non-syphilitic eruptions possessing more of the dull and muddy hue which is generally supposed to be characteristic of syphilis, than syphilitic eruptions themselves; and that when present, this copper, or reddish brown colour, is not seen in undoubted syphilitic skin-diseases until their decline.

I do not attach significant importance to the *circular-shaped* ulcers, of darkish colour, which syphilitic eruptions present in their ulcerative stage.

Among the earlier and less equivocal secondary symptoms, is ulceration of the tonsils; the ulcer formed is excavated, with a

---

* Syphilis and Syphilitic Eruptions, 1852, pp. 60–64.

sharp and prominent, not to say everted, margin. The bed of this ulcer is sloughy, the surrounding mucous membrane dusky red. But even these appearances are not characteristic of syphilis, and original observers, such as Rose* and Carmichael† concur in mistrusting 'the ulcerated sore throat.' Certainly, the excavated ulcer of Hunter is not consequent on the indurated chancre only; and we must acknowledge, with Carmichael, that affections of the throat are too indistinct to afford any certain diagnosis.

Fissures of, and *milky stains* on, the tongue and inside the lips are more pathognomonic, but the former must be distinguished from those cracks which accompany irritable dyspepsia, and the latter—opaque white spots, resemble aphthous spots.

More doubtful are *mucous tubercles* situated on various parts of the buccal mucous membrane, as the tongue, lips, palate and tonsils. These tubercles are of a whitish colour, and may be seen also on the skin, in the form of pale, soft little cushions, bedewed with mucus; the skin surrounding each tubercle appearing puckered around its margin. Such tubercles—condylomata—are commonly found in secondary syphilis, grouped around the anus or on the scrotum; also, fretting along the margin of the external labia in the female; perhaps on the perineum, inner aspect of the thighs, and on the groins. Sometimes these tubercles occur in the axillæ (E. Wilson); and, in fact, wherever ordinary tubercles are warm and moist, they frequently become mucous tubercles, skin readily assuming the appearance of mucous membrane.

But mucous tubercles are not necessarily syphilitic. Wilson notices‡ the transition of roseola into lichen, of the roseola eruption into such tubercles, and that the conversion of lichen into them is by no means uncommon; yet roseola and lichen are not necessarily syphilitic eruptions.

'Ulceration of the nasal mucous membrane, that of the hard and soft palate, of the pharynx opposite the mouth, and of the larynx,' may ensue in secondary syphilis. These ulcerations are

---

* Med.-Chir. Trans., vol. viii., p. 421.

† Essay on Venereal Diseases, 1825, p. 64.

‡ Op. cit.

frequently accompanied with caries of the nasal bones, of the hard palate, even the vertebræ behind the pharynx, and necrosis of the laryngeal cartilages. The breath and discharges are singularly fetid. Is any such ulceration peculiar to secondary syphilis? Colles* acknowledges his inability to determine whether an ulcer in the nose be venereal or not. The appearances of scrofulous ozæna closely resemble those of venereal ozæna. Colles describes an ulcerated opening situated on the septum nasi, about a quarter of an inch from its anterior extremity, this ulcer being uniformly circular, and as large as the surface of a split pea; but adds, that a similar aperture may be found in persons who certainly never had any venereal affection, and that it may remain for years, at least for eight or ten years, in cases under observation. Extensive ulceration of the pharynx, as well as ulceration of the nasal mucous membrane and caries of the nasal bones, were noticed by Carmichael to be frequently associated with the primary phagedænic ulcer, but that similar ulceration of the pharynx arises in constitutional conditions assuredly not venereal. Chronic laryngitis, and ulceration of the rima glottidis, denoted by a broken voice, impulsive cough and foul expectoration, may be a manifestation of advanced and grave secondary syphilis. Portions of the laryngeal cartilages, *e. g.* the cornua of the thyroid cartilage, in an ossified state, are occasionally coughed up. Sir A. Cooper mentions these extreme cases, in his Lectures; but syphilitic laryngitis presents nothing peculiar in its characters, from first to last, whereby it can be distinguished from chronic laryngitis ensuing under other circumstances. The symptoms just mentioned might follow laryngitis, from a common cold.

'Syphilitic iritis' was overlooked by Hunter, in his observation of the course of the venereal disease; but although undoubtedly a frequent form of secondary syphilis, it cannot be distinguished from arthritic iritis; and resembles scrofulous iritis, in so far as regards the appearance of the eye itself.

---

* Prac. Obs. on the Venereal Disease, 1837, pp. 305-316.

An elaborate work,* which still retains its rank, states that in syphilitic iritis the eye presents tubercular depositions of lymph (on the iris), a reddish brown discoloration of the iris on its inner circle, and an angular disfiguration of the pupil, which is also occasionally displaced towards the root of the nose; and that these appearances, coupled with nocturnal exacerbations of pain, experienced in a much slighter degree, or not at all, during the day, are together sufficient to complete our diagnosis, corroborated as it is by the previous occurrence of syphilis, and in most instances the concomitant existence of other syphilitic symptoms. I shall presently notice the diagnostic value of contemporaneous and consecutive symptoms, respecting secondary syphilis; but how far trustworthy are the objective symptoms afforded by those appearances which the eye itself presents? The authority I have quoted himself retracts in part the diagnosis he has laid down; for, says he, "although the effusion of reddish, brownish, or brownish yellow lymph on the iris in the adult, clearly shows the case to be venereal, I have seen analogous appearances in several instances, both of young children and infants, in whom no suspicion of syphilis could be entertained." The symptom in question is not peculiar to syphilitic iritis. Nor is 'displacement of the pupil upwards and inwards' a characteristic appearance. It has been seen, especially by Mackenzie,† in chronic rheumatic arthritis; and still more frequently in scrofulous sclerotitis, without iritis. Moreover, it is present only occasionally in syphilitic iritis. This symptom, therefore, is inconstant as well as equivocal. Mackenzie mistrusts all the special symptoms accorded to syphilitic iritis, excepting the tawny or rusty colour of the iris near its pupillary edge, a condition present in most syphilitic cases, and almost exclusively in them alone.

'Chronic enlargement of the testicle' occurs late, if at all, in the course of syphilis, and cannot be distinguished from scrofulous enlargement of this organ. The physical characters

* Diseases of the Eye. W. Lawrence, 2nd Edit., 1841, p. 428.

† Diseases of the Eye, 4th Edit., 1854, p. 543.

which the testis assumes in these diseased conditions are very similar. In both cases, the enlargement distinctly commences in the epididymis—generally so in syphilitic, always so in scrofulous disease. In both cases, this swelling subsequently engages portions of the testis itself, the intervening portions remaining free and healthy; so that sometimes, by careful manipulation, nodules can be felt in the substance of the organ, through the tunica albuginea. At length the whole testicle becomes considerably enlarged, and feels hard and heavy. Then the scrotum may become inflamed and adherent, eventually undergoing ulceration, accompanied with protrusion of the testicle. In one such case, at the hospital, the whole scrotum was so much thickened and discoloured that I removed the testicle together with the portion of skin chiefly involved, rather than endeavour to save the organ by partial excision. On section, the appearances were those of a scrofulous testis. The epididymis was filled with a yellow friable matter, which, under the microscope, was seen to consist of imperfect broken cells and granules, while nodules of this substance were deposited here and there throughout the testicle, itself otherwise healthy, the reddish grey colour of its tubuli seminiferi contrasting with the yellow nodules. The man bore the mark as of a chancre at the corona glandis, which he says occurred about two years ago, and that the testicle began to enlarge nine months prior to the operation. Judging merely from the condition of the organ itself, in this case, it would have been almost impossible to have pronounced its enlargement syphilitic; and, indeed, the most accurate diagnosticians have acknowledged the resemblance of scrofulous and syphilitic disease of the testicle. Under whatever circumstances chronic enlargement of the testis takes place, the symptoms are precisely the same, says Sir B. Brodie.* Dupuytren, also, was led to this conclusion by his observations.†

'Venereal diseases of the bones, periosteum, fasciæ, and liga-

* Lond. Med. Gazette, vol. xiii., p. 221.

† Clin. Chi., t. i., p. 100.

ments,' are possible manifestations of constitutional syphilis, advancing from the surface to deeper textures within the body; but this is a neutral ground, shared by the mercurial poison, prolonged mercurialization, by scrofula also, and by rheumatism. The question of venereal origin is open, therefore, in every case, to investigation, probably afterwards to doubt.

A true syphilitic *node*, for example, is usually considered to signify merely chronic enlargement of the bone itself. A hard swelling forms, without any redness of the skin in the first instance, nor subsequently for some time; eventually only it becomes red and acutely painful. Such nodes occur mostly in certain situations. The syphilitic virus appears to select certain bones or portions of bones for the production of nodes; they are mostly subcutaneous, as the inner aspect of the shaft of the tibia, the subcutaneous portion of the ulna, the sternum, clavicle, and cranium. These portions of bone more especially form nodes, which become inflamed. Nodes arising from periostitis are softer, and evidently inflamed from their commencement. They suddenly arise, and as suddenly subside.

These distinctions are true, and yet the hard chronic node is no criterion of secondary syphilis. This kind of node seldom, if ever, appears, excepting when mercury has been used, and coming, as it does, late in the career of syphilis, has perhaps been preceded by more than one salivation. Issuing from the mixture of mercury and syphilis, one cannot say how far a chronic node is due to one or the other. It is no sure indication of constitutional syphilis. Carmichael's experience led him to regard this symptom as "equivocal and uncertain." Colles notices "a general nodose affection of the bones," which is liable to be confounded with so-called syphilitic nodes, and he draws some distinctions.*

Caries and *necrosis* are no less doubtful evidence of constitutional syphilis. Spongy softening of the bones, denoting caries,

---

* Op. cit., p. 185.

may happen in an advanced stage of secondary syphilis, and has its chosen seats, these being chiefly those where nodes are prone to form—on the tibia, ulna, clavicle, sternum, and above all, the nasal bones and cranium. But whatever bone or bones undergo carious softening, there is nothing characteristic of syphilis. Mercury as well as syphilis may be at work; and indeed we rarely, if ever, find caries in syphilitic cases, excepting where mercury has been freely used. None of the cases of syphilis which came under the observation of Guthrie,* in the York Hospital, were accompanied with caries; and such was also the experience of Rose, in his series of cases,† upwards of a hundred and twenty in number, and where he was able to ascertain that the patients remained free from syphilis for many months afterwards, or if secondary symptoms returned, caries was not one of them. Necrosis, in constitutional syphilis, is equally the offspring of mercury.

Lastly, the wan, yellowish hue that overshadows the skin in an advanced stage of this disease, and specified as 'the syphilitic cachexia,' bespeaks rather a mercurial deterioration of the blood.

Those who desire further information on the diseases which *simulate* syphilis may consult the subjoined works with advantage.‡

In conclusion, the diagnosis of constitutional syphilis by secondary symptoms is so far determined solely by the calculation of *probabilities;* and this is the basis of diagnosis in respect of *all* diseases, excepting the few that are absolutely determined by pathognomonic signs. The diagnostic value of any one 'symptom' is represented by the constancy of its presence and association with the same disease, and by the early period of its occurrence.

---

* Med.-Chir. Trans., vol. viii., p. 560. † Ibid., p. 422.

‡ A Treatise on the Venereal Disease. J. Hunter. Ed. by E. Home, 1810, p. 404 *et seq.*—Surg. Obs. Abernethy, 1804, p. 108 *et seq.*—On the Venereal Diseases which have been confounded with Syphilis. R. Carmichael, 1814.—De Morbis syphiloideis vel pseudo-syphiliticus. T. H. Burder, 1815.—On some Diseases reputed Venereal. R. Hamilton, 1820.—De Morbo pseudo-syphilitico sive Dithmarsico Pomeraniæ. J. A. Ran, 1824.—De diagnosi et cura ulcerum quæ dicuntur venereorum. J. Trier, 1829.—Ueber Syphilis und Syphiloid. R. Tiling, 1833.

No one symptom of any disease, nothing but the pathologico-anatomical conditions of the disease itself—as when portions of diseased structure, *e.g.* a tumour, are obtained during life—can fulfil the requirements of the most exact and earliest reliable diagnosis; yet, proportionally as symptoms are constant, exclusive, and early, they approach this standard.

But the *coexistence*, or at least the *consecutiveness*, of symptoms, any one of which is equivocal *per se*, makes a weight of evidence, *greater in the aggregate* than that which the *several* items of evidence would represent by being merely added together. This *augmentation* of evidence is by virtue of a law of mental association, and not a property of numbers or of any species of quantity. If in one balance of a weighing scale were placed a weight of five pounds, and in the other a weight of one pound, the balance is as one to five. Other weights of one pound each being successively added to the one-pound scale, would severally tend to equalize the balance; and five such weights would equal a five-pound weight; but five (separate) pounds weight, so to speak, of 'evidence,' taken *together*, *preponderate* over a single five-pound weight of adverse evidence.

To illustrate the force of concurrence by the evidence of secondary symptoms, I pass over the order of priority of these symptoms, and therefore overlook the relative value of each, considered as an early symptom. No one secondary symptom is sufficiently constant or peculiar to syphilis, to make their order of succession a question of much practical interest. But the fact of these symptoms being cotemporaneous, or at least consecutive, in the same person individually, outweighs their inexactitude. Let the weight of *anti*-syphilitic probability be represented by five; then any one of the five usual secondary symptoms may be absent, or, if present, may point perhaps to constitutional syphilis, perhaps to the mercurial crasis, perhaps to both, perhaps to neither of these blood-diseases, but proceed from other causes. Thus, the skin-eruption having a copper hue, the excavated ulcer of either or both tonsils, iritis, enlarged testicle, node, caries, necrosis,

are *severally equivocal* symptoms of (constitutional) syphilis; but taken *collectively* or as *consecutive* symptoms, they *outweigh* the supposed anti-syphilitic counterpoise. Constitutional syphilis is diagnosed by an over-balance of probabilities in its favour —this over-balance being due, not to the actual diagnostic value of each symptom or probability, but to their concurrence. So, also, other circumstances may corroborate our diagnosis. The fact of primary syphilis, present or antecedent, has its weight, the weight of an additional probability, concurring. We look for the remains of a presumed former chancre or chancres, and probably, also, the vestiges of a bubo or buboes.

The diagnosis of constitutional syphilis, therefore, clearly illustrates the (diagnostic) value of concurrent symptoms.

The 'blood-origin' of symptoms is established, in like manner, by *concurrent* evidence; but all the characters of blood-disease are rarely combined, or at least not in the same degree. Thus, constitutional syphilis is shown to be a blood-disease, partly by the 'number of textures' affected with some formof mal-nutrition, as of the skin, mucous membrane, the iris, periosteum, osseous texture, and testicle; partly by the 'migratory' character of these local affections from skin to mucous membrane, to the iris, thence to the periosteum and bone, perhaps to the testis; and partly by the 'symmetrical' character of some such local affections; but they are not serpiginous, like erysipelas.

On the other hand, constitutional syphilis is apparently not a blood-disease. Unlike small-pox, it is neither capable of being caught by 'infection' (inhalation of an atmosphere contaminated thereby), nor of being propagated by contagion, as by 'inoculation.'

While, however, all diseases produced by infection and propagated by contagion *are* blood-diseases, all blood-diseases are *not* thus produced and propagated; yet the characters of 'secondary symptoms' sufficiently concur to establish their 'blood-origin.' As such, they illustrate the Etiological Principle advanced in this chapter—the Constitutional origin of Local disease.

How far can the Principle of Rational Prevention be fulfilled

in the present state of Pathology? A sufficiently early detection of the blood-disease in operation can be accomplished, provided only the particular change which the blood itself has undergone can be (exactly) determined by chemical analysis, by the microscope, or by both these methods of investigation. Then, indeed, we should feel justified in letting a small quantity of blood for examination from any patient who has recently had 'primary' syphilis, without *preventive* treatment at that time. Otherwise, a chancre or chancres may have healed spontaneously or under simple local treatment, and have remained healed for some time; the only evidence of syphilis being the testimony of the individual that the primary sore was slow, perhaps very slow, to heal.

The blood therefore remains poisoned and ready to declare its noxious influence on nutrition—by some secondary syphilitic disease of the skin, throat, mouth or nose, by iritis, or perchance by irreparable textural destruction of the testicle, or by caries of the bones. This impending evil is sure to supervene in some form; the storm is sure to burst. There follows the indurated (infecting) chancre, says Ricord,* "a blood-diathesis pregnant with misfortunes and tempests. An infallible explosion of constitutional affections will ensue. When once produced, it is necessarily followed by symptoms peculiar to syphilis, and that within a space of time which, by long and patient observation, I am able to determine with precision. Among patients in Paris, and who are left without treatment, I affirm with the greatest certainty that *six months* will not elapse without manifestations of syphilitic intoxication." During this brooding period, a sufficiently early diagnosis of the blood-disease might be accomplished, and advantage taken of this one golden opportunity whereby the inevitable manifestations of constitutional syphilis could be anticipated by appropriate preventive treatment. But the time appears yet distant ere this essential diagnosis shall be realised.

---

* Op. cit.

Constitutional Syphilis shares the obscurity of other blood-diseases with few exceptions. The microscope exhibits nothing remarkable; chemical analysis, at present, brings nothing to light. The potent virus works unseen, being known only by the commotion it occasions. As when a diver has disappeared beneath the surface, we watch the troubled waters, without seeing *his* operations in the deep; likewise the syphilitic virus having dived into the blood, we know nothing of its doings there until it throws up some eruption on the skin or mucous membrane. Moreover, as the débris and bubbles thrown up by a diver cannot be distinguished from the commotion produced by some monster sporting in the deep; so also the scales and pustules of syphilis are subsequent and equivocal signs of the kind of poison at work. Meanwhile, its operation proceeds silently yet surely. The virus has not hitherto been detected in the blood; nor does inoculation with syphilitic blood manifest any characteristic results. Ricord failed to discover inoculable pus in the blood, even in veins nearest the chancre. MM. Ricord and Grassi first noticed "a decrease of the globular element in the blood of persons affected with syphilis arising from the simple or non-infecting chancre; and indurated chancre also is apparently followed by a diminished proportion of globules, while the albumen increases. Yet these changes are not remarkable; and to discover *the* blood-condition which precedes the local manifestations of constitutional syphilis, is still "a consummation devoutly to be wished." Rational Preventive treatment would then, and only then, be possible and practicable.

In this brief review of the career of Constitutional Syphilis, I have dwelt on the difficulty of identifying its local manifestations; and I have done so because a similar indefiniteness of character prevails in different degrees throughout the whole class of diseases having a blood-origin. Various forms of mal-nutrition, or, at least, hyperæmiæ,—each of which taken singly *may* not be characteristic, but a group or series of which, being taken collectively, together contrast with all other local diseases with which they can

be compared,—constitute the evidence by which diseases of 'blood-origin' are to be distinguished. The study, therefore, of a good example—like Secondary Syphilis—will *train* the mind to investigate all other blood-diseases, and serve the highly important purpose of showing how they can be diagnosticated by virtue of their local manifestations. But while this process of Analysis, Comparison, and Contrast is being conducted, that of Induction and Generalization should be employed to complete our investigations. The general characters therefore, by which *all* the local manifestations of secondary syphilis, for example, are fairly inferred to have a blood-origin, finished our review of the career of this disease, and illustrated a general Etiological Principle—the Constitutional origin of Local disease; associated with which is, the Prevention of whatever constitutional cause may be in operation, and this by applying our present knowledge of the particular blood-*crasis*, from which the group of local manifestations emanate.

Scrofula is another constitutional disease of 'blood-origin,' because possessing the family features of this class of diseases. In tracing these family features throughout the various local manifestations of scrofula, I shall not include those of an allied blood-disease—tuberculosis; for these diseases do not appear to be identical, as some pathologists have maintained, and even still allege. I endorse the view held by Mr. Paget, that *this* is their relation—"the scrofulous constitution implies a peculiar liability to the tuberculous diseases, and that they often coexist." But "their differences are evident, in that many instances of scrofula (in the ordinary meaning of the word) exist with intense and long-continued disease, but without tuberculous deposit; that as many instances of tuberculous disease may be found without any of the non-tuberculous affections of scrofula; that, as Mr. Simon has proved, while diseases of "defective power" may be experimentally produced in animals by insufficient nutriment and other debilitating influences, tuberculous diseases are hardly artificially producible; and that nearly all other diseases may coexist

with the scrofulous, but some are nearly incompatible with the tuberculous."*

Scrofula—thus distinguished from tuberculosis—exhibits itself locally by 'mal-nutrition and chronic inflammation.' This inflammation is scarcely expressed by pain, or heat, or redness, but rather by swelling, more or less considerable and doughy, slowly enlarging, and tending to suppuration; yet scrofulous suppuration is unwilling, so to speak, and the pus a mixture of curd and serum. Should a scrofulous abscess point, the skin thins, but gradually, and assumes a purplish tint; an irregular rent follows after some time, and the flaky matter rolls out. Perhaps this aperture gets blocked up and imperfectly closed; the matter reaccumulating, again to be discharged, and so on from time to time. Or the aperture may remain free, with puffy everted edges of a purplish colour, and the discharge continue—now thick, now thin. The *scrofulous ulcer* which eventually results is equally indolent. It persists, with a thin, livid, undermined margin, large, pale, flabby granulations, and a gleety discharge; although sometimes pretending to heal, by this discharge crusting over its surface. Should cicatrization ensue, the scrofulous cicatrix appears drawn, puckered, and incomplete. Small bridges form across the ulcer, underneath which a probe can be passed readily, in and out, here and there. Nature does but "skin and film the ulcerous spot."

These general characters distinguish scrofulous inflammation; but the local manifestations of scrofula present the usual characters of blood-disease.

Thus, scrofula is essentially a 'pervading' disease. It blossoms and bears fruit chiefly in the absorbent glands, in the skin and cellular texture, mucous membranes, bones and joints, eyes, salivary glands, tonsils, ears, breasts, and in the testicles. Then, again, in some cases, various parts are simultaneously affected, in others consecutively; the scrofulous affection 'migrating' from one texture or part to another texture or part. But their order

---

* Surg. Pathology, 1853, vol. ii.

of priority cannot be stated with accuracy. In some textures, the scrofulous affection is more *pronounced* than in others.

Absorbent glands, so called, appear to invite the deposit of scrofulous matter. At first soft and fleshy, these glands enlarge and harden; "portions of each gland are observed to have altogether lost their flesh colour, and acquired a degree of transparency, and a texture approaching to that of cartilage."* At length, a soft, white, or yellowish, curd-like substance is deposited. Glandular tumours, thus formed about the neck and groin, sometimes attain an enormous size; in the latter situation, being perhaps half as large as the head of a new-born child. An enlarged scrofulous gland is not necessarily impervious—at least, mercury can be injected in many instances.† Scrofulous glands are remarkably indolent, but eventually they soften and discharge the characteristic pus, — flaky and ichorous, perhaps cretaceous matter; or they remain as soft and spongy tumours, beneath a thin, silky cuticle, which frequently breaks and oozes; or they waste, and are at length represented only by "a few bands of condensed cellular tissue attached to the cicatrized integument."‡

Absorbent *vessels* are said to be rarely the receptacles of scrofulous matter, but that there are such instances on record.

Chronic enlargement with suppuration of the lymphatic glands is one of the earliest characteristic manifestations of scrofula. In childhood, therefore, these glands may be found as just described, in various stages of scrofulous inflammation and suppuration. Yet this is a rare event in children under two years of age. Thomson witnessed it earlier than this, and Cullen mentions a case in which the disease broke out at the very early period of three months. Taking the other extreme, Thomson found the mesenteric glands affected with scrofulous inflammation in persons of *very* advanced age.

In various parts, also, of the body, the lymphatic glands may become scrofulous. Those in the neck—*glandulæ concatenatæ*—

* Med.-Chir. Trans. Edin., vol. i. p. 683. Abercrombie.
† Cyclop. Practical Medicine, 1834, vol. iii. p. 705. ‡ Ibid.

are perhaps most frequently affected; and, according to Thomson's experience, scrofulous enlargement, &c. of these glands is more commonly symptomatic of irritation in neighbouring parts than an idiopathic condition,—provided, in either case, the scrofulous diathesis be present. This enlargement of the cervical glands is apt to arise from slight and transitory injuries and affections of the hairy scalp, ears, eyes, nose, and more particularly from slight and temporary affections of the teeth, gums, and other parts within the mouth. Decay of the first teeth is often the immediate cause of scrofulous glandular swellings in the neck, but their eruption, seldom or never.*

The axillary and crural glands are less frequently affected than those of the neck; and Thomson believes that their enlargement also is symptomatic, in this case, by absorption of scrofulous matter from parts more or less remote. This authority was acquainted with but few instances of idiopathic scrofulous swellings of the glands in the groin or armpit. The mesenteric glands are very liable to undergo scrofulous inflammation, constituting that formidable disease, *tabes mesenterica,* by arresting the absorption and passage of chyle through these glands, and thus inevitably depriving the whole body of its nutriment. A tumid abdomen, with progressive emaciation, begets suspicion of this disease; while detection of the mesenteric mass, by palpation and percussion, will go far towards confirming our diagnosis. Besides, however, the physical signs, and their interpretation, which Pathology supplies, there are, as regards all diseases, with few exceptions, other circumstances whose evidence should be weighed. We look for the concurrence of some other expressions of the scrofulous diathesis. Age, also, should be taken into account; but, in this respect, Thomson found the mesenteric glands affected in children two years old, in persons between twenty and thirty, and in those who had passed their sixtieth year.

The *cellular texture* is peculiarly liable to exhibit scrofulous swellings, bordering on suppuration or actual collections of matter.

* Lectures on Inflammation, p. 157.

In the subcutaneous cellular tissue, small *nodules* are apt to form, closely resembling scrofulous glands in appearance. Of this kind are the swellings described by Thomson as being "soft and puffy," and having little or no disposition to suppurate. "They often appear very suddenly; and from the absence of pain and discoloration, they may exist a long time without being perceived. They are usually of an oval figure, and seem to be produced by the effusion of a fluid into the interstices of the cellular texture; they are very variable in their size, being one day more prominent and tense, and the next more flaccid." Subcutaneous *abscesses* may form, and are usually numerous. When an absorbent gland suppurates and bursts, a fistulous sore is the result; but abscess in the subcutaneous cellular texture commonly terminates in an open scrofulous ulcer.

In the *sheaths of muscles,* large chronic abscesses sometimes gather insidiously, containing the pus which characterises scrofulous suppuration.

The *skin* is more than liable—it is prone—to scrofulous eruptions and ulceration. Its wrinkled seams and puckered scars are familiar to common observation. And these vestiges are not unfrequently 'symmetrically' disposed on either side of the body. Indeed, this symmetrical distribution is more common in scrofulous affections of the skin, or at least, is more apparent in that texture than in those of other textures. An instance of remarkably symmetrical scrofulous scars on the neck and fore part of the chest, in a patient of mine at the Royal Free Hospital, is described in the previous chapter. According to the special experience of Erasmus Wilson,* cutaneous scrofula is presented in two conditions—that of *tubercles,* and that of *ulcers.* Scrofulous tubercles are small, purplish or livid, indolent tumours. They soften internally and discharge an imperfect pus, remain open or fistulous for a long time, and on disappearing, frequently leave hard knots in the skin. They appear on the neck

* Diseases of the Skin, 1857.

and face, and near ulcers resulting from inflammation of the absorbent glands. When such tubercles have partially discharged their contents, a crust of inspissated matter forms, which being rubbed off occasionally, exhibits an open sore, with an ichorous discharge, and no disposition to heal. Eventually an ugly cicatrix or scar marks the site of these sores. Usually but one scrofulous tubercle arises; sometimes a group of three or four close together, which may have a circular arrangement, enclosing an area of thin, shining, livid or purplish skin. Rings of this kind occur chiefly on the back of the hands and feet. They are very intractable.

The characters of the scrofulous ulcer need not be repeated. An irregular, livid, and puckered scar is its remnant. Such cicatrices are seen mostly in the neck, near enlarged glands, and in the neighbourhood of joints.

Inflammation of the *matrix* of one or other of the *nails* is not uncommon, more particularly in young persons having the scrofulous diathesis. Scrofulo-derma ungueale, so named, begins by inflammation of the skin immediately around the edges of the nail about to be affected; then follows considerable swelling, with vivid redness of the end of the finger, extending even to the bone, and presenting the appearance of a clubbed finger. The nail is shed, disclosing an angry raw surface, upon which, from time to time, there re-appears a rugged, ill-formed, and imperfect nail. Fungous granulations and unhealthy pus continue for perhaps many months.

Other *cutaneous* manifestations of scrofula are noticed by some writers. Porrigo favosa, larvalis, and furfurans; eczema impetiginodes and rubrum, in their chronic forms; and that variety of lupus which appears as small, red, button-like, indolent tubercles, chiefly on the lips and nose, occasionally on the genitals. These tubercles excoriate and run into eroding ulcers, with pale, shining, spongy granulations and encrusted margins; or perhaps this work of destruction is concealed by a thick incrustation, which every now and then drops off, exposing its subjacent ravages.

The *osseous system* and the *joints* seem to invite scrofulous inflammation—in this respect contrasting with the indisposition evinced by these structures towards the syphilitic poison, unless reinforced by mercury. The bones and joints, then, are conspicuous in the history of scrofulous manifestations. And both may be coupled together, because it is *near* to joints that the bones are commonly affected. In the extremities of long bones, or in the bones of the carpus and tarsus, their cancellated portion, chiefly, undergoes that series of structural changes described in Chapter XI. So also the symptoms, or rather, signs by which these changes are denoted, are there traced consecutively. A reiteration of both may, therefore, be here omitted. I allude to scrofulous caries. Sometimes this species of mal-nutrition runs its course within the *shaft* of a long bone, but generally speaking, as I have said, in the neighbourhood of joints. The latter are secondarily invaded, and then this caries appears under the denomination of "scrofulous disease" of the joints. An inflammation of the synovial membrane, known as "scrofulous synovitis," was formerly regarded as a primary and distinct disease. More than probably, however, this is that (synovitis) which arises by extension of the destructive process from the cancellated bone.

In the career of scrofula, *mucous membranes* are not exempt from harm, particularly if its blood-relation, tuberculosis, be considered an ally. The eyes, ears, nose, upper lip, tongue, tonsils, salivary glands, and larynx, severally exhibit scrofulous inflammation; yet this is not altogether limited to the mucous membrane in connexion with these parts.

Scrofulous ophthalmia is a variety of conjunctivitis, characterized by great intolerance of light; so that the child (for this affection occurs mostly in young subjects) seeks a dark room, or buries its head in the bed-clothes, and screws its brows together with screaming agony on any attempt being made to examine the eyeball. From habitually endeavouring to exclude the light, the corrugator and orbicularis muscles become hypertrophied, even-

tually giving a remarkable heaviness of expression. When the eyelids are separated, a copious flow of tears trickles down the cheek, excoriating the face. The eyeball is now involuntarily up-turned to avoid the light, a patchy redness is observable on the conjunctiva, and vesicles or pustules are seen here and there at the margin or on the surface of the cornea. These pustules burst and expose small ulcers. Frequently an interstitial deposit overshadows the whole cornea, which thus becomes thickened and opaque (pannus), projecting also, so that the eyelids cannot be closed. This is one destructive sequel, and should ulceration of the cornea not terminate comparatively favourably—in specks of opacity, perforation of the anterior chamber is inevitable, the aqueous humour is discharged with prolapsus of the iris, and the eye collapses.

Fretting ulceration of the meibomian glands, attacking the margin more especially of the eyelids, and known as ophthalmia tarsi, is a frequent concomitant of scrofulous ophthalmia; or this diseased condition extends to the iris, giving rise to scrofulous iritis. But there is nothing characteristic about this variety of iritis, taken *per se;* and indeed it is only as one of a *series* of local manifestations that we venture to designate *this* 'scrofulous' iritis, and refer the whole series to one and the same constitutional cause in operation.

The organs of hearing do not escape. Chronic suppuration perforates the tympanum; the ossicula crumble, loosen, and are washed out by the discharge.

The nose assuredly enjoys no immunity. Habitual swelling, ulceration, and fetid discharge from the pituitary membrane—ozæna—may or may not be accompanied with caries and discharge of portions of the spongy bones.

The upper lip is commonly tumid, protuberant, and chapped. Fissures also and ulcerated spots are seen on the tongue. Nodules, moreover, superficially imbedded in the substance of this organ are said to arise in most instances, and to present the following characters. They vary in size from a small shot to that of a horse-

bean; are painless, unless subjected to firm pressure, which occasions a pricking sensation. The superimposed mucous membrane reddens, soon breaks in the centre, and forms an ulcer, which spreads and destroys by sloughing erosion; accompanied with much pain, profuse salivation, furred tongue, and fetid breath. If cicatrization ensue, hardness still remains; fresh nodules also form in other parts of the tongue.*

Chronic and considerable enlargement of the tonsils, with perhaps indolent ulceration, is another outbreak of the scrofulous diathesis; so likewise is swelling of the sub-lingual, sub-maxillary, and occasionally the parotid salivary glands; but these affections alike owe their significance to the invariable coexistence of other local diseases of more unequivocally scrofulous origin.

This remark holds good of another inflammation of mucous membrane, and the last which I shall notice in connexion with scrofula. Chronic laryngitis may be due to this constitutional cause. The vocal cords become thickened; the voice therefore is hoarse or squeaking, and the breathing embarrassed; a tickling cough from time to time ejects a slimy, not frothy, expectoration, streaked with blood perhaps; or the sputa are muco-purulent. In either case the breathing is relieved by this expectoration; but eventually ulceration of the rima glottidis renders its closure imperfect, the act of coughing incomplete, and expectoration therefore difficult; respiration is proportionately more embarrassed. Should ulceration of the epiglottis supervene, there will be a corresponding difficulty of deglutition. I need not enlarge this description; sufficient for my purpose to identify chronic laryngitis, while the invariable coexistence of other local affections of more exclusively scrofulous origin associates this disease with that series of local manifestations which proceed from the scrofulous blood-crasis.

In like manner I have to notice a certain *mammary tumour*, first described by Sir A. Cooper.† "In young women," says this

* Cyclop. Practical Medicine, 1834, vol. iii., art. Scrofula.

† Diseases of the Breast, 1829, chapter viii.

author, "who have enlargement of the cervical glands, I have sometimes, though rarely, seen tumours of a scrofulous nature form in their bosoms, confined in most cases to a single tumour in one breast; but in one case two existed in one breast, and one in the other." They are entirely unattended with pain, are distinctly circumscribed, are very smooth on their surfaces, and scarcely tender to pressure. They are very indolent, but vary with the state of the constitution, diminishing as it improves, and increasing as the general health is deteriorating. They can only be distinguished from simple chronic inflammation of the breast, by the absence of tenderness, and by the existence of other diseases of a similar kind in the absorbent glands of other parts of the body. They produce no dangerous effects, and do not degenerate into malignancy."

Lastly, a peculiar enlargement of the testicle, or rather, of the *epididymis*, is worthy of special notice among the local manifestations of scrofula; and it particularly exhibits the usual characters of stealthiness and slow development. A small nodule, consisting of yellow friable matter deposited within the tubules or ducts, appears generally at one end of the epididymis; little pain or tenderness attends this structural change, and it may progress without complaint. Another and another such nodule forms on the surface of the testis, but generally connected with the epididymis, which becomes beset with three or four small tumours. Thus the testicle itself feels enlarged and irregular at an early stage of this disease.

It has been stated* that scrofulous matter is also deposited within the body of the testis, in the form of pearly or greyish bodies, of the shape and size of millet-seeds, *i.e.* grey granular tubercles, which I suppose this description denotes. That these tubercles have a linear arrangement, like strung beads, less abundant and less regular in the anterior part of the organ than towards the rete-testis, where they are closely set, and sometimes

---

* Diseases of the Testis, Curling, 1856.

confluent; and that they undergo transformation into a yellow friable cheesy substance, which at a later period softens, and is often broken up into a curdy purulent fluid, the gland-structure being absorbed to give place to this tuberculous matter. But, if tubercles are deposited within the testicle itself, this structural change signifies little in reference to an early and exact diagnosis, for the "testis is often masked by small local effusions of fluid in the tunica vaginalis," the surfaces of which are partially adherent.

Now, the epididymis may remain nodose for many months; the nodules quiescent, or enlarging very slowly, and becoming painful. At length one declares itself more than the rest, attaches itself to the skin, which then assumes a purplish discoloration, ulcerates, and discharges a curdy purulent matter—the substance of the nodule. Other nodules undergo successively this process of disintegration, softening, and evacuation; but, unlike healthy abscesses, they do not heal. Fistulous openings obstinately continue to exude a mixed discharge—now curdy, now serous, now seminal; and in this advanced stage of the disease, destruction of more or less of the gland-substance is inevitable. According to Sir B. Brodie's observations,* occasionally one testis is completely disorganized; more frequently the organ is only partially destroyed, and a considerable portion of the glandular structure remains unimpaired. Sometimes the disease is confined to one testicle; sometimes both are similarly involved.

By this process of disorganization and protracted discharge the testicle is drilled and worm-eaten, as it were; so that eventually the organ collapses and shrivels up—a fragment only of its former self. There is seldom, therefore, any protrusion of gland-substance through the fistulous scrotum; on the contrary, in a favourable case the apertures gradually become inverted and depressed,—leaving, after the lapse of time, a puckered cicatrix, adherent to the remaining portion of gland, as a lasting record of all this mischief.

---

* London Med. Gazette, vol. iii. p. 377.

In concluding this summary of many local diseases, which, possessing the same general characters, are fairly associated under the name of Scrofula, it is impossible to overlook at least two general facts that indicate their blood-origin. These diseases are perversions of nutrition in 'many' textures and organs; and they 'migrate' from one locality to another. So, therefore, scrofula is, properly speaking, a blood-disease, and the diseases alluded to are local manifestations of a constitutional cause in operation.

Now, certain bodily organizations evince a tendency to scrofula, and beget a suspicion that it will declare itself in some way, sooner or later. Individuals thus constituted are ever verging on this morbid condition, with threatenings of its outbreak here or there; but it must be confessed that no one 'temperament' alone possesses this (scrofulous) character. It is the tendency of those whose circulation is habitually weak—are *leuco-phlegmatic*—who have flabby muscles, a dull muddy complexion, large heads, pigeon-breasts, tumid bellies, and large joints; but then the strumous tendency is manifested in those persons also who, with a more active circulation, are rather of the *sanguine* temperament, have firmer muscles, a clear, transparent, ruddy complexion, which readily assumes a purple or livid hue by exposure to cold. The circulation, although active, is susceptible. Chilblains, therefore, not uncommonly occur in children of this temperament; while their yellow or reddish hair, large lustrous blue eyes, crimson-patched cheeks, and pouting upper lip, are associated with that lively, impulsive, affectionate, and precocious disposition which so often raises hopeful expectations, never to be realized.

In contrast, however, with this organization and with these mental endowments, the same strumous tendency may be evinced in the highest degree by those who, without any marked character of circulation, are habitually subject to biliousness. In such persons the liver seems to be their weak point. Sluggish, yet enduring power is theirs also; and hair approaching black in colour, a dark olive or yellowish complexion, and dry skin, are aptly associated with a gloomy, often resolute, and reflective dis-

position. Theirs is the *melancholic* temperament. These are the chief signs of the scrofulous *diathesis*, although it *may* appear also in persons of the *nervous* temperament; and, indeed, this tendency can be induced in those who are congenitally most indisposed to it when subjected to circumstances favourable to its development.

Whatever impairs the nutritive qualities of the blood and its circulation, predisposes to scrofula; therefore, deficient or defective food, insufficient ventilation, want of cleanliness and excretion, poor clothing, cold, damp, and even dark localities, with other *circumstances* of similar character, are the nurseries and nurses of this blood-disease. At the same time, *individual* predisposition, as usual, plays its part; for among a family of children in precisely the same hygienic circumstances, one becomes scrofulous, while the rest escape.

But all these causes, whether 'internal' or 'external,' alike join issue in the *blood;* so that I have spoken of its morbid condition as the origin, or immediate cause, of the various local scrofulous manifestations. *They* also concur in indicating some morbid condition of the blood. What is this? The only sure way of (rationally) fulfilling the Principle of Prevention as regards the manifestations of Scrofula, presupposes the earliest and most exact knowledge of the particular blood-*crasis* in operation, or about to be. Of causes not immediate, some only in the shape of predisposition cannot be removed; these, however, by themselves, *may* prove *insufficient* to induce scrofula; other such causes, being external, can be *removed:* but *all* the hygienic conditions conducive to scrofula are not known; some remain undetected; and as *these* (remote) causes in like manner are reflected in the blood-crasis, the question of prevention necessarily involves this essential element in the pathology of scrofula. Yet what says pathological chemistry on this point? That the blood is rich in its amount of fibrin, but this of inferior plastic quality, and poor in its proportion of red corpuscles;—such is the answer given by Andral and Gavarret: that the "solids of the serum" are in-

creased, and the "blood-globules" diminished, was the reply of Dr. Glover,* according to his analysis; and so on I might enumerate the conclusions of many other distinguished chemists.

With these indefinite, if not conflicting, views to guide the Preventive practitioner, it is vain to hope for success with any justifiable confidence. When, therefore, either a predisposing temperament, or (external) hygienic conditions favourable to scrofula, are met with, it is impossible, in the present state of knowledge, to interpose any measures which, restoring the blood to its healthy condition, and at a sufficiently early period, are entitled to be considered preventive of scrofulous diseases of nutrition.

Scurvy and Purpura are diseases obviously of 'blood-origin;' they are therefore here placed in the same category as Secondary Syphilis and Scrofula. And as with them, so with scurvy and purpura: taking consecutively the Etiological Principle involved in 'blood-origin,' and its Preventive consideration; we have, in the first instance, to draw the line of distinction betwixt these diseases and all others, and between themselves. Their diagnosis demands our first consideration.

Now, the characteristic phenomena of these diseases are easily recognised. Both, as regards their local manifestations, are 'hemorrhages' into many textures, contemporaneously or consecutively; and it will be readily imagined that these hemorrhages occur with greater facility in some textures than in others. Into *cellular tissue*, for example, blood is very apt to escape. I have already† briefly described the appearances presented in scurvy. A sallow and dejected-looking man, whose strength has been gradually failing, is at length prostrate. Our patient's gums are turgid, spongy, and rotten; they bleed on the slightest pressure; the teeth loosen in their sockets and drop out. This fungous condition of the gums ceases abruptly at the reflexion of the mucous mem-

---

* Pathology and Treatment of Scrofula. Fothergillian Prize Essay, 1846, p. 115.

† Chapter VII.

brane to the lips, which are extremely pale; so also are the tongue, fauces, and inside of the cheeks. In some rare instances, however, this lividity extends nearly all over the hard palate. Red or livid spots are found—principally on the legs—together with bruised-looking patches, of a yellowish-green colour, swollen and hard as brawn.

Extensive effusions of 'fibrin,' rather than pure blood,—forming very hard, broad, and painful swellings,—are found imbedded in the deep cellular texture and between muscles. Over these fibrinous mats, the skin sometimes retains its natural colour, but usually appears bruised—is always thickened and brawny, and adherent to the subjacent textures. Swellings such as these occur particularly in the thighs and legs, but most commonly in the hams, occasioning stiffness and contraction of the knee-joint. Nodes also arise from this effusion taking place between the bones and their periosteal investment; the tight swellings formed thereby giving great pain upon the slightest motion, even by turning in bed. None of these effusions, whether of fibrin or blood, ever suppurate; nor do the nodes just mentioned, however large their size, ever cause the bone to exfoliate.

The dark livid or purple colour of scurvy overshadows any skin-eruption, wound, or ulcer, which may chance to be present during this disease. A dark grumous coagulum juts out from the surface of an ulcer; and this—which, owing to its appearance, has been named by sailors *bullock's liver*—often attains an incredible size in the course of a single night. To conclude the catalogue of hemorrhagic lesions, repeated issues of blood from the nose are common; blood may be coughed up or vomited, lost by the bowels, and perhaps passed with the urine.

Scurvy is apt to prove fatal suddenly, from exhaustion. This remarkable feature in its career, with others of instructive moment, were exemplified in an equally remarkable manner during Lord Anson's expedition, 1740-44. The narrative states:—"Many of our people, though confined to their hammocks, ate and drank heartily, were cheerful, and talked with much seeming vigour and

in a loud, strong tone of voice; yet, on their being the least moved, though it was only from one part of the ship to another, and that in their hammocks, they immediately expired; others, who confided in their seeming strength, and resolved to get out of their hammocks, died before they could reach the deck. It was no uncommon thing for those who could do some kind of duty and walk the deck, to drop down dead in an instant, on any endeavour to act with their utmost vigour; many of our people having thus perished during the course of this voyage."

Similar phenomena characterize Purpura, but there is not the same marked dejection and feebleness, nor are the gums always fungous. Extravasation of blood occurs in the form of small, round, purple spots, rather than parti-coloured blotches. These spots of blood are scattered in almost every texture. According to Dr. Watson's experience,* they are not peculiar to the skin, nor to the subcutaneous tissues, but occur occasionally upon all the internal surfaces also, and within the substance of the viscera. For example, on the mucous membrane of the mouth, throat, stomach, and intestines; on the pleuræ and pericardium, in the chest; on the peritoneal investment of the abdominal organs; in the substance of the muscles; and even upon the membranes of the brain, and in the sheaths of the larger nerves; and they may be accompanied with large extravasations of blood in most of the vital organs of the body. Such lesions are necessarily perilous. Bateman states that he witnessed three instances in which persons were carried off, while affected with purpura, by hemorrhage into the lungs. Watson saw two post-mortem examinations, in both of which a considerable quantity of blood was found spread over the surface of the brain, between its membranes; and in one of these cases blood was extravasated also into the cerebral substance, with extensive laceration.

Scurvy and Purpura are *plainly* of 'blood-origin.' The *blood* itself spontaneously exudes, and appears as a bruise, yet without any

---

* Lectures on the Principles and Practice of Physic, 4th Edit.

bruising force having been applied. This hemorrhage and ecchymosis takes place in many textures, and visits one after another. The whole organism, in fact, becomes leaky, yet without the blood-vessels themselves being in any diseased state.

To what morbid condition of the blood must the hemorrhage of scurvy be ascribed? The *rational* mode of preventing such hemorrhage necessarily presupposes a due knowledge of this essential point. To the blood all our preventive measures should have immediate reference, and therefore it is imperative that we first discover in what respects this vital fluid is impaired.

But in the present state of Chemical Pathology, according to one authority, *potash* is *deficient*. Dr. Garrod's analyses* lead him to conclude that the proportion of this alkali is reduced. Other authorities—Becquerel and Rodier†—find the proportion of *soda increased* in scorbutic blood, and that of *fibrin diminished*.

Let us see how far these unsettled views affect the prevention of Scurvy.

It is an undoubted fact that certain articles of diet possess anti-scorbutic properties. Lemon-juice is the grand anti-scorbutic, whereby thousands of persons have been rescued, who otherwise would inevitably have perished from scurvy. 1457 cases of scurvy were sent to Haslar Hospital in the year 1780. Subsequently, in 1795, lemon-juice was provided by order of the Admiralty, through the representation of Sir Gilbert Blane and Dr. Blair. Then only one case of scurvy appears in the hospital returns for 1806; and for 1807, one. Potatoes, whether in a raw state or cooked, are equally anti-scorbutic. Many other articles of diet, more or less in use, are enumerated by Dr. W. Budd,‡ and their anti-scorbutic properties compared. So far, the prevention of scurvy is practicable. But this kind of knowledge is *empirical*; we are ignorant (as Dr. Budd justly remarks) of the *essential*

---

* Edin. Month. Journ., 1848.
† Pathological Chemistry, 1853.
‡ Library of Medicine, art. Scurvy.

element common to the juices of anti-scorbutic plants, and in which their efficacy resides. Therefore, one plant cannot be substituted for another—prior to actual experience of its anti-scorbutic value—with the sure and certain prediction that it will prove equally efficacious, or more so. And why are we still ignorant of this "essential element," and incapable, consequently, of substituting an untried for a known anti-scorbutic? Because the "blood-condition" essential to scurvy is yet unknown, and *that something, by virtue* of which various plants are anti-scorbutic, is *therefore* equally unknown. Accordingly, the prevention of scurvy can be determined only by the results of actual experience.

Mark the *further* consequences of this empiricism. If *potash* were assuredly (known to be) *the* thing in question, it could be procured in almost any emergency from the ashes of any plant or of any wood, and especially, as Dr. Garrod has suggested, from that ubiquitous weed, tobacco, which is rich in potash.

In the present state of knowledge, the commissariat of an army, a navy, or commercial marine—ever liable to be placed in straitened circumstances with regard to all food—are in the dark on this most critical point. In encampments far from home, during sieges and long voyages, the allowance of lemon-juice has perhaps long been exhausted, and fresh vegetables are a dream; when, therefore, under these adverse circumstances, scurvy stealthily threatens, with pallid hue and dejected mien, the light of Pathological Chemistry would supply an unerring guide in search of that yet unknown something—be it potash or whatever else—by which the impending scourge would assuredly be averted. It might be possible, by a simple process perhaps, to extract that needful something from an abundant source at hand, in a locality otherwise well-nigh barren and desolate.

To conclude, on behalf of the prevention of this disease, any new and *untried* kind of food cannot be *substituted*, in an emergency, for another less plentiful; and, should all food run short, then the *essentially* anti-scorbutic constituent cannot be *extracted* from substances perchance close at hand, in which, like a precious

pearl, it remains undiscovered, while scurvy is already overshadowing its victims. Empirical experience is our only resource under these circumstances, and the rules which it authorizes for our guidance are necessarily of a very general character. They are enumerated by Dr. Budd as follows:—

1stly. Anti-scorbutic properties reside exclusively in substances of vegetable origin.

2ndly. These properties are possessed in very different degrees by different families of plants; least so by the farinaceous, as wheat, oats, barley; most so by the succulent, as the aurantiæ, comprising oranges and lemons; lastly by potatoes.

3rdly. The anti-scorbutic property is impaired by the action of strong heat; nevertheless, boiled potatoes are anti-scorbutic (Dr. W. Baly); impaired, also, by vinous fermentation, but improved probably by acetous fermentation.

To show the difficulty of approaching the prevention of scurvy by this method of investigation, I might add, in striking contrast, the conclusions of Dr. Christison in favour of *azotised* substances, and of *animal* origin, perhaps exclusively,* such as milk.

These and similar discrepancies, on the part of practised observers, should direct the attention of chemists to the (unknown) 'blood-condition' as the only standard of comparison whereby to estimate the anti-scorbutic value of different substances, their composition being presumed to be known.

In like manner the prevention of Purpura should be made the subject of original inquiry.

Although resembling scurvy in appearance, the curative treatment of purpura, by abstinence, purgation, and venesection, as recommended by the late Dr. Parry, of Bath, is altogether at variance with that which is so efficacious in cases of scurvy; and points, therefore, to some essential difference between these two diseases, otherwise allied. The manifestations of both are unquestionably of blood-origin, yet essentially different in this respect;

---

* See chapter vii., p. 257.

and not until Chemical Pathology has determined the blood-condition *peculiar* to Purpura will it be possible to interpret rightly the whole etiology of this disease. Its rational prevention will follow.

Taking next in order those blood-diseases which manifest themselves locally in *special* textures, I shall consider—

1stly. Those that are declared by the skin and mucous membranes—singly or conjointly.

This class includes—

a. Two *non*-infectious fevers—gangrenous typhoid and hectic, the types of which correspond to those of certain infectious fevers; the former answering to continued fever—typhus and typhoid—the latter to relapsing fever.

The facts and arguments adduced in Chapter XI. show the dependency of these non-infectious fevers respectively on some blood-condition peculiar to each. Both are fevers of blood-origin as regards their *immediate* cause, however varied may be the local lesions from whence they emanate; and their blood-conditions are reflected on the skin and mucous membranes, although these textures do not exhibit any perceptible structural change.

b. Infectious diseases—or more correctly speaking, diseases caught by infection, *i.e.* by inhalation — belong also to this class.

'Eruptive fevers' are blood-diseases, manifested by the skin and mucous membranes, which, singly or conjointly, present certain definite forms of inflammation, having distinctive characters, and by which they can be accurately distinguished. These fevers are the "exanthemata" of Cullen; they are "contagious diseases, affecting a person once only in the course of his life, beginning with fever. At a definite period small inflammations appear, often numerous, scattered over the skin." This group comprises typhus fever, typhoid fever, and relapsing fever, measles, scarlet fever, small-pox, chicken-pox, erysipelas, and plague.

Other infectious diseases, *not* thus characterized, are hooping-cough and glanders.

c. Epidemic diseases are fairly included among "blood-diseases, manifested either by the skin or mucous membranes, or by both these textures." According to the Registrar-general's reports, they are ten in number—namely, small-pox, measles, scarlatina, hooping-cough, influenza, croup, thrush, cholera, diarrhœa, and dysentery. *Some*, therefore, of these diseases are also infectious; but one and all prevail epidemically, *i.e.*, spread rapidly over the population of a whole country, evidently without personal communication. And the recurrence of such visitations is independent of any particular season of the year.

d. Endemic diseases may be associated with the foregoing. They comprise 'malarious' fever, of which there are different species. I allude to *intermittent* fevers, *e.g.* ague, in which the paroxysms occur, as expressed by the terms—quotidian, tertian, and quartan agues. *Remittent* fevers also — in which the paroxysms have no distinct cessation or intermissions from time to time, but only remissions—are of similar character, *e.g.* yellow fever (?)

The fever of ague is determined to the skin, as shown by critical sweats; while yellow fever is expended on the intestinal mucous membrane (?)

'Malarious' fever of every kind is due to a miasm, an effluvium or aerial poison, *inhaled*, and in this single particular resembles the various kinds of 'infectious' fever. But the malarious poison does not proceed from the body of another infected person; it arises from marshes, jungles, and other swampy localities; for when the wind sweeps across such districts, persons who reside to leeward become affected, while those to windward escape. The source, therefore, of malarious fever, is not an animal poison, like that issuing from infected persons; nor do the subjects of malarious fever communicate their disease to others; in both these respects contrasting with infectious fevers, properly so called.

Unlike Epidemic disease also, 'malarious' fever is evidently connected with the season of the year, and moreover is limited to that particular district from whence it emanates and spreads among the people therein.

Other endemic diseases, although of malarious origin, are not exhibited by the skin or mucous membranes. Bronchocele, or goître, is an enlargement of the thyroid body; and cretinism, a general abortion of the whole organism and of the powers alike of mind and body.

Lastly, *many* endemic diseases are *non*-malarious, although of equally circumscribed geographical distribution, in keeping with the essential character of all diseases denominated endemic. The plica of Poland, and the pellagra of Northern Italy, are the offspring of filth; and the Guinea-worm is developed in the bodies of those people who are in the habit of drinking water containing its ova.

The generic term Endemic disease, therefore, includes many species of very different kinds, *some* only of which fall within the class I am about to consider—namely, that of blood-diseases manifested locally, either by the skin or mucous membranes, or by both these textures.

Omitting the non-infectious diseases—gangrenous typhoid and hectic fevers, which will subsequently engage our attention—*Infectious* blood-diseases now claim an adequate space in the present chapter. Their 'discrimination' and 'blood-origin' are respectively most important in reference to their Prevention; and the prevention of these diseases is of National concern, because of their desolating mortality.

Eruptive fevers come first in order. They were all formerly regarded as identical; measles and scarlet fever were not believed to be distinct diseases until the publication of Dr. Withering's Essay; while typhus, typhoid, and relapsing fevers were recognised as but varieties of one disease, which presented differences merely in its phases, according to epidemic and individual peculiarities and hygienic conditions. Agreeably to this

view, many works and monographs were published.* Of an opposite tendency, however, were the researches and investigations of some few authors;† but it was reserved for Dr. W. Jenner ‡ to *fully* establish the difference of these three fevers, in the sense of their being *distinct species*.

Guided by his observations, the diagnosis of typhus and typhoid fevers turns on the skin-eruption when present, while that of relapsing fever is to be determined by its course and symptoms. The full proof of the essential distinction of these fevers is supplied by their etiology. They are equally the offspring of infection, but exposure to the source of either fever will generate only its own kind, and neither of the other two, which are thus shown to be distinct species. I may add that this fact points not merely to the cause of each being 'specific,' but also to *distinct* specific causes; and this question of identity or non-identity respecting the specific causes of these fevers, and thence of their essential distinction, was the final purpose of Dr. Jenner's investigations.

In conformity with the plan of this chapter, I shall first consider the diagnostic character of these diseases. Here the observations referred to are the most original source of information; I shall not hesitate, therefore, to avail myself of their results.

Typhus fever is known by its "mulberry rash." This eruption appears from the fifth to the seventh day, and reaches its maximum amount in a day or two. It occupies the trunk and extremities, and occasionally the face. It consists of distinct spots and a subcuticular rash. The frequent absence of one of these elements of this eruption, the proportions they bear to each other, their depth of hue, as well as the changes their physical

* Library of Medicine, art. Fever (Christison). Lectures on Principles and Practice of Physic (Watson), 2nd Edit. British and Foreign Med.-Chir. Review, vol. xii.

† American Journal of Med. Sciences, 1837, art. by Gerhard. Cases collected by Shatmak, analysed by Valleix. Monograph (A. D. Stewart), 1840. Fever (Bartlett), 1842.

‡ Monthly Journ. Med. Sci., 1849. Serial communications Med.-Chir. Trans., 1850.

characters undergo, represent considerable varieties as regards the appearance of the rash in individual cases.

Distinct Spots.—Their number varies; in most cases they are very numerous, in some only being very few, and pretty equally diffused over the whole surface, while in others, with but few spots on the anterior surface of the body, the posterior is covered. Their size varies from a mere point to two, three, or four lines in diameter. Sometimes two or three spots coalescing here and there, form very large irregularly-shaped patches. These spots pass through two, and in many cases three stages.

First Stage.—They are slightly elevated, somewhat flattened on their surface; have a dusky pinkish red colour, somewhat like the stains of mulberry-juice, and disappear completely on pressure, re-assuming their distinctive characters as the finger is being withdrawn.

Second Stage.—In from one to three days these spots undergo a marked change; they are no longer elevated above the level of the cuticle; their hue grows darker and more dingy, and instead of disappearing on pressure they only fade. In some cases, on reaching this stage, they pass into faintly-marked reddish-brown stains, and then vanish.

Third Stage.—In many cases, and especially those that are severe, the spots reach this stage; their centres become dark purple, and are unaltered in appearance by the firmest pressure, although their circumferences still fade; frequently entire spots, circumference as well as centre, change into petechiæ.

Each spot endures from its first appearance till the termination of the disease; but a few large, almost scarlet patches, occasionally seen on the back of the hand on the fifth or sixth day of the disease, usually disappear entirely in a day or two.

The Subcuticular Rash.—When the trunk is covered with mulberry-rash, many spots are usually pale, very imperfectly defined as such, and run into each other; these spots appear as if situated beneath the cuticle, or, in common parlance, "are not well out." They give to the skin a mottled aspect, on which, as

on a ground, the darker, more defined, and distinct spots are situated. Like them, the subcuticular rash is deepest coloured on the most dependent parts of the body.

The subcuticular rash may precede, or be preceded, for a day or two, by the distinct spots; *i.e.* the eruption is for a day or two very pale, and then some spots grow more distinct; or a few well-marked ones first appear, and then after a day or two the rash becomes more abundant.

The diagnostic characters which *distinguish* the spots of typhoid from those of typhus fever, are differences in point of their colour, shape, duration, and the changes they severally undergo in the course of each disease.

Typhoid Fever—*Rose-spots.*—This eruption appears from the seventh to the twelfth day of the disease; very rarely later, and still more rarely at an earlier period. The characteristic spots are frequently preceded for a day or two by a very delicate scarlet tint of the whole skin, which closely resembles that of the skin of a person soon after leaving a hot bath. (When more marked than usual, and sore-throat also is present, this tint may be mistaken for the rash of scarlet fever). The eruption itself consists of small spots irregularly scattered over the anterior and posterior surface of the trunk. The number of spots on the surface at one time ordinarily ranges from six to twenty; in some cases there are very few, and far between; in others, but infinitely more rarely, they are distributed so profusely that scarcely an interval of normal cuticle is free between them.

The separate spots are circular, and of a bright rose-colour, which passes insensibly at their basis into that of the surrounding cuticle. Their usual diameter is about two lines. They are somewhat elevated; but, although perceptible to the finger passed lightly over the surface, have not the seed-like hardness of the first day's eruption of small-pox, nor are they so prominent and perceptible to the touch as the papulæ of lichen; their surface is rounded, lens-shaped, never acuminated. No trace of vesication can be detected on their apices. If tolerably firm pressure

be made on these spots they entirely disappear, but they resume their distinctive colour and elevation as the finger is withdrawn.

These characters are preserved by each spot from its first appearance to its disappearance. When, however, the duration of a spot is prolonged to five or six days, it usually becomes, before that time, very small, and less bright in colour; still it disappears on pressure. The ordinary duration of each spot is about two days, but it varies from two to six. Fresh spots appear every day or two, from the outset of the eruption till from the twenty-first to the twenty-eighth day of the disease.

This successive daily eruption of a few small, very slightly elevated, rose-coloured spots, disappearing on pressure, each spot continuing visible for three or four days only, is, in Dr. Jenner's experience, peculiar to, and absolutely diagnostic of, typhoid fever.

Moreover, the 'anatomical' characters of their eruptions being invariable and peculiar to typhus and typhoid fevers, each to each, are therefore *relatively* diagnostic. In distinguishing these two diseases by their eruptions alone, not a single error was made during the course of two years clinical observation, so far as could be proved by examination after death of the fatal cases, or by the progress of the non-fatal cases after their diagnosis had been recorded.*

The anatomical nature of these eruptions formed part of Jenner's observations. Respecting that of typhoid fever—in one case only was there a trace after death of any spot which had been marked, during life, with a circle of ink for identification. This, a very faint brownish point, on section was found limited to the surface; it did not extend into the cutis. In typhus fever, when the spots had advanced to their *second* or *third* stage, and were ineffaceable under pressure applied during life, they were persistent also after death. This advanced eruption was most conspicuous on the posterior aspect of the body. A piece of skin being removed for more minute examination, inspection, aided by

---

* Monthly Journ. Med. Science, 1849.

a lens, showed a faint brownish-red or purple hue, coinciding with each of those spots that had been observed to fade only on pressure during life; but this hue was limited to the *surface* of the cutis. The purple colour of the well-defined spots, and which were ineffaceable during life, extended through the *whole* substance of the cutis, and even beyond into the subcutaneous tissue; a few capillary vessels, still loaded with blood, converged towards each of these spots, but none were discernible in the spots themselves; their colour appeared to be due to infiltration of the tissue by solution of the hæmatin. The hue of these spots was darker below the epidermis than on the surface.

No death having occurred in this series of observations, while the mulberry-rash was in its *first* stage, left it an open question whether or not that phase of the eruption would have been visible after death.

Both these eruptions, although characteristic when present, are absent in *some* cases. The mulberry mottling of typhus, and the rose-coloured spots of typhoid fever, *may* alike never make their appearance. We must look, therefore, for other distinctive characters, if such there be, to *complete* the diagnosis of these diseases. But their symptoms are less definite; they are those of 'functional disturbances,' the aggregate of which constitute the Fever itself, whether typhus or typhoid. A few words will convey a general idea of this Fever, and then some shades of difference can be drawn.

The earliest, and subsequently the predominant, character of this Fever, is depression of all the functions of animal life. The 'nervous system' exhibits this depression in various ways. For some days prior to the actual accession of fever, the fever-struck person flags. He lounges about, incapable of his usual pursuits or occupation, unnerved and unstrung, fatigued in mind and body. With a feeble gait and dejected mien, he looks pale and spiritless. Without appetite, thirst may be unquenchable; at length the night comes, but not so any refreshing sleep. Broken dreamy slumbers prolong each night, and weariness each day.

In about a *week—shivering* inaugurates the fever. Severe headache, clasping across the forehead, bewilders; the face is flushed, the eyes are blood-shot; and now staggering about, as if muddled by drink, the patient, quite knocked up, goes to bed, though not to rest. The heart soon responds to this *febrile oppression*, the pulse rises in frequency to a hundred, a hundred and twenty or more beats per minute; but it is also weak, jarring, and compressible; for the heart's action is ineffectual. The potent poison works, overwhelming the nervous system; its spell-bound victim lies powerless and motionless on his back, sleepless, and with his eyes open, yet insensible to all things around—the *coma vigil* of Jenner. Or every now and then a disturbed doze steeps the patient in forgetfulness, itself forgotten by him when questioned; otherwise, on being roused, he replies rationally, although with a puzzled manner, and soon relapses into his former lethargy. Now also the fæces are voided involuntarily and heedlessly; the urine likewise dribbles away; in fact, the sphincter muscles of the anus and bladder are paralyzed.

It would appear as if the 'functions of organic life' were struggling to throw off by excretion something noxious. Thus the breath becomes fetid; a white fur overspreads the central portion of the tongue, often divided by a brown line; the urine is high-coloured, offensive, scanty; the skin dry and pungent, or moist and cooler; the bowels constipated, or voiding thin pultaceous excrements from time to time.

In the course of another, the *second week* of this Fever, *exhaustion* is more subduing, the victim more helpless. Still supine, he glides down in bed, and if replaced in his former position, knows it not, but lies with an open black mouth,—too weak to speak aloud, and perhaps to swallow, or to protrude his tongue. Black crusts — *sordes* — have formed on the tongue, teeth, and lips; a fair criterion of the depraved state of the secretions in general. *Delirium* supervening, the patient mumbles incoherently, and talks quietly to himself; or he apparently addresses others, with a raving loud voice, and makes efforts as if

to escape from some restraint. Spasmodic twitchings of the muscles of the hands, arms, or face, still evince the subjugation of voluntary power. There may be general convulsions. Yet with all this spurious exaltation of the nervous centres, the functions of special sense gradually become obscured. Deafness, defective taste, smell, touch, and imperfect vision gradually withdraw the poor wanderer more and more from this world. Small black objects appear to flit across the area of his veiled vision; in vain his hands saunter tremulously over the bed-clothes to pick away these creatures of his imagination; in vain do friends whisper around him: there is no recognition and no response; *coma* overshadows all things with oblivion; the mind is not there; —he is dead. Occasionally, other modes of death,—*apnœa, cardiac syncope,* or both combined, are witnessed.

Certain shades of difference, perhaps points of distinction, can be drawn between the fevers,—typhus and typhoid; and these points of evidence, in aid of their diagnosis by the two eruptions, are worthy of notice, especially in further illustration of that concurrence of evidence by which alone any diagnosis can be determined. The fevers in question are to be distinguished, not by comparing *any single symptom* of either, but by the *concurrence* of many symptoms, which *collectively* contrast. This 'weight' of evidence comprises symptoms and anatomical characters; external causes also, when they can be determined; and even the behaviour of a disease under a given mode of therapeutic treatment, may be thrown into the scale. This concurrence of evidence must be *repeated* again and again; its weight, respecting the identity or the non-identity of any two diseases, thus increasing proportionably. Concurring evidence, to be conclusive, therefore implies an appeal to the results of 'statistics;' which, in proportion as their number, if accurate, increases, become more and more conclusive. The diagnosis of typhus and typhoid fevers—as established by Dr. Jenner—is a *model* of the results of statistical investigation, ever worthy of admiration and imitation.

Accordingly, I have collated the following results of this method, chiefly to inculcate its *educational* value, as applied to clinical observations in general, and diagnosis in particular. The fevers in question are thereby found to contrast, more or less, in at least twenty-four particulars; the *aggregate* of which—like the features and other personalities of any two individuals—present two distinct portraits.

1. Age.—*typhoid* fever was limited to persons under forty; but, in nearly one-third of forty-three cases of *typhus*, the patients were over fifty years of age.

2. Mode of Attack.—*typhoid* fever, as a general rule, commenced more insidiously than *typhus*.

3. Duration.—In *typhoid* fever, the average duration of fatal cases was twenty-two days; that of *typhus*, fourteen days. In *typhoid* fever, half the cases survived the twentieth day of the disease; in *typhus*, not a single case survived that period.

4. Appetite and Thirst.—In *both* these fevers, loss of appetite was one of the earliest symptoms, and continued till their termination. In both also, more or less thirst was experienced by every patient.

5. Headache.—A constant symptom in all the cases of *both* typhoid and typhus fevers, and also one of the earliest symptoms in a large majority of both, was fixed in the forehead, rarely the vertex or temples, never the occiput; in some cases it was severe, in others trifling; and it had no definite character; *i. e.*, the patients could rarely give it any descriptive name,—as darting, bursting, &c.

But, in *typhoid* fever, this headache continued until the end of the second, or middle of the third week; while in *typhus*, it disappeared by about the tenth or twelfth day.

6. Expression, Manner, Hue of Face.—In *typhoid* fever, expression much less that of prostration, and more anxious; complexion tolerably clear, and the flush, when present, of a brightish pink colour, limited to one or both cheeks, and often distinctly circumscribed. In *typhus*, on the contrary, complexion thick and muddy,

flush uniform and dusky red. Expression and manner, in the majority of cases, so peculiar as alone to be diagnostic—dull, heavy, oppressed, confused like that of a drunken man just disturbed from sleep. The mind rarely intelligent enough, after the commencement of the second week, to be disturbed as to the final issue; and *this* disease being in itself free from serious organic lesion, all automatic as well as mental expression of anxiety is absent.

7. Eyes and Pupils.—In *typhus*, conjunctivæ *very much* more constantly and intensely injected, which injection probably began during the second week. Pupils abnormally contracted, in eleven out of twenty-five cases; while in *typhoid* fever the pupils are absolutely larger than natural, in the majority of cases.

8. Pulse.—In *typhoid* fever, its frequency varied very greatly in different cases, during the same period of the disease. During the first week, the pulse ranged from 110 to 132 beats per minute; the second week, from 80 to 128; the third week, from 60 to 160; and during the fourth week, from 96 to a rapidity beyond calculation. Its frequency fluctuated also much, from day to day, in the same patient; and, as far as could be ascertained, these variations were independent of increase or diminution in the severity of local complications. Its character was never hard or bounding, occasionally full and soft, usually small and weak. Irregular in two of twenty-two cases. In *typhus*, the pulse ranged in frequency from 108 to 120. In seventeen out of twenty-four cases, it either remained at the rate it had when first observed, or rose till the patient's death. Its character, from the earliest period this disease came under observation, was decidedly soft, gradually becoming weak, then very weak, and in many cases, during the last few days of life, imperceptible. It was generally small, occasionally full, but still retained its extreme softness. Irregular in six of twenty-four cases, or irregular and intermittent.

9. Somnolence, in *typhoid* fever, was present in eight of nine cases, and commenced after the fourteenth day; in *typhus*, this symptom was present in seventeen of eighteen cases, and commenced *before* the end of the second week.

10. "Coma Vigil."—In *typhoid* fever, not present in a single case; in *typhus,* more than one-fifth of the patients experienced it from one to four days immediately preceding their death.

11. Delirium—in *typhoid* fever, commenced *before* the fourteenth day in three only of ten cases; while in *typhus* it commenced before that period in fourteen of fifteen cases; and, as a rule, its character was far *less vivacious* than in the typhoid disease.

12. Retention of Urine, and Involuntary Discharge of Urine and Stools.—Of equal frequency in *both* typhoid and typhus fevers, but it occurred at a *much earlier* period in *typhus.* In both diseases this involuntary discharge of urine and of fæces was simultaneous.

13. Loss of Muscular Power.—In *typhoid* fever, little more than a quarter of the cases kept their beds entirely before the seventh day; in *typhus,* all before that day. Prostration was rarely so extreme in typhoid as in typhus fever. When extreme prostration did occur, it was not in typhoid fever until from the fourteenth to the thirtieth day; while in the large majority of typhus cases it was marked between the ninth and twelfth days.

14. Spasmodic Movements are of nearly equal frequency in *both* diseases, and affect the muscles of the face, arms, or hands. In *typhoid* fever, not a single case of general convulsions; in *typhus,* they may occur.

15. Hearing.—*typhoid* fever, deafness in one-fourth of the cases; *typhus,* deafness in one-fifth.

16. Tongue.—In *typhoid* fever, more frequently moist; when dry, was often red, glazed, and fissured; when brown, its hue was much less deep, and yellowish instead of blackish brown.

The small, dry tongue, with red tip and edges, smooth, pale brownish-yellow fur, fissured, the surface between being deep red, is *differentially* diagnostic of *typhoid* fever. The patient was unable (through exhaustion) to protrude the tongue, in one case only out of twenty; but in *typhus,* was unable to do so in eight cases out of forty—or, in one-fifth of the cases, at some period of this disease.

17. Epistaxis—present in *typhoid* fever in one-third of the cases—was absent in *typhus.*

18. Cough and Physical Chest Signs.—In *both* diseases, cough was nearly equally frequent—viz., in typhoid fever, twelve of twenty-three cases; and in typhus, twenty-one of forty-three. Sonorous râle was heard, more or less, in eleven of the (former) twelve, accompanied, generally, with a little mucous rhonchus; two expectorated a little colourless mucus. Sonorous râle was heard in seven of the twenty-one cases. (After death, intense congestion of the pulmonary texture was found *near* the bases of both lungs, passing occasionally into absolute consolidation.)

19. Abdominal Pain, Tenderness.—Physical signs, diarrhœa and hemorrhage from the bowels.

Typhoid Fever.—Pain in the abdomen, as one of the earliest symptoms, was experienced by nearly one-half the patients. Tenderness of abdomen in three-fourths of the cases where this point was noted. This tenderness was sometimes limited to the right iliac fossa, and although frequently extreme, in some cases it was trifling. Gurgling—generally situated, also, in the right iliac fossa—was present in nearly one-fourth of the cases; the bowels being relaxed in every one of these. The abdomen was full *or* resonant, or full *and* resonant. Its size often extreme; but whether much or little distended, its *shape* was *invariably* the same, and peculiar—viz., *tub-shaped,* never pot-bellied—the convexity being from side to side, and not from above downwards. This characteristic shape was probably due to fiatus, chiefly occupying the colon, ascending, transverse, and descending.

Typhus.—Pain in abdomen, as a first symptom, was experienced by one only in twenty patients. Tenderness in but few cases, and then usually trivial and transient. Gurgling in one case only, and in that at the same time accompanied with considerable diarrhœa. Fulness and resonance were only occasionally present; the peculiar tub-shape never.

In *typhoid* fever, diarrhœa was very common. The evacuations varied in colour from pale brown to almost black; when watery, they were usually yellowish brown, with a sediment of small, solid, yellowish particles. Hemorrhage occurred in one-third of the

cases, and the amount varied from one or two ounces to two or three pints; in colour, from black to bright red; and in consistence, from a reddish watery fluid to that of treacle, and even small clots.

In *typhus*, on the contrary, twenty-two of forty-three patients required, while under observation, from one to four doses of aperient medicine; while in one case only was an opiate administered for diarrhœa. The evacuations were watery in two only of seventeen cases; pultaceous in eight; solid in seven. Of hemorrhage, not a single instance occurred.

20. The eruptions of the typhoid and typhus fevers, respectively, have already been contrasted in every particular.

21. Miliary Vesicles, in typhoid fever, appeared at a variable period in the course of this disease, and were always preceded and accompanied by a warm skin and profuse sweating. These vesicles were present in an equal proportion of the cases of both typhoid and typhus fevers that occurred under forty years of age; but in no case of the latter disease, beyond forty, were they detected.

22. Hygrometric and Thermometric conditions of the Skin.—In *typhoid* fever, the variations of temperature in the same patient were considerable: and in several instances, where this fever was prolonged, they were observed to have no relation to its duration. In *typhus*, these conditions were also variable—a hot and dry skin, or profuse sweating.

23. Sloughing, without pressure, in *typhoid* fever, began at a variable period—at the end of the second or of the fourth week; in *typhus*, sloughing (a frequent event) over the occiput, shoulders, angles of the ribs, trochanters, condyles of the femur, and the malleoli, or over any part exposed to the slightest pressure, began from the thirteenth to the sixteenth day of this disease.

24. Erysipelas, in *typhoid* fever, on the head and face, in seven of twenty-three cases; whereas, in *typhus*, two cases only occurred, was limited to the head and face, in both cases was very severe, and supervened toward the termination of the fever.

Of the above nine cases, seven were females.

These are striking results. They well illustrate the value of the statistical method of investigation. I might proceed in like manner to contrast the anatomical lesions of these two fevers, as disclosed by post-mortem examination. Remarkable differences are found, more particularly ulceration of Peyer's glands, agminate and solitary, in *typhoid* fever; thus accounting for the peculiar *abdominal* symptoms presented during life, and singularly contrasting with the entire absence of any such lesion and corresponding symptoms in typhus. Hence the appropriate name proposed by Dr. William Budd for the former—*intestinal* fever. This typhoid fever appears to expend itself rather on Peyer's intestinal glands, while typhus is directed to the skin. A full and authentic summary of *all* these anatomical lesions concludes Dr. Jenner's valuable papers already referred to.

Relapsing fever is allied to both typhus and typhoid fever, but contrasts with both in its course and symptoms, which are peculiar to itself. This fever is manifested by sudden rigors, headache, very rapid pulse, hot and dry skin, loss of appetite, white tongue, occasional or frequent *vomiting* of *brown* or *black* fluid, the bowels being regular; by the *absence* of abnormal physical *abdominal signs*, and, in severe cases, by *jaundice; profuse sweating* on about the *seventh* day, followed by apparent restoration to health; but on the fifth to the eighth day after this apparent convalescence, *relapse* and repetition of the original symptoms, with greater or less severity, again terminating in sweating, and then permanent convalescence. In comparatively few cases is there a fatal issue.*

In the family group of 'eruptive fevers,' the next four may be conveniently associated together—namely, Measles, Scarlet fever, Small-pox, and Chicken-pox.

The diagnosis of these diseases, by virtue of their specific skin-eruptions and affections of the naso-pulmonary mucous membrane, with the general phenomena of the concurrent fevers, being established in the first instance; the 'blood-origin,' of *all* eruptive

* For further information, see References in Science and Practice of Medicine, W. Aitken, 1858, p. 167.

fevers, will further illustrate the Etiological Principle advanced in this chapter.

Their Prevention will then be considered.

The diagnosis of the 'rubeoloid eruption,' or that of Measles, is not difficult. Minute red points, slightly prominent, first appear on the face and neck. These pimples (papulæ) of measles are not unlike those of small-pox on the first day of its eruption, but subsequently they contrast remarkably; the latter becoming more and more prominent, then vesicular, and eventually pustular; the former assuming the character of flattened blotches. The rubeoloid pimples, therefore, soon become confluent, they coalesce and form crescentic patches of a dull raspberry colour, which are brighter, however, on parts exposed. This eruption thus contrasts with that of scarlet fever, which usually consists of innumerable scarlet points grouped together into patches, but having no definite shape—of the bright crimson hue of a boiled lobster, and brightest on parts not exposed to the air.

The papulæ of measles, therefore, contrast with those of small-pox in their *course*, and with those of scarlet fever in the *shape* and *colour* of the groups, into which they become aggregated.

The rubeoloid eruption having appeared on the face and neck, then visits the trunk, arms, and legs; its distribution in this respect resembling that of both small-pox and scarlet fever; but the eruption of measles spreads over the body in about three days, while that of scarlet fever does so in about twenty-four hours only, and that of small-pox, more leisurely than the former, in about five days.

Measles and scarlet fever end alike by desquamation of the cuticle, small-pox by incrustation of the pus and desquamation of scabs; but the eruptions of the two former are consummated in about four days a-piece—rather less, then decline and disappear in about seven days; while that of small-pox ripens only in about eight days, and finishes its course in about four days more—say, in twelve days altogether.

In this description of the rubeoloid eruption, and its compa-

rrison with the eruptions of scarlet fever and small-pox, I have purposely omitted to notice that of chicken-pox, which being *vesicular* from its first appearance, bears no resemblance to that of measles, and therefore cannot be mistaken for it.

But if the eruption of measles be *characteristic* of this disease, its most important, because most perilous, manifestation, is a 'catarrhal inflammation of the naso-pulmonary mucous membrane.'

This is denoted by swollen eyelids, with red and watery eyes overflowing with tears. Some intolerance of light may be experienced. The nasal mucous membrane being inflamed, sneezing is another symptom, recurring in some cases every few minutes until the child is quite exhausted. An excoriating discharge distils from the nostrils, the fauces are seen on inspection to be redder than natural, and there is some soreness of the throat. The laryngeal and tracheal mucous membrane having shared the inflammatory hyperæmia, hoarseness is not an unfrequent symptom, and a loud, hollow, dry cough is perhaps the most characteristic feature of this catarrhal affection.

In truth — as Dr. Gregory well expresses it—the mucous membranes of the head and chest receive the first impetus of the poison of measles. Bearing in mind, however, this fact, and the priority also, usually speaking, of the catarrhal affection, instances are alleged by some authors of the rubeoloid skin eruption being present, without any implication of the aforesaid mucous membranes, at any period of the disease—"rubeola sine catarrho." The participation of the rubeoloid poison would seem, therefore, to be neither a constant nor prior event, in all cases. But these authors—Willan, for example—acknowledge that persons who have had this supposed variety of measles enjoy no immunity against its recurrence. Perhaps, therefore, the first disease was not *true* measles. Gregory believes these alleged instances to have been febrile lichen instead.

Scarlet fever, or scarlatina, is identified by its peculiar 'skin-eruption' and 'sore throat.' There are varieties, however, of this disease shading into each other, just as there are shades of dif-

ference only between varieties of the same species of flower or fruit. Scarlatina *mitis* (simplex) is manifested by a scarlet efflorescence, without any, or scarcely any, sore throat. Scarlatina *anginosa*, the most common variety, is declared by a vivid rash, with decided sore throat and inflammation of the mucous membrane of the larynx, mouth and nose. Scarlatina *maligna* (cynanche maligna, angina maligna putrida) is exhibited by the throat alone.

These varieties, therefore, are expressed by reference to the skin eruption, and the portion of mucous membrane involved. Each of these features requires a few more descriptive remarks, beyond the comparison already drawn, in speaking of measles.

The 'eruption'—consisting of small red points, scarcely elevated, patchiform, but without any definite shape, disappearing on pressure—is distributed all over the body, giving a diffused tint like that of a boiled lobster; and this overshadowing hue travels from the face and neck downwards; but in simple or mild scarlatina, it is first perceived on the trunk, arms, and thighs, without appearing in many instances on the face. A partial eruption only, as on the thighs, appears in some instances of scarlatina anginosa, or if the eruption be more general, it may recede for a few hours and then recur.

More or less *swelling*, doubtless from effusion of serum into the subcutaneous cellular texture, accompanies this skin eruption, whereby the fingers feel swollen and stiff. Œdema is more palpable in scarlatina anginosa, and in this variety the subcellular texture also of the neck is apt to swell, and the parotid glands to enlarge, so that the jaws are opened with some difficulty. Miliary vesicles may be interspersed with the rash (scarlatina varioloides). According to Dr. Watson's experience, these vesicles are most thickly set on the thorax, and on the front and sides of the neck. Their liquid contents are soon absorbed; the enclosing cuticle shrivels up, turns white, and effloresces in a thick white scurf, the part from which it separates looking at first sight as if it had been powdered.

A general desquamation of the cuticle ensues as the eruption disappears. This desquamation takes the form of a mealy or

branny powder over the whole surface of the body, while a glove, or slipper, of cuticle comes away at once from either hand or foot.

Respecting the 'sore throat,' uneasiness is experienced in deglutition from the onset of scarlet fever, and inspection of the fauces discloses a bright red colour, with some degree of swelling, extending over the tonsils and palate, which soon become overlaid with ash-grey films of lymph; thus contrasting with common tonsillitis. These films have been mistaken for sloughing ulcers, but ulceration is a rare event in the most severe cases of scarlatina *anginosa*. It is a specific inflammation, and not limited to the fauces; it creeps along the mucous membrane of the tongue, by which its papillæ became enlarged and prominent; so that when the cream-white fur which had accumulated clears away, the organ presents a characteristic appearance — "the strawberry tongue." Along the posterior nares, also, this inflammation extends, and an acrid fluid is discharged from the nostrils, resulting in loss of smell. A similar discharge from the ears— pending, perchance, destruction of the ossicula — ends in permanent deafness. One or both eyes may be destroyed; even the cellular texture of the orbit sloughed in one case under Dr. Gregory's observation. In rare cases the larynx shares the inflammation of the fauces; but all these extensions along neighbouring offsets of mucous membrane, are of later occurrence than either 'the sore throat' or 'the skin eruption.' *They* are the primary local manifestations of scarlet fever.

Scarlatina *maligna* is known by its peculiar sore throat alone, without any skin eruption, or any worthy of notice. Fortunately this is not the common variety of scarlatina. The fauces become swollen almost to suffocation, and of a livid red colour. Ulceration quickly succeeds, ash-coloured sloughs are seen on the tonsils, and the ravages of gangrene are often extensive. The voice acquires a hoarse and hollow tone, and respiration is accompanied with a strangling noise; then come puffs of putrid breath, and a sanious fluid bubbles forth abundantly from the nostrils, excoriating the lips and cheeks.

The skin eruption, if present, is livid and scanty. It is apt to appear and then recede; occasionally, purple petechiæ are interspersed. In fact, the typical rash of scarlatina may be absent in the worst cases; the skin eruption, therefore, is conspicuous chiefly by its absence.

Small-pox, or variola, is another exanthematous fever, characterized by a peculiar 'skin eruption,' and somewhat by the 'sore throat' which it exhibits.

The varioloid eruption begins in the form of small pimples scattered over the face and neck,—on which parts, in the great majority of instances, they first appear. These pimples feel something like shot in the skin. After five days or so have elapsed, a little vesicle, containing whey-coloured fluid, arises on the summit of each pimple. The pimples have become vesicular on the face and neck, and by this time other pimples have appeared successively all over the body; this eruption spreading from above downwards with tolerable regularity. During the next two days, those pimples which are now vesicular, undergo a remarkable change. The vesicles enlarge in breadth only, and a cup-like depression is seen in the middle of each, or most of them; but by the eighth day these cryptiform vesicles are matured into plump spheroidal pustules. The whole crop of pimples, with few exceptions, follow their example, and the skin is everywhere studded with pustules of a honeycomb colour. Such at least is the appearance of *discrete* small-pox; the pustules are isolated. But they are often arranged in groups of three or five, forming crescents or circles, an arrangement distinctly seen when the eruption is not too copious; and this arrangement contrasts with chicken-pox, which differs also in more essential particulars, to which I shall presently advert.

The vesicles or pustules of small-pox are, from their first formation, severally encircled by an inflamed margin of skin, swollen and painfully tense. Thus the fingers are stretched and shining, while the eyelids eventually puff up like bladders, closing the eyes, and the swollen face assumes an aspect unknown by

the patient's most intimate friends. As the pustules ripen, this surrounding inflammation acquires a damask-rose hue, which Sydenham states* was more exactly the colour both of the pustules and their interspaces, in the *mildest* forms of scarlet fever, as *he* witnessed it. The pustules, he adds, of the face, become rougher and yellower, those of the hands and body generally being smoother and whiter.

At this stage of the eruption—that of mature pustulation—I pause for a moment to describe the *structure* of the variolous pimple and pustule,—a subject, however, of pathological interest rather than of diagnostic importance. It was first investigated by Cotugno in Italy, followed up by John Hunter, Dr. Adams, Bousquet, Gendrin, Judd, Dr. Petzholdt, &c., and embodied by Gregory in his valuable treatise,† from which I extract the following particulars:—

"Inflammation begins in the true skin. From a central point (stigma-phlyctidium) it extends by radiation on the surface, penetrating to a greater or less depth in different cases. Beneath the epidermis, and constituting the greater portion of this central spot, is found a substance or disc of the consistence of pulp or thick mucus, which apparently is no part of the skin altered, but a new specific product. It is the variolous slough of Hunter and Adams. When suppuration is completed, this substance is swollen and moist like a sponge. The floor of each central spot presents the papillated structure of the skin, elevated and marked with fissures.

"The 'vesicle' itself is multilocular, like an orange, being divided into numerous cells, twelve or more in number. A filament of cellular tissue binds down the central portion of cuticle to the lower surface of the phlyctidium. The fluids—lymph and pus—which, in different stages, distend its cells, destroy at length the filamentous attachment of the stigma to the cuticle, and that

* Works. Trans. for Sydenham Society, 1850, by R. G. Latham, vol. ii., p. 251.

† Lectures on the Eruptive Fevers, 1843.

which was first a depressed or umbilicated vesicle becomes at last an acuminated pustule."

This description, probably accurate in so far as it goes, relates only to the *central* vesicle of the small-pox pustule; it does not comprise the structure of the *whole* pustule. Around this central vesicle, which is multilocular, and contains whitish lymph, runs a circular trough, containing yellow pus. Either receptacle can be emptied by puncture without disturbing the other; and the vesicle is said to contain the purest portion of the variolous poison.

Having advanced to maturity by about the *eighth* day, the yellow hemispherical pustules are now the size of a large pea, the swelling and inflammation of the face subside, and about the eleventh day a dark spot appears on the summit of each pustule; shortly after, they spontaneously burst, some of their yellow matter exudes and forms a crust, while the pustules themselves shrivel up, retaining perchance a portion of matter, which becomes thick and hard. After four or five days more the crusts and withered pustules underneath them alike fall off, leaving the subjacent skin of a *clarety* hue; and these remarkable stains wear off only in three or four months. In severe cases inflammation of the true skin does not cease with suppuration (maturation); portions of skin actually slough, and leave *pits*, when the pea-like pustules drop off, the claret tint remaining also for a time. But this *pitting* of the skin is permanent, or at least wears off only in the course of many years. Such is the career of the varioloid eruption when uninterrupted and undisturbed. But it may be arrested. Thus, some of the pustules, especially on the extremities, wither, without bursting; others may be broken prematurely by the patient's scratching them, and as prematurely become encrusted. Lastly, maturation and scabbing are delayed in bad cases until the ninth or tenth day.

Confluent small-pox presents many irregularities as compared with the typical form of eruption. Its *differential* characters are as follows:—

The pimples appear at a more early period of the fever, and

are more numerous; both these circumstances being indicative of the severity of the disease, are of themselves alone important points of difference. Not unfrequently, in the first instance, there is general inflammation of the skin, resembling that of measles, scarlet fever, or erysipelas; for either of which, therefore, the incipient small-pox eruption is liable to be mistaken at first. Any doubt, however, is soon dispelled by the pimples becoming vesicular. As the eruption advances, the pustules do not so much rise to any notable height as *run together*, and form irregular-shaped islands of puriform matter, having a whitish or brownish tint rather than a yellow colour. This confluent form of pustulation occurs more especially on the face, which, in some instances, is entirely overspread with a large sheet of matter.

Maturation, such as it is, may be postponed until the tenth day or so, and separation of the scabs delayed until the twentieth day. Then irregular crusts, some large, some small, drop off. The face sheds a regular mask in severe cases.

Other modifications of the varioloid eruption occur, besides those ordinarily known as discrete and confluent small-pox, with their differently shaped and shaded pustules. Blood, rather than true matter, collects within the pustules in the most terrible form of this disease,—happily rare now-a-days; but described by Sydenham as *variolæ nigræ*, and afterwards named more correctly by Mead, *bloody* small-pox.

Passing from the skin to its subcellular tissue, this texture also supplies a ground of early and exact (differential) diagnosis, scarcely inferior to that supplied by the skin-eruption.

I have already mentioned the *swelling* and tension of the *skin* in distinct small-pox, and that infiltration of the subjacent cellular texture supervenes. But in *distinct* small-pox the *skin* alone is the *seat* of inflammation; whereas in the *confluent*, it dips deeper into the *subcutaneous cellular texture also*, sometimes extensively, sometimes partially—*e.g.* that of the scalp very commonly. Diffuse suppuration follows, or a succession of small and most troublesome abscesses. In this way the neck is peculiarly

liable to great turgescence, accompanied with sore throat and profuse salivation. Occasionally the tongue becomes involved,—an event which few, if any, survive.

More diagnostic even than *this* 'sore throat' is that *specific* form of inflammation which arises by extension of the pustular eruption to the mucous membrane of the fauces. It occurs in a large proportion of those who have confluent small-pox. And not only is the throat thus affected; the nose, mouth, larynx, and trachea are so also; indeed, the mucous membrane of all those parts to which the air has access are said to be subject thereto. It was alleged, moreover, by Cotunnius,* that desiccation of the mucous membrane about to be invaded was an essential condition; and that the atmospheric air predisposed by drying those parts which are exposed. For example, pustular eruption comes out more abundantly on the face and hands; pustules appear on hemorrhoidal tumours only when they project beyond the margin of the anus; that portion alone of the glans penis is thus affected which is unprotected by the prepuce; and they do not form on the inner surface of the eyelids, except in cases of eversion. This last-chosen instance is erroneous, for in confluent small-pox especially, pustules arise on the palpebral conjunctivæ; and their evolution on the fœtus in utero is quite incompatible with the theory advanced.

Certain characteristic symptoms announce this mucous complication. Varioloid pustules, more often confluent than distinct, appear on the tongue, inner aspect of the cheeks, palate, and velum. Hoarseness and alteration of the voice indicate—in Dr. Gregory's experience—that this condition extends to the mucous membrane of the larynx and trachea. Great pain in swallowing, with cough and dyspnœa, in bad cases, are further evidence of like import. The cough is at first dry and tearing, but as the disease progresses, expectoration relieves it; and about the eighth day, a copious viscid secretion exudes from all these parts. Far more serious results accrue from this mucous complication than any

* De Sedibus Variolarum.

local mischief it occasions. Both the quality and circulation of the blood are impaired by the persistent dyspnœa. After eight days or so have elapsed, the crimson areolæ around the vesicles are faded, while those on the extremities never acquire any marginal inflammation—by which alone the surface can be cicatrized. On the trunk, this inflammation is livid or claret-coloured. The vesicles do not acuminate. They are flat, and appear much the same as after death. Sometimes the superficial inflammation is erysipelatous rather than phlegmonous, and large blebs of ichorous fluid arise here and there. In twenty-four hours, low muttering delirium supervenes. But the circulation is feeble, as well as the blood being vitiated; the tongue swells and has a purple hue, the extremities grow cold, and yet colder, until finally benumbed with the coldness of death.

Chicken-pox, or varicella, is a disorder about which there has been, and still exists, considerable difference of opinion as to its non-identity or identity with small-pox of a minor degree than that of the ordinary type. Although in itself a very trifling disorder, its early and exact diagnosis is very important; because, if only modified small-pox, the same preventive precautions are indicated as for the ordinary type of this serious disease, which it might *assume* if communicated to another person. If *non*-identical therewith, then at least we shall know well when *these* precautions are unnecessary, and that the members of a family or a school run no risk of the greater evil emanating from the lesser in the person of one of their companions. I shall have occasion to advert again to the importance of this question of identity in relation to prevention.

What, then, are the leading characters of chicken-pox? I gladly avail myself of Dr. Gregory's original observations, of which the following summary will prove sufficient:—"Varicella is a slight disorder, the offspring of a specific miasm, which, without initiatory fever, throws out an eruption of confluent vesicles, which maturate in three days, and desiccate into granular scabs that speedily fall off. Little or no fever accompanies the maturative

stage, and no secondary fever follows. This disorder chiefly prevails among children, and occurs but once in life."

Already do the terms of this definition suggest the *non*-identity of varicella with small-pox. For, as a tree is known by its fruit, so are eruptive fevers specially known by their several eruptions; and the *primarily* vesicular eruption of varicella is unlike that of small-pox in any, even its mildest form. Let us then examine the fruit of varicella more closely.

Its 'vesicles' are simple elevations of the cuticle, or minute blisters, presenting an appearance as if the skin had been exposed to a shower of boiling water. The parts chiefly visited are the back and scalp, sparing the face, a part that never escapes in small-pox. These vesicles vary in shape somewhat, are the size of a split pea, and encircled with a slightly red halo. They contain a clear lymph, which, if the vesicles remain unbroken for twenty-four hours, acquires a slight opacity. A tingling itchiness also disposes the child to scratch the part affected, and if this desire be not restrained, a degree of superficial inflammation succeeds, sufficient to convert the clear lymph into an imperfect pus. After two or three days, the vesicles shrivel up, very small scabs appear, and, as the lymph is not mucilaginous, these are granular. Fresh crops of vesicles recur in succession for two or three days, so that about six days elapse ere the complaint finishes its course.

All this is unlike small-pox. The structure of the varicelloid vesicle bears no relation to that of variola. Here, no umbilication, no central depression, no multilocular structure, no slough, but simple elevations of cuticle. Then, again, they have no regular arrangement—no grouping into threes and fives, no crescentic or circular figures. Lastly, everything in varicella is hurried forward—the incubation, the eruption, the desiccation; and not the slightest *pitting* commemorates this transient and trivial exanthem, although in a few instances shallow cicatrices have been noticed. No throat affection of any kind complicates; *i.e.*, the mucous membrane of the fauces is not involved. Above all, varicella is caught indiscriminately by children (almost exclu-

sively) who have, and by those who have not, been vaccinated; nor is its course altered in the slightest degree by having undergone this substitute for small-pox. Therefore, *à fortiori*, the two diseases in question are *non*-identical.

But if chicken-pox be a disorder distinct from typical variola, does this latter ever approach the former? Is there a *modified* small-pox which resembles varicella, and for which it is liable to be mistaken? There is. Variola varicelloides, as this modification is named, is an approach towards true varicella, yet without overstepping the line of demarcation between them. The respective characters of the two eruptions at their first appearance—one vesicular, the other papular—is one point of distinction; and when the hard papules of small-pox have become vesicular, the peculiar structure of its vesicles is another; subsequently there are those shades of difference which have just been enumerated. But with all these points of distinction between the eruptions of true and feigned varicella, it must be confessed that their diagnosis is not always obvious. Nevertheless the non-identity of these two diseases is obvious. What says 'vaccination'? That they who have been placed under its protection enjoy no immunity whatever from chicken-pox, but are privileged to hold intercourse with small-pox in *any* shape without scarcely any risk of infection. This guarantee of non-identity at least, if it does not aid our diagnosis, bears immediately on the prevention of infection in its worst sense. For they who are *unprotected* by vaccination, as well as they who are thus fortified, may equally associate where chicken-pox is prevalent, without any fear whatever of small-pox in any shape being generated. A distinct individuality, albeit invisible, proper to each of these two diseases, estranges both; so that trivial and transient varicella may be propagated freely, and yet will never give birth to that terrible tyrant small-pox. This is anomalous, considering their close relationship. But they are members of the same family in likeness only, not in blood.

There is yet another member of this family, about whose relationship opinions are divided. Erysipelas is an eruptive fever cer-

tainly, yet its origin by infection is disputed, and indeed open to doubt.

This doubt, and this disputation, seem to have arisen from one oversight—that of identifying erysipelas, a peculiar inflammation of the skin (and subcellular texture) of the *head* and *face only*, with *erysipelatous inflammation* of the skin (and subcellular texture) of any other part of the body. The former is decidedly infectious; the latter, apart from co-operating causes, *less* decidedly so.

Commencing then with the integuments of the head and face, (true) erysipelas exhibits the following characteristic appearances:—On the nose, either cheek, the margin of either ear, or sometimes on one of the temples, a slight blush of redness becomes visible, accompanied with stiffness rather than swelling of the *skin*, which has lost only its wonted suppleness, and acquired a shiny roseate hue. A tingling, burning sensation also, rather than pain, is experienced, and hence the popular name of this inflammation—St. Anthony's Fire.

The redness of Erysipelas assumes different tints; in some cases being more scarlet, in others rather purple; but of whatever tint it may be, it disappears entirely on pressure, and returns immediately the finger is withdrawn—so free and persistent is the determination of blood. The tension also of the skin is readily perceived on passing the finger from the sound to the inflamed part. An abrupt margin defines this redness and this stiffness; both are circumscribed by an irregular line.

The inflammation, thus mapped out, spreads continuously: erysipelas is, par excellence, a 'serpiginous' skin affection; and while it diverges, creeping like water spilt on an impervious surface, the skin becomes swollen, some serum is effused into the *subcellular* texture also, and *this* swelling is *soft* and *diffused*. Nature makes no adequate reparative effort to limit and circumscribe the serum with lymph. Serous effusion, therefore, proceeds, and the swelling increases—so much so as to close the eyelids, distend the cheeks, disfigure the features, and, at length,

obliterate all traces of personal identification. "What great events from little causes spring:" that trifling red nose, and now this defaced visage. The turgid textures are tense and painful, although still a burning pain.

At this stage of the inflammation its course is sometimes arrested, the redness fades, the swelling subsides; and this termination by 'resolution' occurs with or without shedding of the cuticle. In many instances, however, the issue is less speedy. Serous effusion having continued for a period varying from twelve to thirty-six hours, elevates the cuticle into vesicles, or larger blebs, exactly like those which follow a burn or scald. These semi-transparent and yellowish, or sometimes livid blisters, soon burst, and discharging their contents—serum, pure or bloody—subside into thin incrustations. When, in the course of a few days, these peel off, they disclose the subjacent skin either in a sound state or beset with superficial ulcerations. In some instances the true skin is less spared. It has a reddish-brown or livid hue; and even gangrene of the *skin* succeeds, announced by great tension, heat, and acute pain. But this issue of erysipelas is rare, and generally fatal.

Such are the origin, course, and terminations of *simple* erysipelas,—an inflammation akin to erythema, and contrasting with the *phlegmonous* variety, in which the skin and subcellular texture are *both* the *seat* of inflammation; the latter tissue being more especially, but secondarily, engaged. The roseate hue, if it should appear in the first instance, is soon exchanged for a brownish or livid tint, which mottles the skin. Simultaneously, the subcellular texture is gorged with serum, and presents a considerable swelling, which, however, readily pits on pressure with the finger. This *œdematous* swelling, of *considerable size,* and this purple *shade* of *colour* exhibited by the skin, contrast with the characters of simple erysipelas as it first appears. The burning pain also is more severe, and perhaps accompanied with throbbing. Otherwise the simple and phlegmonous varieties have points of resemblance. In both, the redness presents a defined margin;

in both, the inflammation extends itself continuously by creeping over the surface. But then again, in phlegmonous erysipelas, engorgement of the subcellular texture advances and deepens with alarming rapidity; and although this serous infiltration is itself diffused, the stuffed cushion of cellular tissue feels *brawny*. A few small vesicles only represent the *overflow* of serum under the cuticle; they do not reveal the perilous state of affairs *beneath* the *skin*. Burrowing suppuration and rapid sloughing threaten; these dangers *not* being attended with *increased* tension, swelling, and pointing, as in phlegmon; on the contrary, rather with diminished tension, subsidence, and flaccidity. Such is the experience of Lawrence,* and of another original observer of this disease. At that period, writes Dupuytren,† when phlyctenæ have formed, and the cellular texture becomes thickened and indurated, the symptoms appear for two, three, or four days to be stationary; and an inexperienced surgeon is even led to hope for the resolution of the inflammation, while the danger is really urgent and suppuration already exists. This, then, is the critical period of phlegmonous erysipelas. If unchecked, the conclusion of its career is told in a few words.

Very soon after this period of deceitful calm comes an outburst. A more livid hue overshadows the integuments; the skin melts away in patches, accompanied with a discharge of bloody sanious fluid, and the exposure of white sloughs of cellular tissue, portions of which bulge here and there through apertures in the integument. Sometimes this texture perishes extensively, but if partially spared, large sloughs of cellular tissue, resembling masses of soddened tow, are eventually detached; while the adjoining cellular texture is drilled with small abscesses, or a profuse suppuration burrows wherever fluid can find its way—between muscles, and possibly into a neighbouring joint. Thus all parts around become involved in the ravages of phlegmonous erysipelas. In one fatal case, under Dupuytren's observation, the whole leg was laid

---

* Med.-Chir. Trans., vol. xiv. † Clin. Chir., t. ii., p. 311.

bare of skin and cellular tissue, exposing the tibia and patella. After prolonged suppuration and sloughing, those textures which do escape—the muscles, fasciæ, tendons, and bones—are so agglutinated together as to seriously and permanently impair their uses in the animal economy.

It will be readily imagined that erysipelas may spread from the scalp and face to the neck, thence to the thorax, and occasionally extend as far as the extremities; or it may begin in some other part than the head and face—such as the leg, sometimes the trunk.

In whatever part of the body erysipelas first shows itself, its characters and course are similar; but while fading in that portion of the skin where it first appeared, it travels to the neighbouring skin. The various stages of this inflammation, therefore, usually coexist in different parts of the skin; the portion last affected being red and swollen, another part vesicated, while others are undergoing desquamation.

It seems unnecessary to add a third variety of erysipelas—the "œdematous" of some authors; for the phlegmonous variety is itself œdematous in one stage of its career. In both, a diffused infiltration of the subcutaneous cellular texture with serum is conspicuous, and the pitting of this œdema on pressure with the finger its most distinctive character.

There are, however, certain particulars in which phlegmonous *inflammation* contrasts with phlegmonous erysipelas, and which determine their diagnosis—a question of much importance.

Circumscribed and limited infiltration of the (subcutaneous) cellular texture with coagulating lymph, is the essential pathological condition peculiar to phlegmon. It is an inflammation of the *cellular texture primarily*, the skin being only secondarily involved; whereas phlegmonous erysipelas selects, first, the skin, and then involves the subcellular tissue. *Coagulating lymph*, rather than serum, is effused in phlegmon; the consequent swelling is therefore *circumscribed* and *limited*, instead of being diffused

and wide-spreading; it is also *brawny in the first instance,* that of phlegmonous erysipelas only becomes so gradually; lastly, respecting the issue of these two species of inflammation—that of erysipelas is *prone* to slough, while that of phlegmon is liable only to this termination.

One word about a disputed matter: the liability of 'mucous membranes,' as well as skin, to erysipelatous inflammation. This question is answered affirmatively by Stevenson, who gives a series of twenty-one cases,* and subsequently by Arnott in an elaborate paper.† The former describes an affection of the throat, characterized by a red or purplish blush of the velum pendulum and uvula, with very little tumefaction, but considerable pain in swallowing. Excoriation of the inflamed surface frequently occurs, and superficial ulceration.

This inflammation is preceded by febrile symptoms, even in mild cases, and may occur before or after the skin affection; it may, indeed, constitute the whole attack of erysipelas, without that of the skin supervening. Other men of observation—Lawrence, for instance, with whom I am inclined to concur—do not acknowledge erysipelatous inflammation of any mucous membrane. But this is still an open question.

Pursuing the diagnosis of Eruptive fevers, I might conclude with that of Plague. Now-a-days, however, having retreated far to the rear of these diseases, this, once foremost in the van, scarcely concerns us, in this country at least. Verily, "Time by the hour tells off the roughest day," and we may contemplate, not without awe relieved by gratitude, that period of England's history when hearses paraded the desolated streets of the metropolis, and amid the lurid glare of torchlight arose the appalling cry—"Bring forth your dead." For Plague counts by thousands. In 1665, the "great plague," then raging in London, slew 7165 persons in one

---

* Cases illustrating the contagious nature of erysipelas, and its connexion with a severe affection of the throat. Edin. Med.-Chir. Trans., vol. ii., 1826.

† Lond. Med. and Physical Journ., vol. lvii., 1827.

week only; while, in one year, no fewer than 68,586 died in the city and its suburbs alone; this mortality being more fearful when compared with the far smaller population of that period. Happily, these returns are now matters of historical interest, rather than of personal concern. "Sufficient for the day is the evil thereof." I need only glance at the pathology and diagnosis of that evil, which may never return; and if it should, would *then* have to be investigated, without prejudice, as a *new* disease.

A commission of inquiry, appointed at Cairo, of which Clot-Bey was the representative, found that commonly "plague is preceded by *prodromes*, which last a greater or less length of time. These precursory symptoms are lassitude, loss of strength, general uneasiness, and mental anxiety, soon succeeded by shivering, headache, vertigo, and vomiting. Then appear the different local and general phenomena; and among them, bubo, carbuncles, and petechiæ, preceded or followed by coma or delirium, too often terminating in death." Analytical investigation of many cases reduced this disease to three *degrees:* the first degree being a slight fever, without delirium or buboes; the second—fever, delirium, and buboes; the third—fever, high delirium, buboes, carbuncles, and petechiæ. For further information the following authorities* may be consulted with advantage.

In concluding the diagnosis of 'eruptive fevers,' as determined *mainly* by their *anatomical* characters, let me here advert to the Principles which have been inculcated.

Omitting the two Principles—one fundamental in Etiology; the other, Preventive, and therefore eminently Conservative—which, hand in hand, have been specially advanced; four more

---

* Plague at Malta (1813). Faulkner.—Plague in Aleppo. Russell.—Medical Sketches. M'Grigor. — Plague among French Army in Egypt. Assalini. — Histoire Médicale de l'Armée d'Orient. Desgenettes.—De la Peste Orientale, d'après les Matériaux recueillies à Alexandria, au Caire, à Smyrne, et à Constantinople, pendant les Années 1833 à 1838. A. F. Bulard.—De la Peste observée en Egypte. A. B. Clot-Bey, 1840.—A Treatise on the Plague, &c., and on Quarantine, 1846. A. White.—Select Medical Bibliography. J. Forbes.

have been illustrated. 'Pathologico-Anatomical' diagnosis has been reinforced. The nature and importance of 'concurring' evidence have been demonstrated. The inferiority of 'Functional' symptoms, in aid of diagnosis, has been corroborated. But that the 'results of statistics' applied to these as well as to Pathologico-anatomical data, may elucidate positive grounds of distinction between diseases apparently identical.

Resuming, then, the diagnosis of eruptive fevers, how far does the functional disturbance, Fever, attending each kind, contribute any concurring evidence whereby to distinguish them?

The fever of each exanthem is *initiatory* to, as well as concomitant with, the specific eruption. So far, therefore, it contributes data for a *more early* diagnosis than that furnished by the eruption; but are there any sufficiently marked characters in the constituent elements of these functional disturbances to render them subservient to the most *exact* diagnosis?

We have already seen how far the results of statistics have determined this question on behalf of typhus and typhoid 'fevers.' Unfortunately, no such body of evidence on this point as that which exists respecting these two has yet been collected on behalf of other eruptive fevers. But the results of a short analysis, and fair comparison of their phenomena, tend to show that the fevers of measles, scarlatina, small-pox (chicken-pox), and erysipelas are not subservient to the most exact diagnosis of these exanthemata.

Every exanthematous fever runs its course in four stages: the initiatory or incubative (period of incubation or brooding), the eruptive stage, the maturative, and secondary fever; this latter signifying that revival of febrile symptoms which occurs when the specific fever ought normally to subside. These phases of exanthematous fever are *tolerably* defined in Nature, and are so far convenient for the purposes of exact description. But they severally represent an association of functional disturbances referrible either to the nervous system, or to the sanguiferous and secretory systems; and *these* phenomena, in the successive stages

of each eruptive fever in question, are not sufficiently unlike to be distinguished.

Thus, in common with typhus and typhoid fever, exanthematous fever of any variety denotes certain morbid phenomena referrible to the nervous system—namely, disturbances of the functions of voluntary power, sensation, and thought. These disturbances are expressed by the general term, 'exhaustion,' or prostration, and in an extreme degree by 'febrile oppression.' Therefore lassitude, weariness, restlessness, wandering pains, especially in the back and limbs, thirst, loss of appetite, nausea or vomiting, headache, disturbing dreams, and even delirium, are represented by the expression 'exanthematous fever;' but they are rather initiatory and *premonitory* symptoms. The fever itself is usually announced by 'rigors' in some degree. Shivering, more or less violent, is soon succeeded by reaction, more or less vigorous. The sanguiferous system responds to this depression, and the fever now runs its course as an effort of Nature to eliminate some 'blood-poison' from the body. I here *assume* the presence and operation of such poison, of which this fever is the exponent. And the poison being different in each exanthem, the fever also exhibits differences. Yet these are scarcely appreciable. The pulse becomes rapid in all exanthems. In some, moreover, it becomes feeble. Exanthematous fever is either of an inflammatory or typhoid type—*i.e.*, there is a *malignant* variety of measles, a *malignant* scarlatina, a malignant, *confluent*, and *bloody* small-pox, malignant *phlegmonous* erysipelas.

With such general features of resemblance, are there any points of difference between these fevers, whereby to determine their earliest and most exact diagnosis?

The symptoms of catarrh—sneezing, lachrymation, and a peculiar dry, hollow cough—are *early* symptoms of measles; but then, excepting perhaps this cough, they may announce *only* catarrh. On the other hand, measles without catarrh would seem to be not measles at all.

Sore throat is *most characteristic* of scarlet fever, and in severe

malignant cases is the most prominent symptom; but can *this* be distinguished with certainty from ordinary tonsillitis?

Vomiting and pain in the back and loins are *early* symptoms of small-pox. Cerebral symptoms predominate in some cases. Intense headache, delirium, stupor, have thus far a significant character; and I think it was Sydenham who first noticed the fact, that an epileptic fit in children over-night is very often followed by small-pox the next morning. None of these symptoms, however, are *peculiar* to this disease.

Erysipelas is not, in my experience, announced by any *peculiar* functional disturbances. Gregory enumerates some that are usually present "when this fever is breeding, whether in a hospital or private house, whether following a wound or arising from some inward heat of the blood, whether ultimately to develope itself on the face or extremities;" but he adds, "there is nothing characteristic of approaching erysipelas, as contra-distinguished from any other kind of eruptive ailment." "The pulse is peculiarly quick and sharp at the onset of this fever." Erysipelas of the face is said to be *preceded* and accompanied by a fever, of which "a peculiar affection of the sensorium" is the prominent symptom; and the initiatory fever of idiopathic erysipelas, in every case, is said to be accompanied by "inflammation of the fauces." Both these statements are affirmed by Arnott,* and the latter—sore throat—is associated, in Dr. Watson's experience,† with erysipelas of the head and face.

Granting that the phenomena of (exanthematous) fever afford equivocal evidence for the most exact (differential) diagnosis of the aforesaid exanthems, yet the *initiatory* stage of such fever—*i.e.*, the period of incubation prior to the specific eruption—is peculiar to each by virtue of its *duration;* for, while differing in this particular according to the kind of exanthem about to appear, the period itself is, nevertheless, invariably constant as regards each

---

* Op. cit.

† Principles and Practice of Physic, 1857, vol. ii., p. 915.

kind, with few exceptional cases. The particular period of incubation is, therefore, so far *characteristic* of the *species*. Measles puts forth its eruption on the fourth day of the incubative fever. Scarlet fever does so on the second day. Small-pox, on the third day. Erysipelas, in a period varying from twenty-four to sixty hours.

It is also here worthy of notice that small-pox, unlike measles, is most intense in proportion as its eruption appears earlier than usual and is copious. But the fever continues and rather increases with the eruption of measles, while with that of small-pox it ceases entirely. This at least is true of *distinct* variola.

Having analysed the evidence whereby the earliest and most exact diagnosis of eruptive fevers is determined, we are now in a position to identify each of them in order to discuss the question of their 'blood-origin,' and this in order to supply rationally appropriate measures for the Prevention of these Fevers. Their blood-origin, if proved, will illustrate the General Etiological Principle advanced in this chapter.

The propagation of these diseases by 'infection' is alone conclusive evidence of their blood-origin. By infection I mean the communication and the reception of disease through the *breath*, rather than by actual contact. Infectious thus differs from contagious disease. Infectious disease is not necessarily disseminated through the breath, possibly also through exhalation from the skin or diseased excretions; but in either case it is received as effluvium by the act of inspiration. This propagation of disease by exhalation and inhalation obviously implies not only that, in common with contagious disease, an animal poison is transmitted, but also that disease thus propagated is a 'blood-disease' of some kind. If, then, this mode of *propagation* can be traced in the history of Eruptive Fevers, their blood-origin will be conclusively demonstrated.

All the evidence on this subject is reducible to two general propositions.

Firstly, healthy persons acquire Fever during intercourse with

those who are similarly affected, and this in proportion as the intercourse has been intimate or protracted.

Secondly; but healthy persons, placed under precisely similar circumstances in every respect, *except* that of intercourse with fever patients, do not acquire Fever. Each of these propositions admits of illustration in various ways.

Respecting *intercourse* with the sick: let a single case of fever arise in a limited district; it gradually spreads from that patient, as a centre, to the circumference of the district; this successive radiation from house to house being guided and regulated, cæteris paribus, by the distribution of the population.

For example, Dr. Alison observed, that in the new town of Edinburgh, where almost all the houses are spacious and well-aired, fever cases are commonly isolated, and when a succession of cases occurs, it rarely *extends* beyond a few individuals. But in the *crowded* and ill-aired parts of the old town there was scarcely an instance—even during those years when the disease was least generally prevalent—of a patient in fever having lain at home during the whole course of the disease without some other cases speedily following; and in many instances, when the disease was most prevalent, this succession *extended* to ten, twenty, or thirty persons, within a few yards of the residence of the first patient.*

When an infected person travels from place to place, his fever lingers after him, and spreads successively in each locality shortly after his arrival.

Referring again to Dr. Alison's experience, he states that in the beginning of the winter 1826-27, a family consisting of a labourer, his wife, and four children, long out of employment, became affected with fever—the mother first. During her convalescence, the father and two sons were sent to the infirmary, and she and the others were turned out of their house, while still very

---

* Observations on the Epidemic prevalent among the lower orders in Edinburgh. Edin. Med. and Surg. Journ., vol. xxviii.

feeble, and found shelter with an acquaintance. When the father and sons left the hospital, the whole family again removed to a third house, considerably distant from the former, taking with them only some very dirty clothing. After many inquiries, no case of fever could be traced in either of these three places prior to the successive occupancy of these poor persons; but it is certain that *many* occupants of the same floor in which they first lived (and no others in that neighbourhood) had fever immediately after them; that in the little court to which they next removed thirty cases of fever occurred within a few weeks after their arrival there, the inhabitants of the same room with them being the first affected; and that from the third lodging-house in which they resided during this winter, and within a fortnight after their arrival in it, four patients with fever were removed to the Queenshberry House.

Similar testimony respecting the *importation* of fever is afforded by the Army and Navy Reports, furnished by Sir Gilbert Blane, Lind, and Trotter.

Intercourse with fever patients being the source of fever, those persons are most liable to be infected whose communication with them is most *intimate* and *constant*.

I can state, writes Dr. Tweedie,* from the most authentic sources, that every physician, with one exception (the late Dr. Bateman), who has been connected with the Fever Hospital, has been attacked with fever during his attendance, and that three out of eight physicians have died. The resident medical officers, matrons, porters, laundresses, and domestic servants not connected with the wards, and every female who has ever performed the duties of a nurse, have one and all invariably been the subjects of fever.

At the Edinburgh Infirmary, formerly, many students, in their attendance on the medical practice, were attacked with fever; but it was also noticed that those who attended the surgical wards

---

* Clinical Illustrations of Fever, 1830.

invariably escaped. Such is the testimony of Dr. Alison, who has also shown, most satisfactorily, that in the year 1827 those of the inmates of the infirmary who had no intercourse with fever patients almost uniformly escaped infection; while six clinical clerks, four employed in the general wards, and twenty-five nurses and servants, who necessarily had close communication with these patients, all became the subjects of fever. In Queensberry Fever Hospital alone, during nine months, when fever was prevalent, no fewer than forty persons, including the resident physician, two clerks, the apothecary, several servants, and all the nurses except two, the whole of whom were much with the sick, were also seized with fever.

In some cases personal experience bears direct testimony that 'infection' has suddenly taken place. Nineteen cases of this kind are recorded by Dr. Marsh,* of which the following is an example:—

A young woman, aged twenty-four years,—admitted into the Whitworth Hospital, in November, 1817,—gave the following account of herself. She made this statement voluntarily, without any question having been put to her, and it appeared to be the result of a strong mental impression which she was anxious to communicate. A few days only prior to her admission, a person not yet recovered from fever came into the house where she resided, and sat down close beside her. She immediately became sensible of a heavy disagreeable odour arising from the person of this individual, which odour disgusted her exceedingly; she was instantly affected with headache, and became so weak as to be scarcely able to move her limbs or stand. That very evening, long-continued rigors supervened, followed by heat, succeeded by perspiration; she spent a restless night, slept in an agitated manner, and awoke unrefreshed. When received into the hospital, she was labouring under the ordinary symptoms of fever, in an intense degree;—she complained of severe headache, great

* Dublin Hospital Reports, 1827, vol. iv.

prostration of strength, and was covered with large and florid petechiæ; her fever was tedious, and her recovery slow.

On the other hand, when communication in every way with the sick is *intercepted,* Fever is not propagated.

Thus the sanitary efficacy of 'quarantine' is often conspicuous; and conclusive evidence of a similar character is to be found in Cheyne's and Barker's Government Reports.

Besides prevention by exclusion, prevention by removal of the sick affords also indirect evidence as to the propagation of fever by infection.

At the time of Alison's observations,* in Edinburgh, there were upwards of one hundred houses, in any one of which, if a single case of fever occurred, the patient was speedily removed, and the place cleaned: no recurrence ensued. But there were hardly five houses among all the closes of the old town, in which a patient with fever resided, during the whole period of his disease, or even half that time, without other cases having speedily occurred. The contrast of these two observations exhibits the inestimable advantage of *Fever* Hospitals, although many other *special* hospitals are superfluous, to say the least. The timely removal of any fever-struck person to an hospital specially appropriated to their reception, may save a whole neighbourhood from infection. For example—in a Fever Hospital at Belfast, one hundred and ninety patients were congregated at one time. Very near this hospital was a school, containing seven or eight hundred young persons, a poor-house with three hundred inmates, and a barrack with one thousand soldiers; and yet these places were never more free from fever than at that time.

All the foregoing evidence in favour of 'infection' is confirmed by the impossibility of admitting any other explanation of the origin of Fever—consistent with well-ascertained facts. They alike lead to the conclusion, that intercourse with infected persons is not only *a* cause, but *the* only cause of Fever.

---

* Dublin Hospital Reports, vol. xxviii., p. 243; also vol. xv., p. 312.

Other causes have been alleged, which, if not disproved, are at least open to doubt. Exposure to cold,* fatigue,* depression of mind,† and personal filth,* have severally been advocated, and found wanting. *They* indeed, and other debilitating circumstances, are each and all *predisposing* causes, possibly; but not the exciting and immediate cause of Fever. Then again, destitution,‡ and impure air—from deficient ventilation,§ or from the putrefaction of dead animal and vegetable matter,‖ in the shape of sewage emanations, have severally had their partisans and opponents. Recently *this* effluvium theory has been revived. Formerly in favour with Sir John Pringle and Cullen, it is now supported by Murchison, Simon, and others.

Typhus fever is ascribed to impure air—from deficient ventilation—co-operating with destitution. Typhoid fever is attributed to impure air, from decaying organic matter—as sewage emanations.

Such is the creed of the Sanitary School; and highly appreciating as I do the truly great purposes of Preventive Medicine, above all things also, impartially seeking truth, I shall state the particulars of Dr. Murchison's conclusions¶ in his own words.

"Over-crowding, with deficient ventilation and destitution, appear to be the essential causes of *typhus* and *relapsing* fever, and to be capable of generating them *de novo*, while there is no evidence that they have any such influence over the production of typhoid fever."

"There are many circumstances which tend to the belief that the emanations from decaying organic matter, or organic impurities

---

* Propagation of Contagious Diseases, &c., 1839. Scott Alison.

† Febrile Diseases, 1800 (Treatise on Fevers, 1820, ed. 4.) Wilson Philip.

‡ Obs. on the Epidemic of 1843 in Scotland. P. Alison.—Management of the Poor in Scotland. Ibid.

§ On Yellow Fever, Typhus Fever, &c., 1811, Bancroft.

‖ Ibid., and Edin. Med. and Surg. Journ., 1810, vol. vi., Contagion. Chisholm.

¶ Med.-Chir. Trans., vol. xli., 1858.

in drinking-water, or both of these causes combined, are capable of generating *typhoid* fever;* but there is no authenticated evidence whatever to prove that such causes can give rise to typhus or relapsing fever."

"There are some grounds for believing that a combination of the causes mentioned in the two last paragraphs may occasionally, though rarely, generate a disease intermediate in its characters between typhus and typhoid, or may (to speak, perhaps, more correctly) cause typhoid fever to *assume* some of the characters of typhus; but such cases cannot be used as an argument in favour of the identity of the poisons of these two diseases."

The first of these conclusions, touching the origin of typhus fever, is endorsed by Dr. Tweedie,† with some qualification. He regards as an indisputable fact "the *possibility* of typhus being engendered by over-crowding in infirmaries, prisons, workhouses, transport-ships, and dwellings of the poor." That "famine and destitution are most powerful predisposing as well as exciting causes of typhus, their potency being attested by various outbreaks of typhus fever in Ireland;" while, respecting typhoid fever, "no connexion can be traced between it and destitution; on the contrary, we meet with it in the habitations of the middle classes, and even in the mansions and palaces of the rich, generally in isolated cases—the subjects of it having been previously in good health, and in the enjoyment of worldly comfort." "Therefore, other causes must be sought for, and concurrent testimony points to this conclusion,—that, whatever they may be, these causes are local and limited—not wide-spread, as in typhus."

Simon‡ corroborates Dr. Murchison,—as to the source of typhoid fever. In reporting on the Windsor epidemic, this fever is attributed to defects in the main sewer of that town, and to the generally imperfect system of house drainage. So also the

---

* Hence the name *pyogenic* fever, as proposed by Dr. Murchison.

† Lumleian Lectures on Fevers, Lancet, 1860.

‡ Report of the Med. Off. of the Privy Council for 1858.

Government commissioners reported of typhoid fever at Croydon; and in this instance we have the testimony of Tweedie that "the disease was essentially enteric fever."

But, in weighing these conclusions, the other scale of the balance is found loaded with evidence of totally different import.

I may refer again to Bancroft's accumulation of facts, and in particular to such as the following. Respecting typhus fever, and *personal* effluvia;—in slave-ships formerly, less space was allotted to each person than he would have occupied in his coffin; and yet, *no* kind of fever was generated, typhus not excepted.

In the Decade frigate, at the breaking out of the French Revolution, one hundred and ninety-three emigrants were deported from France to Cayenne, so closely packed as to form almost a dense mass; and yet *no* kind of fever was engendered, typhus not excepted. In the Black Hole, at Calcutta, one hundred and forty-six persons were forced into a dungeon, so small, that in a few hours one hundred and twenty-three of their number perished by heat and suffocation; and yet the remainder suffered from *no* kind of fever, typhus not excepted. This evidence is augmented by Alison, who uses *this* argument. The very same houses and districts, in which a succession of typhus fever cases is observed in one season, will remain perfectly free during a succession of other seasons, although inhabited by persons presumably liable to this fever,—although equally crowded, dirty, and ill-ventilated, and although the disease be prevalent in other localities, even neighbouring parts of the same town.

Then again, as to typhoid fever, and the *putrefaction* of dead animal matter.

Observe our over-crowded churchyards, with their moving mass of corruption; and yet no true typhoid fever is generated in these localities.

Read Bancroft's work, and Chisholm's article on Contagion, both concurring on this point. That decomposing animal (and vegetable) matter in every conceivable form—whether in sewers,

privies, or in the shape of manure, &c., *never* generates fever of any kind,—typhoid fever, therefore, not excepted.

Coming down to the present day, Dr. Tweedie avows his "scepticism" with regard to this new source of typhoid fever; for, although traceable in some instances to sewer emanations, yet "the large bulk of the population may be thus exposed with impunity, without even suffering in health; and this fever may cease entirely in such places without any sanitary measures having been adopted, and may not reappear for years, though the same influences continue in operation."

Fairly balancing all this evidence *pro* and *con*, we cannot detect fever lurking in sewers and cesspools, or in over-crowded, ill-ventilated dwellings, amid squalid poverty. These circumstances may possibly, indeed, *predispose* to, but do not generate, fever; may be the nurse, but not the parent. In this conclusion the observations of Guy and the judgment of Watson alike concur. The 'sewer spectre' is less terrible than imagination might suggest.

Monstrum, horrendum, informe, ingens, cui lumen ademptum.

I have not said anything specially in *proof* of the *uniformly* infectious origin of 'relapsing fever.'

But if typhus, typhoid, and relapsing fevers are caught by 'infection' alone, and are therefore essentially *blood*-diseases, are they varieties only of one and the same blood-disease? Certainly not. They are shown to be distinct species, by comparing their individual course, their symptoms, their lesions of structure, and their causes. The latter being 'specific,' supply a conclusive test of distinction. This was the upshot of Dr. Jenner's investigations.*

"The peculiarity which entitles a cause to be termed specific, is that of exciting in those exposed to its action one, and only one, species of disease. Further, all specific causes, the products of individuals labouring under disease, can excite in other individuals only diseases resembling in all essential characters those present in

* The identity or non-identity of the specific cause of Typhoid, Typhus, and Relapsing Fever. Med.-Chir. Trans., 1850.

the individual from which they themselves spring. Herein lies the test—the *experimentum crucis*, by which the absolute non-identity of small-pox, measles, and scarlet fever is proved." By parity of reasoning, Jenner fairly argues, that typhus, typhoid, and relapsing fevers are distinct species, because communicated only by persons having these fevers *respectively*. Of two hundred and eight patients brought to the Fever Hospital, from seventy-five sources of infection, it was found that, with only one doubtful exception, when several patients came from the same family or the same house, they all had the same species of fever.

Therefore, by virtue of their causes especially, together with their individual course, symptoms, and pathologico-anatomical lesions, three distinct fevers are indicated; so that I may add, these fevers having been caught by 'infection'—by inhalation—are not only of blood-origin, but of *distinct* blood-origin. Three blood-poisons must exist, as *distinct* as the diseases they induce.

The etiology of other eruptive fevers demands less consideration. Their propagation by 'infection,' and consequently their 'blood-origin,' are generally credited. On this subject popular and professional experience concur. Nevertheless, I shall glance at the remaining exanthems, etiologically, from the same points of view that have just been taken of typhus, &c.; namely—their infectious character, and the question of predisposing causes; finally, that of their spontaneous origin.

Measles spreads by 'infection.' In families and schools this is well known; and it is sometimes witnessed on a far larger scale. In the Feröe Islands, which are separated from each other by narrow but dangerous channels, and which are debarred from much intercourse with the world, both by their geographical position and by their having no external commerce, measles had been totally unknown from the year 1781. The disorder was brought to this locality in 1846, by a man who left Copenhagen on the 20th of March, arrived at the Island of Thorshavn, apparently well, on the 28th, and sickened on the 1st of April. In October, the disease had again disappeared from these islands. But, during that

interval of about six months only, 6000 persons underwent the disease in a population of 7782, distributed among the seventeen islands.*

Propagation by *fomites*, as a mode of infection, is a well-ascertained fact.

A boy—writes Gregory†—belonging to the Foundling Hospital, was permitted to visit his friends at a house where another child lay ill of measles. In the evening (of that day) the boy returned to the hospital and mixed with his playfellows as usual; but in the course of fourteen days he, together with sixty other boys, was sent to the infirmary, ill of measles. The strictest demonstration of this mode of infection is the experimental investigation of Home and Speranza, in which measles was communicated by the direct application of substances infected with the poison.

Rubeola assuredly acknowledges certain *predisposing* causes.

Period of the year has some influence; this disease being most prevalent in the winter. Age more especially predisposes; for childhood is the period of life prone to measles. Lastly, there is the remarkable fact, that recurrence of this disease in the same individual is rare.

Measles is never generated *de novo*. It first appeared at St. Helena in 1808. For twenty-five years it was absent from the island of Madeira, and when in 1808 it did invade this island, almost the whole population were susceptible; in four months it destroyed seven hundred lives!

Scarlet fever likewise spreads by 'infection.' Its propagation in this way among the members of a family, or of a school, is a matter of common observation.

Fomites also convey this disease with remarkable facility, and retain their poisonous influence for a considerable length of time. So readily do fomites convey scarlatina, that, according to Willan's experience, a nurse having received on her clothes or pocket-

* Archives Générales de Médecine, April, 1851. † Op. cit.

handkerchief the vapour from the lungs, the phlegm from the throat, or the discharge from the nostrils of a patient affected with this disease, would infect any child predisposed thereto whom she attended or caressed. He also affirms that any clothes, bedding, or furniture of the sick, are for some weeks capable of infecting those who handle them, and that even the carriage which has conveyed the patient should be avoided. Other authors have made observations of similar import.*

The tenacity with which fomites retain the poison of scarlatina has some remarkable illustrations. An example of this kind came to the knowledge of Dr. Watson.† The disorder had attacked several persons in a large household. When it had subsided the house was vacated, and then (as was supposed) most thoroughly ventilated and purified. A year afterwards the family returned to this house. A drawer in one of the bed-rooms resisted for some time the attempts to pull it open. A strip of flannel had got between the drawer and its frame, thereby causing the drawer to stick. This piece of flannel the housemaid put playfully round her neck. An old nurse who was present, recognising it as having been used for an application to the throat of one of the former subjects of scarlet fever, snatched it from her, and instantly burned it in the fire. The girl, however, soon sickened, and the disease ran a second time through the household, affecting those who had not had it on the first occasion.

Scarlatina is therefore essentially an infectious disorder, yet it is subject to certain *predisposing* circumstances for its development.

Of these, climate has some influence. This disease is peculiarly one of temperate climes. So at least says Dr. Gregory, and that it is comparatively rare in Bengal. All debilitating circumstances favour the production of scarlatina; hence the poorer classes are more liable to it. In point of age, children are apparently most

---

* Memoirs of Medical Society of London, vol. i., p. 438.—Edinburgh Medical and Surgical Journal, vol. xvii.

† Op. cit., vol. ii.

prone to be affected; and also, as with measles, the recurrence of scarlet fever is no less rare. This 'exhaustion of susceptibility,' observed with regard to the recurrence of these and certain other blood-diseases, is at present inexplicable, unless by virtue of Simon's *theory* of the blood-origin of infectious diseases, to which I shall presently advert, in detail.

Does scarlatina ever arise *spontaneously—i.e.*, without infection? The history of this disease answers—No. For example, scarlatina was unknown in America until the year 1735.* Its progress over that vast continent was remarkably slow, but remarkable also for its fatality.

Small-pox is the prince of 'infectious' diseases. This is so generally acknowledged, that by an old law, still I believe in force, "to expose a person in a public highway, infected with this disease, is considered a common nuisance, and is indictable as such."

The breath is not the only source of infection; an equivalent poison emanates from every part of the body of a person labouring under small-pox. The skin, the matter of the pustules, the scabs, are alike sources of infection. Nay, even the dead body is eminently infectious, and this too when undergoing rapid decomposition. The body of a middle-sized man, who had died of small-pox, was brought to the Windmill-street School of Anatomy, and four gentlemen caught the disease, as follows:—One saw the body, but did not approach it; another was near it, but did not touch it; a third, who had frequented the Small-pox Hospital, and had been accustomed to make drawings there from the dead subject, saw this one, but did not touch it; while the fourth touched it with both hands.† All four having caught the disease, three must have acquired it through infection. The reception of small-pox subjects into schools of anatomy is therefore judiciously prohibited by law.

Fomites, impregnated with the variolous poison, are no less virulent than the breath, &c. of a person affected with the disease

* Med. Observations and Inquiries, vol. i., p. 211, C. Colden.

† Medical Gazette, January 31st, 1829.

itself. It was thus first conveyed to the Cape of Good Hope. Fomites, moreover, retain this poison for a long time, if excluded from the air; free exposure soon dissipates it. Therefore, during a walk from house to house, the visitor, friend, or medical attendant ceases to be small-pox in disguise.

The influence of *predisposing* causes, if any there be, can scarcely be discerned in the production of a disease so eminently infectious.

Very few persons indeed escape, and children therefore (if unprotected) must at least pay this penalty to purchase the pleasures of life. All ages, however, are equally susceptible of small-pox. The possibility of its recurrence in the same individual has been asserted again and again; but well-authenticated instances are not common; and if a second edition be improbable, still more so is a third in the same person.

Touching the question of *spontaneous* origin, all we know of the history of variola is irreconcilable with an affirmative answer. For liable, and even prone, as all persons are to this disease, its geographical course can always be traced to intercourse with those who are affected. Take a *modern*, and therefore more authentic, illustration of this law. Small-pox was unknown in America before the year 1492. In 1517 it was first imported into St. Domingo. Three years subsequently the disease was conveyed to Mexico, in the person of a negro. From this parent stock it spread so fatally among a people, all of whom were freely susceptible, as to destroy 3,500,000 of the population of Mexico alone within a very short period.

Chicken-pox, mild and ephemeral as it is, is the offspring of a specific blood-poison. Being, however, a disorder so unimportant, practically speaking, it has not been the subject of much etiological investigation. But its pathological individuality among the exanthemata is undoubtedly owing to its *distinct* blood-origin. The tree is known by its fruit.

Erysipelas is the last of that family group whose birth and propagation, in proof of 'blood-origin,' we are tracing. It is no

exception to its kindred in being capable of propagation by 'infection'—*i.e.*, it may be breathed from person to person. But this mode of reproduction is observed only in *some* cases. Of such, the following, from original authorities on this subject, are noteworthy. Certain cases reported by Dr. Wells* are much to the point.

An elderly man was attacked with erysipelas of the face, and died in about a week from the time when Dr. Wells first saw him —viz., on the 8th of August. On the 19th of the following month an elderly woman, landlady of the house in which the old man had been a lodger, came under the care of Dr. Wells, also with erysipelas of the face. On inquiry it was found that the old man's *wife* had been seized with erysipelas a few days after his decease, and had died in about a week. Another old woman who had nursed the landlady was also attacked with this disease and died. Lastly, a young man, nephew of the old man, was seized, shortly after visiting his uncle, and died in a few days.

In this last case the probability of infection having been the cause of erysipelas is obvious, and with some probability the same mode of propagation may be inferred respecting the whole series of cases. For the report further states, that the landlady had been several times with the old man and his wife during their sickness, and that after their death she had removed some furniture from the room they had occupied to her own apartment.

Cases of like import occurred in the practice of Dr. Wells's contemporaries—Mr. Whitfield, apothecary for very many years to St. Thomas's Hospital, Pitcairn, and Baillie, who made similar observations in St. George's Hospital during the years 1795-96. Many years afterwards, to show the growing impression in favour of this doctrine, I might refer to cases of infectious erysipelas, recorded severally by Dickson,† Blackett,‡ and Stevenson.§

---

* Trans. of a Society for the Improvement of Med. and Chir. Knowledge, 1800, vol. ii., p. 214.

† Med.-Chir. Journ., April, 1819, p. 615.

‡ Med. and Physical Journ., April, 1826.

§ Edin. Med.-Chir. Trans., vol. ii., 1826.

Then Mr. Arnott* took up the inquiry. He collected what had been done by others, and added the results of his own observations. "In one family, the mother was first affected with inflammation of the pharynx, terminating in mortification. On her death the husband was attacked with inflammation of the throat and erysipelas of the face. As he recovered, the daughter was similarly seized with inflammation of the pharynx and severe erysipelas."

Five years prior to this Paper, although published subsequently, a series of observations yet more convincing, if possible, were made by Dr. Gibson.† For example, the infant son of a gentleman was seized with erysipelas on one foot. Afterwards, the mother became affected with erysipelas of the face and scalp. Then the nurse, who suckled the child, was attacked with symptoms of pneumonia. She was removed to her father's house, four miles off. He, who some days before her arrival had received a wound of the scalp, was now seized with erysipelas of the face and scalp, and died. Soon afterwards, a sister, living in the same cottage, had fever with sore throat, from which she slowly recovered. Two children, in the same house, were cut off with what appeared to be croup.

In this series erysipelas ran through seven persons successively; for there can be no doubt, in point of fact, that they were all affected with the same disease, although the description is not *forced* to this conclusion, which is in favour of its being an unprejudiced statement.

Taking a fair estimate of all these cases, it will be obvious that those are most decisive where the persons who became affected resided at some distance from the house in which erysipelas first manifested itself. Persons living together might possibly be subject to some *endemic* source of disease—as bad drainage, or insufficient ventilation.

But if the fact of persons acquiring erysipelas, after *associating*

---

* London Med. and Physical Journ., 1827, vol. lvii.

† Edin. Med.-Chir. Trans., vol. iii., 1829.

with those who have this disease, is in favour of its propagation by infection; so also, when the source of contamination is *removed*, the disease should cease to spread. Of this the following is an illustration:—

The wards of the Dublin Fever and Dysentery Hospital were large and extremely well ventilated. "On paying my daily visit," writes Dr. Brereton,* "I observed one of the patients, who had been admitted with fever some days previously, to be formidably attacked with erysipelas. On the following day I found the patient in the next bed seized with it. On the third day, two patients in the adjoining beds were similarly attacked. I then," continues Dr. Brereton, "became seriously of opinion that the disease was contagious, but resolved not to have those already affected removed until I had tried the result of another day. On the following morning, I found three more in like manner labouring under the disease; and what made it more remarkable, they were all similarly attacked in the head and face. I had them immediately put into another ward, where there were no patients; they all recovered, and no more erysipelas afterwards appeared." In this series seven patients were attacked successively, and here also, the (supposed) cause being removed, its effect ceased.

Respecting, then, the propagation of erysipelas by infection, we have the *double* test of causation fulfilled; the presence of erysipelas is at once followed by this disease in persons associating therewith, and, being withdrawn, the disease at once ceases to multiply.

It has been affirmed by some men of observation that this law holds good only of erysipelas affecting the *face* and *scalp*. Arnott and Watson are of this opinion; but, restricting, as they do, their definition of this disease to its manifestation on the head and face, the law of infectious propagation becomes absolute. Other authorities, however—and the majority—who do not take this circumscribed view of erysipelas, are inclined to acknowledge its propagation by infection.

---

* Dub. Journ. of Medical Science, vol. vi., p. 176.

My own observation has not been sufficiently directed to this matter to warrant me in offering a decisive opinion; further than this, I can throw into the scale my testimony as to the infectious character of facial erysipelas, and probably of every *idiopathic* manifestation of the disease on any part of the body.

Are *fomites* capable of transmitting this disease? Yes, certainly. Among the earlier writers, Wells observed that a certain patient caught erysipelas in consequence of being laid in the unchanged bed of one who had died of it. Similar instances have occurred in the experience of most practitioners, unless due precautions were taken to prevent this risk of infection. But the wards of an hospital may themselves become contaminated, and communicate the disease on a larger scale. By Gibson's Report, already quoted, we learn that a woman, with erysipelas of the hand, having been admitted into the Montrose Infirmary, and the patients in either adjoining bed having become affected, the whole ward was then cleared out, cleansed, whitewashed, and fumigated. "Yet when they were again placed in that ward the disease reappeared," and it became necessary to remove all the patients from this little infirmary, and to take every precaution ere the infection was eradicated.

Respecting the contaminating power of fomites, in the shape of furniture, floors, &c., I may mention that dry rubbing instead of washing the floors of an hospital, or the decks of a ship, is the surest safeguard against this source of infection. But fomites of all kinds retain most tenaciously the poison of erysipelas; and, indeed, Gregory is of opinion that this poison is banished with more difficulty than any other known miasm.

Erysipelas acknowledges, apparently, a *traumatic* origin, in some instances. Hence, therefore, all kinds of wounds, injuries, surgical operations, and irritants,—such as blisters, caustics, &c.,—are accredited causes of this species of inflammation. Besides mechanical and chemical irritants, heat and cold are accorded their share of importance.

All these assaults, however, from *without* the body, are rein-

forced at least by conditions *within;* resulting mostly from previous habits of intemperance, previous hardships, or both; perhaps also from mental depression—circumstances which have depraved the blood and enfeebled its circulation. Apart from these *predisposing* causes, erysipelas, probably, would not arise; with their concurrence the scratch of a pin only may be *apparently* the *traumatic* origin of this disease. Yet even in such cases its infectious character becomes developed. In one instance, noticed by Lawrence,* —who is not, or was not, disposed to acknowledge this point,—erysipelas of the head and face, consequent on the insertion of a seton in the neck, was the only presumptive cause of this affection in two other persons. Travers,† indeed, goes so far as to affirm that he has *repeatedly* seen the idiopathic arising from the traumatic, or this from the former, and either from its own source.

There is, then, abundant evidence to show that erysipelas is propagated by 'infection,' and is *essentially,* therefore, a 'blood-disease;' those persons being prone to catch it whose blood has become vitiated, and circulation enfeebled, by various circumstances conducive to this joint result. Consequently, we are not surprised to find erysipelas most prevalent in spring and autumn, or at least when hot and cold wet weather alternate; and if period of the year has some predisposing influence, so also has the period of life, they being most liable who are *old* in years, or old for their years. As to the recurrence possibly of this disease in the same individual, I can speak from my own personal experience, having had it *four* times, severely, in my head and face, when from nine to thirteen years of age. I was then a pupil at the King's College School (London), and although many years have elapsed, I well remember the flaming pain, and that my face was like a distended bladder. I just mention these particulars to verify so early a reminiscence in evidence of an important fact, that susceptibility to *true* erysipelas—*i.e.,* affecting the head and face—is not exhausted by one attack. Unlike by virtue of most

---

* Op. cit. † Constitutional Irritation. Further Inquiry, 1835, p. 149.

eruptive fevers, the blood does not lose its capability of undergoing this defection, again and again.

Lastly, the possibility of erysipelas arising *spontaneously* —that is to say, in an individual without any assignable external cause, and therefore without infection—is, I think, indisputable.

Plague is undoubtedly a blood-disease, and in common with the *whole* (?) class of eruptive fevers with which it is associated, this disease also *may* prevail *epidemically*. Omitting, however, any discussion respecting its etiology, there is yet a group of diseases whose blood-origin is a question of much interest as bearing immediately on their rational prevention.

Certain infectious disorders, unlike eruptive fevers, are expended on the 'mucous membranes' alone. Hooping-cough, influenza, and cholera belong to this class. Each demands, in the first place, sufficient description to identify the particular disorder; then its 'blood-origin' will be investigated. This Etiological Principle having been demonstrated as regards these diseases, and extended, as it has been, to the exanthemata, we shall thus gain a vantage ground from whence to consider the *whole* category of infectious diseases from a Preventive point of view.

Hooping-cough, or pertussis—known, also, by many other names unnecessary to specify—is, anatomically speaking, an inflammatory affection of the *laryngeal* mucous membrane, extending perhaps to that of the trachea and bronchi. The laryngeal character of this inflammation was advocated by Dawson,* while its extension was the doctrine of Guersent, Marcus, Dewees, Laennec, and Watt.† Other authors, however, have held that hooping-cough is merely a spasmodic affection. Such was the pathological interpretation inculcated by Cullen, Leroy, Lobenstein, Jahn, and Webster. Expressing themselves more definitely, Lobenstein and Breschet ascribe this presumed spasmodic affection

---

* Nosological Practice of Physic, 1824.

† Treatise on Chin-cough, 1816.

to irritation of the phrenic and pneumogastric nerves. Arriving yet more nearly at the truth, Desruelles suggested that hooping-cough is at first inflammatory and then spasmodic; wherefore he designated it "broncho-cephalite."

The phenomena of this disease are remarkable. It is one of those few diseases of which the functional manifestations are uniformly the same, and present in all cases; but, as if to illustrate the general insufficiency, at least perhaps inaptitude, of 'functional symptoms' to determine diagnosis, the symptoms of hooping-cough are those also of laryngismus stridulus,—a disorder essentially distinct, being in nowise infectious.

Respecting hooping-cough, then, what are the earliest and most assured symptoms of its approach? A child (for it is mostly children who are affected) is overtaken with the symptoms of an ordinary cold in the head. Having continued for about ten days, it is succeeded by that cough from the character of which the disease derives its (popular) name. This (hooping) cough is remarkable in two particulars. A number of short, but violent, expirations, or choking coughs, in rapid succession, exhaust the breath and leave the little sufferer verging on suffocation. Immediately, a long-drawn inspiration brings relief, this being accompanied with a 'hooping' sound, as if the air was admitted tardily through the larynx. And so it is; the rima glottidis is partially closed, spasmodically, from first to last. Without delay, the series of choking coughs is renewed, and the child fights for breath. In this agony of suffocation, the face becomes red and turgid, the external jugular veins visibly swollen, and the eyes bloodshot—even hemorrhage therefrom, or from the nose and ears, may happen. Again is heard a long crowing inspiration, then immediately convulsive coughing, until at length, these efforts having been repeated perhaps many times, a small quantity of thin mucus is expelled from the air-tubes, or vomiting without expectoration, or both together, terminate the paroxysm. Slight fatigue and breathlessness are experienced for a short time in some cases, but generally the child feels quite relieved, and at once

returns to play; if, indeed, the fit passes off with vomiting, it gives a good appetite in exchange. This ease and comfort are not of long duration. Similar attacks of coughing and crowing recur in paroxysms by day and night, so that any occupation or amusement is suddenly interrupted, and sleep as suddenly disturbed. After a time, the mucus becomes thicker and more abundant, and being expectorated more readily, the attacks are shorter and easier. In bad cases, however, there is obvious breathlessness throughout the disease,—in the intervals of paroxysmal seizure, as well as when spasm is urgent. An incessant *catching* breathing allows no ease. All this suffering, if not danger, lasts usually from one month to three, or more. Having in view only the diagnosis of hooping-cough, I need not describe its possible complications—with structural lesions of the lungs, with cerebral disorder, with irritation of the mucous membrane of the intestinal canal. Fever, in some degree, may accompany this cough from its commencement, and exacerbations of pyrexia occur perhaps, as evening approaches; but it was observed by Sydenham that, in many cases, pyrexia is altogether absent.

Hooping-cough is undoubtedly 'infectious,'—so much so that, although doubted by Laennec, Desruelles, and other competent witnesses, its infectious character is established by popular observation.

Thus, this disease is quickly propagated through a family or school where children are *congregated* together; and when any one affected is removed to a distant part previously free from the disease, then and there it begins to spread, being obviously disseminated by the new comer. Conversely, *remove* the infected person, if only one (the cause), and the disease does not reappear. These well-known facts are strengthened by cases recorded by Rostan* more particularly; also by Guersent, Dugès, Dumeril, and Brettoneau.

Are *fomites* capable of conveying the poison of hooping-cough?

---

* Médecine Clinique, tom. ii., p. 352.

Yes, verily. For example :—A ship, having seventeen children on board, labouring under this disease, touched at St. Helena. The captain was refused permission to send the foul linen on shore to be washed, because, under similar circumstances, hooping-cough broke out among the laundresses, and spread so extensively among the inhabitants as to kill upwards of sixty persons.*

Scarcely anything can be said with certainty respecting any causes *predisposing* to hooping-cough. It is perhaps equally common to both sexes. Susceptibility is usually exhausted by one attack ; but Blache mentions an instance of a child, affected with this disease, being sent to his grandfather's residence, when both grandfather and grandmother also became affected and suffered severely ; showing, moreover, that old age is not exempt.

Influenza is an 'infectious' fever, yet prevailing epidemically. It is exhibited locally by inflammation of the *naso-pulmonary* mucous membrane chiefly. And if hooping-cough resembles another functional disorder—laryngismus stridulus—although an essentially distinct disorder ; so likewise the (earliest) symptoms of influenza cannot be distinguished from those of ordinary catarrh ; thus affording an additional and convincing illustration of the diagnostic misguidance of 'functional symptoms.' This misguidance would become more conspicuous by comparing the several epidemic visitations of this disease which have occurred in Great Britain during some hundreds of years. These different epidemics alike presented a great similarity of symptoms, and they were those of ordinary catarrh. "Epidemic catarrhal fever,"† therefore, is synonymous with influenza, and its symptoms are as follows :—

Sneezing, tingling, and an acrid discharge from the nostrils, are accompanied with a short, frequent, harassing cough, hoarseness, and a feeling of constriction of the throat and chest.

---

* Morbid Poisons. R. Williams.—See also by Rosen—Versuche für die pract. Heilkunde, t. i., p. 134.—Praxeos Medicæ. Frank. Vol. ii., part ii.—Mem. cui aggiudicata. Panada. P. 41.

† Annals of Influenza, or Epidemic Catarrhal Fever, in Great Britain, from 1510 to 1837. Theophilus Thompson. 1852.

These symptoms betoken inflammation of the corresponding mucous membrane; and inspection of the fauces discloses considerable redness and turgescence. The eyes are suffused and bloodshot, and pain is often felt over the frontal sinuses, the cheek-bone, or behind the sternum. In fact, all the offsets of the naso-pulmonary mucous membrane are involved. Expectoration is at first scanty and difficult, the sputa consisting of thick viscid mucus, usually devoid of air-bubbles; subsequently it becomes copious, opaque, and muco-purulent. Sonorous and sibilant rhonchi are readily detected by auscultation, and frequently partial crepitation also, which is most apt to occur at the lower portions of the lungs. These physical signs contrast with the absolute stillness everywhere within the thorax during an attack of hooping-cough. The atmospheric engine is there, but the air can scarcely enter to set it in motion.

The *intestinal* mucous membrane shares the irritation elsewhere experienced in full force. A white tongue, covered with creamy mucus, or coated with moist yellowish fur, and presenting along its edges elevated papillæ of a vivid red colour, is one concomitant of influenza; nausea or vomiting another. Pain and tenderness of the abdomen, with mucous diarrhœa, are not unfrequent, especially when the pulmonary mucous membrane is less involved. In most instances the urine is scanty and high-coloured, soon becoming thick and reddish, or assuming a whey-like appearance, and depositing copiously a pink or whitish sediment.

The fever of influenza is its *most* characteristic expression.

A feeble, quick, soft pulse at first, afterwards a slow and perhaps intermittent one, suggests no conception of the *nervous* collapse almost peculiar to influenza. In degree, it is compared by Thompson to that of cholera. Commencing with shivering and creeping chilliness, a sensation as if cold water were running down the back, with aching pains in the neck, back, and loins; the skin is at first hot and dry, and more so towards evening, when a febrile paroxysm commonly occurs. As the disease progresses, perspiration relieves this heat of skin, which now exhales

a flat, musty smell, and sometimes assumes a bluish hue. Nervous depression is overwhelming, and sleeplessness, vertigo, perchance delirium, complete the picture. But in some cases the pulmonary, in others the intestinal, in others the cerebral functions, are chiefly involved; and every epidemic catarrhal fever, besides presenting examples of either variety, is characterized by the *predominance* of some one in particular, and in some instances by phenomena peculiar to itself. Influenza holds possession of an individual for a period varying from three or four days only, to a fortnight or three weeks. From two to fourteen days (Thompson).

A blood-poison is the immediate cause of all this functional disturbance; for influenza is 'infectious.'

This proposition can be demonstrated in various ways. If an infectious, as well as epidemic catarrhal fever, it should spread more rapidly, in proportion as the population travels more freely from place to place. Agreeably to this law of progression, "in 1803, *four* months elapsed before the malady had accomplished its circuit, and *six* before it finally ceased in Britain; whereas, in 1837, the visitation was effected in *two* months, and over completely in *four*."* Other remarkable facts, of similar import, can be gathered from the epidemics of 1803, 1782, and 1775.†

Epidemic diffusion, unquestionably, is the chief mode by which this disease spreads. It may be inferred when influenza appears *simultaneously* in localities *distant* from each other. For example, *simultaneously*, in the fleets of Admiral Kempenfelt and Lord Howe, in the year 1782; again, *simultaneously*, in Cape Town and London, at the close of 1836.

Predisposing causes also are possibly at work; but generally speaking the attack is so universal, that large portions of the population, irrespective of age, sex, and different hygienic conditions, are equally affected. Locality, however, seems to have some influence; the inhabitants of low parts of towns being visited,

---

* Op. cit., Thompson.

† Ibid.

usually, in a greater proportion and more severely than those who reside in higher districts. Susceptibility to this disease is so far exhausted by one attack, that it scarcely ever recurs to the same individual in the same season; yet relapses are not unfrequent. Many persons who suffered in 1775 were again attacked in 1782; and notwithstanding the prevailing epidemic of 1832-3, that of 1837 overspread the entire population. Liability to recurrence therefore seems to be actually increased by recurrence.

Cholera, prevailing epidemically, is *also* 'infectious,' and therefore a blood-disease. It is expended on the *gastro-intestinal* mucous membrane. I do not mean autumnal diarrhœa, properly called (summer) *cholera*, being denoted by a remarkable flow of *bile;* I allude to Asiatic or Malignant Cholera, as it is sometimes termed. This being epidemic, the other is sporadic only.

The diagnostic symptoms of epidemic cholera are narrated in a previous chapter; yet their reintroduction here forms part of the design of this one. These symptoms further illustrate the inconclusiveness of 'functional' disorder; and moreover, that the turning point of diagnosis in this case is the chemical (and physical) character and significance of choleraic evacuations.

A man suffers for some days from copious bilious diarrhœa. At length his skin feels cold and clammy (sometimes dry), loose also, and shrivelled; while its usual pink colour shades off to a dusky blue tint, more obvious about prominent parts, as the fingers, ears, nose, lips, and around the eyes, which are suffused and sunken. With these outward appearances, the pulse is feeble, and at last scarcely perceptible; for although the heart beats forcibly, the blood is now too viscid—from prolonged alvine evacuations—to be adequately propelled. The breathing, therefore, is hurried and oppressed, the cerebral functions are blunted and obscured. These functional disturbances constitute the common autumnal cholera of this country, and are symptomatic of collapse. But they are *premonitory* only. Others may supervene of very different significance. A whitish flocculent

fluid, resembling rice-water rather than bile, is expelled from the bowels. Pint after pint, and quart after quart, are discharged at short intervals, and apparently quite involuntarily, suddenly and often with great violence, as from a tap; at the same time a stream of fluid, white or greenish, is copiously spirted from the mouth, and thrown to some distance, without, however, any straining effort. How much the blood's *volume* is thereby reduced, will be obvious. No drain of bile merely could produce such utter collapse. Hence the more enfeebled circulation, which the heart labours in vain to restore; hence the livid, cold, and shrunken surface, the abortive breathing, and fatal lethargy of Asiatic cholera. The poor victim, around whom the shades of death are gathering, may indeed be for a time disturbed and restless, but soon an apathetic indifference steals over him, from which perhaps he is roused only by cramps of the legs, thighs, and abdomen. With these he will cry out in a hollow and sepulchral voice for drink. A ferocious thirst instinctively prompts his entreaties for water, wherewith to supply the reduced volume of blood—apt proof of a natural restorative tendency. But water is not blood, only one of its components. The evil lies deeper. Examine the rice-water evacuations, and we at once detect those materials which the blood has lost; analyse the blood itself, and we further discover the absence of these components, and the presence of those excretions which should have been thrown off, but are now retained. In other words, the (chemical) composition of choleraic evacuations, when contrasted with that of the blood (in this disease), supplies the surest ground for our *diagnosis* of collapse in epidemic cholera, and that consequent on bilious diarrhœa alone. Rice-water evacuation is the only sign peculiar to, and constant in, epidemic cholera.

Respecting the question of 'blood-origin,' the evidence of chemical analysis (just adduced on behalf of diagnosis) is corroborated by the 'infectious' character of malignant cholera; both alike declaring it to be a blood-disease. Of the latter species of

evidence there is ample abundance, on the testimony of many reliable witnesses. In the following instances, I have selected what appears to me most incontrovertible.

Numerous cases of the *importation* of cholera from an infected to a distant healthy town or locality, have been collated by Dr. J. Y. Simpson.*

William Woodley, aged twenty-three, went from East Hagbourn, on the 22nd of March, 1832, to London. He slept the four following nights in Tooley-street, where cholera then prevailed. On the 24th, two days only after his arrival in this locality, diarrhœa began. On the 5th night he slept in the neighbourhood of St. Albans, and returned to East Hagbourn the following day. Vomiting, with copious rice-coloured fluid evacuations, cramps, &c., supervened in two days more. The cramps ceased the next day, but the diarrhœa subsided only during the four days ensuing, when he was quite convalescent. Mary Woodley, aged fifty-one, the mother and nurse of the above person, was seized on 1st of April, with violent cramps of the trunk and extremities, universal coldness, vomiting and purging, with rice-water evacuations, blueness of skin, &c. She died in twenty-three hours. Mary Winders, aged seventy, nurse to the first case, had symptoms precisely similar to those in the last, commencing on April 5th. She sank in fifteen and a half hours.

A foot-note states that up to the date of these cases, no other instances of malignant cholera had ever been observed in East Hagbourn, or nearer it than Oxford, twelve miles off; excepting two suspicious cases that occurred at Milton, five miles distant, the one case recovering without medical assistance, the other after ten days' illness.

Eight cases were imported into Glen, as follows :—First and second cases. Robert Anderson and his wife, after spending some nights at Airdrie, about fourteen or fifteen miles distant, in a house where some of their relations had died of cholera, returned

* Edinburgh Medical and Surgical Journal, 1838, vol. xlix.

from thence to Glen on the 4th of April. At the time of their arrival, both had diarrhœa, but concealed the fact, owing to the strong prejudice then existing regarding it. By the 8th, collapse had supervened. The woman died next night; the man recovered, after being in this state for thirty-three hours.—Third and fourth. Two of Anderson's children, who had remained at home while their parents were visiting the infected house at Airdrie, were seized with distinct, and in one case severe, symptoms of cholera, on the 12th. They both recovered.—Fifth. A man named Benny was suddenly taken ill, with all the symptoms of cholera, on the morning of the 10th. Being immediately subjected to active medical treatment, he recovered.—Sixth. Jenckens, another man, similarly attacked on the evening of the same day, also did well. It is not stated what particular communication the two last persons had with the Andersons.—Seventh. A daughter of Jenckens was seized on the 11th.—The eighth and last case occurred on the 15th; it was that of a person who had repeatedly visited Anderson's wife, on her death-bed.

Very many other instances of importation were traced at Bathgate, Dollar, Carnwath, Ferryden, and Boddin, Hutton-Hawick, Campbelton, Greenock, Doura, Dromore, and Portaferry, North Shields, Durham, Hartlepool, Warrington, Prestonpans, Cockenzie, Edinburgh, and in various towns of France.

In all these localities cholera spread, more or less freely, among those persons to whom it was imported. In some places it attacked one, or a few only, of those residents who had communication with the imported sick. Such was its *limited* course in the cases I have quoted, and at Bathgate.

But, in other localities, the malady spread to a greater or less extent, over the *whole* community, from the first centre of importation. This more extended range was observed at Ferryden and Boddin, for example. Cholera was brought thence by a sailor from a smack, on board which two cases had occurred. He "carried his clothes and bedding" to his house at Ferryden. A day or two afterwards, two children in the village, who were

reported to have been seen tumbling, during the preceding day, on this man's mattrass, as it was laid out to air, were seized with rapidly fatal cholera, and died. This occurred at a time when the disease was considered to have nearly or entirely disappeared from Scotland. The mother of the two children alluded to fell a victim to cholera, and it spread through the whole village, though not rapidly; yet, during the four weeks the disease prevailed, it carried off twenty-seven out of the seven hundred inhabitants. At Campbelton, thirteen inhabitants were infected from one centre of importation, in the short space of eighteen or twenty days; and eventually ninety-eight persons were attacked.

Numerous cases of importation of cholera from an infected to a healthy part of the *same* city are also recorded by Dr. Simpson. They occurred in London, Edinburgh, Glasgow, Manchester, Paris, Prussia, and Russia.

A third series of cases show the importation of cholera by infected ships.

With this consolidated fund of evidence, no one can, I think, disbelieve the infectious character of cholera.

Fomites also are capable of conveying the poison, whatever it be. In the Ferryden case a mattrass was the vehicle. Similar cases occurred at Hulton from washing infected clothes; and at Edinburgh a person caught the disease from having slept in the bed of a cholera patient.

Cholera, then, is another offspring of 'infection,' and therefore a blood-disease. But its blood-origin is in no way incompatible with the fact that this disease may also spread epidemically. Infection is not necessarily its *only* source.

The *epidemic* diffusion of cholera is suggested by its history.

"Here is a disease," observes Sir H. Holland,* "which, appearing first in the Delta of the Ganges, and diffusing itself gradually over the provinces of India, subsequently spread with

* Medical Notes and Reflections, 1855, p. 575.

more rapid course, so as to embrace within a period of seventeen years almost the whole habitable circumference of the globe—reaching China on the one side, the Mississippi and Mexico on the other; its general course traceable, step by step, over the whole of this vast distance, yet very irregular in details both as to time and space; frequently appearing in remote parts long before it affected countries much nearer the general line of its direction; yet never, even in these cases, without traces of its presence and progress throughout various parts of the intermediate distance. During the whole period of its first diffusion, as well as during the twenty years which have since elapsed, under every climate and in every place of its occurrence, the disease has been absolutely identical in kind; the only variation, that of the degree of intensity and virulence. In whatever countries it has existed, a tendency has been observed to its reappearance as an epidemic in successive years; though often with change in the particular localities affected, and under other conditions still very imperfectly known."

Observing more closely the *route* by which cholera has travelled, its diffusion *epidemically* becomes even more conspicuous. With the map in hand, taking a bird's-eye view, we trace cholera in its course, overleaping all barriers, natural and artificial. From India it spread to Persia, thence to Russia, and across through Poland to Germany. At length it reached Hamburg. Then, crossing over to this country, it first visited Sunderland, on the eastern coast, and eventually overspread the whole of these islands. Spanning the vast Atlantic Ocean, the disease invaded America; turning, at the same time, in a south-easterly direction, it ravaged France and Spain, the north coast of Africa, and Italy.

Atmospheric conditions would certainly seem to facilitate the diffusion of cholera. Reporting on this subject, Mr. Glaisher states that the three epidemics of 1831-32, 1848-49, and 1853-54, were severally attended with a particular state of atmosphere in London and its immediate neighbourhood. That they were

characterized by "a prevalent mist, thin in elevated localities, dense in low. During the height of the epidemic in all cases, the reading of the barometer was remarkably high, and the atmosphere thick. In 1849 and 1854 the temperature was above its average, and a total absence of rain, with a stillness of air amounting almost to calm, accompanied the progress of the disease on each occasion. In places near the river the night temperatures were high, with small diurnal range." Among atmospheric conditions are enumerated "a dense torpid mist, and air charged with the many impurities arising from exhalations of the river and adjoining marshes; a deficiency of electricity; and (as shown in 1854) a total absence of ozone, most probably destroyed by decomposition of the organic matter with which the air in these situations is strongly charged."

Associated with atmospheric conditions is the geographical question of altitude. Elevation of the soil in London, Dr. Farr affirms, has a more constant relation to the mortality from cholera than any other known condition. The mortality is inversely as the altitude.

Epidemic diffusion must be referrible to *some* condition of the atmosphere, but it would appear that the cholera-poison may be taken into the body by drinking water in which it is dissolved. The mucous membrane of the alimentary canal, therefore, no less than that of the lungs, will transmit this poison. And *sewer*-water is probably its representative. Some striking facts have been collected by Dr. Snow, which warrant the presumption that a most fearful outbreak of cholera in Soho was attributable to the water of a certain pump, contaminated from a neighbouring sewer. A remarkable converse fact was reported by Lawrence. Bethlem Hospital and an asylum for children, called the House of Occupation, stand near together on an open space of ground between fourteen and sixteen acres in extent, lying in the parish of St. George, Southwark. Being dissatisfied with the filthy water then supplied by the Lambeth Company, the Governors, some thirty years ago, sank Artesian wells on the premises, and

the pure water thus procured is used exclusively in these two institutions, which number between them about seven hundred residents. Not a single case of cholera occurred in the hospital or in the House of Occupation in either of the three epidemics; although the disease prevailed extensively in the parish and in the streets in their immediate vicinity.* Mr. Simon reports† that "the population drinking dirty water appears to have suffered three and a half times as much mortality as the population drinking other water."

Taking a retrospective view of all these observations concerning the etiology of cholera, it appears that this disease is propagated by infection, aided considerably by epidemic diffusion, *i.e.* by atmospheric conditions, the nature of which has not yet been discovered;—that drinking impure water, especially sewer-water, as it may be termed,—or, perchance, water tainted with cholera evacuations,—is another source of this disease.

Analyzing these conclusions, we arrive at this final one: that an *animal* poison certainly, emanating from a person affected with cholera, and some undefined, but perhaps similarly contaminated state of the atmosphere, co-operate to propagate this disease.

I have yet to add that the whole population of any place is not equally susceptible. Certain *predisposing* causes undoubtedly prepare the way. Pallid poverty soon makes acquaintance with cholera, which, with trembling intemperance, readily ripens into friendship, and all dwell together.

The question of *spontaneous* generation, and other theories of Cholera, has been fully discussed elsewhere.‡

I shall conclude this etiological investigation of 'infectious' disorders by adverting to the intimate nature of blood-poisons—a branch of pathology hitherto unexplored, and necessarily obscure.

Certain *general theories* have been propounded, more or less

---

* Op. cit., Watson, vol. ii., p. 531. † Ibid.

‡ Registrar-General's Report on the Mortality of Cholera in England, 1848–49, pp. 74–83.

consistent or inconsistent with known facts. Availing ourselves of all that evidence by which the infectious character of a large class of diseases is demonstrated, let us now take a cursory view of the theories in question,—standing, as it were, on the elevated ground to which we have been conducted.

Nearly all infectious disorders occur commonly but once in life; and Paget's explanation* of this distinctive characteristic is as follows:—The 'maintenance of morbid structures' is so familiar a fact, that not only its wonder, but its significance, seems to be too much overlooked. What we see in scars and thickenings of parts appears to be only an example of a very large class of cases; for this exactness by which the formative process in a part maintains the change once produced by disease offers a reasonable explanation of the fact that certain diseases usually occur only *once* in the body. The poison of small-pox or scarlet fever, for example, being once inserted, soon, by multiplication or otherwise, affects the whole of the blood—alters its whole composition; the disease, in a definite form and order, pursues its course; and finally, the blood recovers, to all appearance, its former state. Yet it is not as it was; for now the same material—the same variolous poison—will not produce the same effect upon it; and the alteration thus made in the blood or the tissues is made once for all; for commonly, through all after life, the formative process assimilates, and never deviates from, the altered type, but reproduces materials exactly like those altered by the disease; the new ones, therefore, like the old, are incapable of alteration by the same poison, and the individual is safe from the danger of infection. So it must be, Mr. Paget conceives, with all diseases which, as a general rule, attack the body only once.

The best refutation of this theory is supplied by its author. For, he continues, "in another set of diseases, we see an opposite, yet not a contradictory, result. In these, a part once diseased is, more than it was before, liable to be affected by the same disease;

---

* Lectures, vol. i., p. 50.

and the liability to recurrence of the disease becomes greater every time, although in the intervals between the successive attacks the part may have appeared quite healthy. Such is the case with gout, with common inflammation of a part, as the eye, and many others, in which people become, as they say, every year more and more subject to the disease." Yet, as Mr. Paget adds, "in reference to the physiology of the formative process, these two classes of disease both prove the same thing—viz., that an alteration once produced in a tissue, whether by external influence or by morbid material in the blood, is likely to be perpetuated by the exactness of assimilation observed in the formative process—*i.e.*, by the constant reproduction of parts in every respect precisely like their immediate predecessors."

If, then, this "constant reproduction" *protects* the individual from a second attack of certain diseases—small-pox, &c.—why does it not also preclude his susceptibility to gout, &c., having once undergone an attack? The fact that such is not the law is quite incompatible with the theory of its entailing subsequent immunity to *certain* diseases only. This cannot be the true explanation of (their) *non*-recurrence. On the contrary, the "constant reproduction of parts in every respect precisely like their immediate predecessors," begetting (as is alleged) an increasing tendency to the recurrence of one class of diseases, as that of gout and rheumatism, &c., should also predispose to the reproduction of every member of the whole family of eruptive fevers. Recurring small-pox, &c., should be the rule, not the exception. And this suggests another objection to Mr. Paget's theory.

The possible *recurrence* of all these diseases is itself antagonistic. For if the mal-assimilation once engendered is perpetuated, so as to protect the individual against a recurrence of the same blood-disease, its recurrence should be absolutely impossible—which is contrary to experience.

Mr. Paget himself foresaw this difficulty, and thus endeavours to meet it. Susceptibility to the recurrence of the same blood-disease, of which there are numerous instances, is but "illustrative

of the operation of that inner, yet not less certain, law, that after a part has been changed by disease, it tends naturally to regain a perfect state. Most often the complete return is not effected, but sometimes it is, and the part at length becomes what it would have been if disease had never changed it." In fact, the blood thus regains its original susceptibility of undergoing the same disease again.

Fully acknowledging, as I do, the operation of this restorative power,—exhibited in a thousand forms, and therefore, *a priori*, possibly on behalf of blood-diseases,—I cannot recognise its operation in the prevention of some such diseases—*e.g.*, gout, which tends to return more readily with each return. And if this restorative power obviously does not operate in favour of certain blood-diseases, we are not warranted in *imagining* that other diseases of the same kind are subject to it.

Moreover, when restoration does ensue, it does so slowly ("at length"), whereas recurring blood-diseases—*e.g.*, gout—return more readily with each recurrence; an increasing liability altogether at variance with the operation of a restorative power.

To crown the argument from this point of view against Mr. Paget's theory, I would urge the pathological law on which it is based. The *perpetuation* of mal-assimilation is altogether inconsistent with any restorative tendency.

To sum up, then, my objections to this theory, the fact of certain blood-diseases—*e.g.*, gout—acquiring an *increasing* liability to return with each recurrence, is irreconcilable with the theory that susceptibility is precluded by the perpetuation of mal-assimilation. The formative process once being diverted from its normal character, and perpetuating, as it does, any morbid impression it has received, cannot be said to prevent that impression a second time. The individual is not thus protected. Rather does this pathological law favour the recurrence, even again and again, of the same blood-disease. Then, again, cases (not a few) of recurrence cannot be ascribed to the operation of a natural restorative tendency, renewing a healthy condition of blood, and thereby

its original susceptibility; for certain blood-diseases—*e.g.*, gout—exhibiting no such tendency, we cannot *assume* this reservation in favour of other blood-diseases; more especially seeing that restoration is a very slow process, whereas those blood-diseases which recur do so more readily with each recurrence; thus disproving *any* restorative tendency (and renewed susceptibility); while, lastly, the original tendency of gout, &c., to recurrence disproves the *law* implied by Mr. Paget's theory. Such is this theory respecting non-recurring blood-diseases, and such are the objections to which I conceive it is open.

Extending our view to the *whole* class of 'infectious' disorders, three theories present themselves for critical examination, each claiming consideration, and one more especially. I shall take them inversely in their order of probability.

The 'parasite' theory, advanced by Sir H. Holland in these terms—"The hypothesis of animalcule life as a cause of disease"*—is applied by this author to explain the epidemic diffusion of cholera. But the question is put generally, thus:—"What weight may we attach to the opinion that certain diseases, and especially some of epidemic and contagious kind, are derived from species of animalcule life, existing in the atmosphere under particular circumstances, and capable, by application to the lining membranes or other parts, of acting as a virus on the human body?" This by no means new speculation is, in substance, the theory of Henle also.

Without stating fully all the arguments brought forward in support of this theory, they are briefly these:—That by it can be explained three well-known facts which characterize the diseases referred to—viz., their latency after infection, the reproduction of the specific poison within the body infected, and their dissemination epidemically.

But the objections urged by Simon are to my mind so convincing, that every argument in favour of the parasite theory is altogether anticipated.

---

* Medical Notes and Reflections, 1855, p. 563.

Briefly stated, these objections are: that "symptoms are absent which parasites, if injurious, would unfailingly produce; symptoms are present which parasites, however injurious, could not produce; and thirdly, the parasites themselves elude discovery."*

Compare infectious diseases with those of known parasitic origin, and what analogy, much less resemblance, do we discover between them? "If you examine parasitic diseases from first to last, you will find that they are, perhaps of all known maladies, the most essentially *local*." "An animal invaded by parasites dies only when, in an ostensible mass, they occupy the space or the nutriment of its body. Their effects on life are in direct proportion to their manifestness in parts. The severest operation of morbid poisons, on the contrary, leaves no trace; local disorganization or detriment, instead of having advanced to its utmost possibility, will not even have commenced. Therefore, as respects the poisoned condition of a patient with cholera, plague, or scarlatina, its phenomena bear no similarity whatever to that of a sheep perishing with rot, or a silk-worm with muscardine; it is anything rather than death arising in the encroachments of local disease."

In contrast with all this, infectious diseases *begin* with fever, or at least evince signs of blood-origin—of *constitutional*, and not local, origin.

These positive as well as negative characters clearly distinguish infectious from all parasitic diseases; and to negative characters we may add the absence of any trace of parasites themselves. Take a piece of small-pox skin, for example; do you find it occupied by parasitic development, animal or vegetable? Certainly not, observes Simon.

Turning to the 'fermentation' theory, we have to inquire how far it is consistent with known facts in the pathology of infectious diseases. "Nothing (thinks Professor Liebig)† can be simpler than this very large question. The morbid-poison-changes in the

---

* General Pathology, 1850, p. 276.

† General Pathology, &c. J. Simon, 1850, p. 276.

blood are fermentative; just such as occur in beer-making. The morbid poison, acting as ferment, may be any organic matter in a state of change. The blood represents the sweetwort. The multiplication of the poison is analogous to the increase of yeast in fermenting liquors; and as this latter increase is contingent on the presence of gluten in the saccharine solution, so the former increase is dependent on the presence in the blood of some specific substance admitting of transformation." This process, extending from particle to particle, at length pervades the whole blood of the individual affected. "It is communicated in like manner to the blood of another individual, to that of a third person, and so on; in other words, the disease is excited in them also." Fermentation boils over, as it were, poisoning the air around, and infecting the blood of others within reach. All this is but an expression of a higher law, long since proposed by La Place and Berthollet, that "a molecule set in motion by any power can impart its own motion to another molecule with which it may be in contact."

Tried by the test of facts, the fermentation theory squares with most of those that characterize eruptive fevers. They are 'infectious' diseases, commonly occurring *once* only in life, *beginning* with fever, and running their course in a *definite* period.

If, then, a substance analogous to gluten in its relation to yeast, did not exist in the blood, the blood-poison would not be reproduced; and the particular disease, be it small-pox, excited by quasi-fermentation, would not be infectious. However, being infectious, we infer the existence of this reproductive substance. And this *reproductive* material, no less than that *analogue* of *sugar* —upon the conversion of which into new products the infectious poison expends itself—must both vary in kind with the particular eruptive fever. The gluten, so to speak, and sugar, of small-pox blood must be different from those of scarlet fever; and so with the other eruptive fevers, for they are severally distinct diseases, although related by family affinities. Furthermore, when those ingredients of the blood upon which the infecting blood-poison acts are themselves exhausted, the individual is protected, temporarily

or permanently, against the *recurrence* of that particular disease to which their existence previously had made him or her liable. And this liability will vary in different individuals according to their specific blood conditions, and in the same individual at different periods of life. The blood of one person is not that of another, although apparently alike; and the blood of youth is not that of age.

The fermentation theory, implying as it does a blood-disease, implies also that fever—the constitutional—precedes the local affection, or eruption: exanthems *begin* with fever. A tolerably definite period of incubation, while fever is *brewing*, and, guided by this analogy, a tolerably *definite* duration also, become intelligible.

Unhappily, however, this theory accords with the properties of yeast in more respects than one. Frothy, as well as fertile of explanation, it is blown away by facts of even greater moment than those by which it is apparently supported.

In the first place, without foundation, this theory is built upon itself. The philosophic Liebig has in this instance argued from an isolated and *exceptional* species of fermentation. He compares the increase of the morbid poison to that of ferment in the production of alcohol. But ferment (with one exception) does not increase, and Liebig deals only with this exception. Yeast certainly increases, because it is an organized vegetable production, and because the chemical changes attending its vital growth are, in the particular case where it is employed, the fermenting influence. The fermenting process may, however, be induced by many other chemical changes. Throw into your saccharine solution a lump of decomposing animal matter, or pour urine into it, and fermentation assuredly follows, yet with no increase of the animal matter or of the urine employed. Increase of the ferment is therefore quite an *accidental* concomitant of true fermentation, occurring only as when yeast, a living growth, is employed to induce the chemical metamorphosis. This, the substance of Simon's leading objection to Liebig's theory, is also, in my opinion, the soundest argument, so

far as it goes, which that pathologist has brought* against this theory. But some one will say, fermentation being possible without reproduction of the ferment, may not the blood, diseased by infection, undergo chemical changes analogous to fermentation without reproduction of the poison? An unqualified negative answer cannot yet be given to this question. An approach to it, however, may be gathered from certain experimental observations made by Simon. To test the fermentability of blood, he repeatedly performed experiments by introducing within the circulation materials in the fullest activity of chemical change, not putrefactive—*e.g.*, saccharine solutions in which yeast was vegetating; starch undergoing conversion into sugar by the action of diastase; solutions of pepsin; pulp of growing animal tissue,—and on no occasion was there a result ever so little like the progress of infective disease, or like fermentation of the blood, induced by such injections.

Ignoring all preconceived theories, Simon advances more cautiously from facts to philosophy. Taking small-pox as a good sample of infective disease, he argues from the import of two facts, considered *in conjunction*. The facts themselves are these:—

The *immense reproduction* of infectious matter witnessed in small-pox; that from a single point of 'inoculation' proceed a crop of pustules covering the whole body, each of which contains matter equally infectious with that of the original pustule by inoculation, itself the produce of an almost imperceptible stain on a lancet charged with this matter.

Then the very suggestive fact, that having once undergone small-pox, the individual is rendered *insusceptible* of a second attack—the blood incapable of this reproduction of matter a second time, or at least for a very long period, if ever again. "You charge your lancet, you make the puncture as before, or you make half-a-dozen—you accumulate all means of infection about the subject of your experiment—but no longer will he give a single sign of the specific infection. You have got from him all the phe-

* Op. cit., p. 278–79.

nomena he can give in answer to that particular reagent. I may compare his condition to this:—Suppose you had a tumblerful of a solution of carbonate of soda, and added an excess of nitric acid; you will get vehement effervescence, more or less, according to the abundance of your dissolved carbonate, and continuing till it has effected complete disengagement of the gas; then, that particular moment having passed, you might add nitric acid *ad infinitum*, but not another bubble would rise. Just so, your patient refuses to *effervesce* any more from new infection, certainly for a long time, perhaps for the rest of his life. You re-apply the same cause that produced the phenomena before—identically the same material—and you get a different result. This fact conclusively proves that a change has occurred in the subject of your experiment; *a change in him* has altered his relations to an *unchanged exterior cause;* by this personal change the poison is rendered inert to him, while it retains its activity towards others.

"We have it accordingly demonstrated that for the production of the disease there must be a *specific internal* as well as a *specific external* condition; that the former is liable to be exhausted, and as it becomes exhausted in the production of material phenomena—namely, in the generation of pustules—this must be a *something material,* like that outward condition with which it co-operates: as the *poison* of small-pox is a something material and tangible, so the *susceptibility** to small-pox is a something material and tangible. Of this material, whatever it may be, no trace remains in the blood when the disease has completed its course.

"Thus we get a general formula for the pathology of small-pox, which is useful as a standard of comparison for the other poisons in our list. A certain organic material, A (soluble and partially volatile, as shown by infection), affects particular relations with B, an ingredient (apparently a normal ingredient) of the blood; the

---

* Correctly speaking, *implies* a something material and tangible. 'Susceptibility' signifies only a capability of undergoing this or that change, and is nothing material.—? F. J. G.

results of their coming together are, (1) the utter destruction of the latter, B; and (2) the immense increase of the former, A; not, indeed, at the spot of infection, but elsewhere. On the one hand, the *virus* augments so much that it is found all over the body, forming innumerable pustules, and contaminating the breath with its volatile miasma; on the other hand, the inward natural *ingredient of the blood* simultaneously diminishes, and at the end of the process is found totally exhausted.

"Look at this as a chemical experiment:—You add A to B; presently you find that B has vanished, and that A has undergone an immense augmentation. What is the meaning of this? What has become of B? Whence has the new A been derived? It is difficult to avoid the conviction, which arises with almost logical certainty, that the increase of one material, and the decrease of the other, have stood in essential mutual relation; that, in short, it has been a process of *conversion;* that the essential relation of the two matters (that derived from *without,* and that contained *within,* the blood), has consisted in the ready convertibility of one into the other; that the specific power of the virus is its power of effecting this transformation, and no other."

Simon thus enlarges this argument:—

"The line of argument which I have followed in regard of small-pox, leads to very similar, though not identical, results in respect of measles, scarlatina, typhus, glanders, plague, and probably hooping-cough; there is the same evidence that a certain definable state of the blood is one of two conditions for the formation of the disease; that this preparatory and permissive state (different and characteristic for each separate infected disease) is a peculiar chemical state, dependent on the presence or the excess in the blood of a material *convertible into identity with the poison;* that the poison, thus augmented, endeavours to eliminate itself by surfaces, the choice of which is a distinctive and specific mark of each poison respectively; that, for a greater or less time after the fulfilment of this eliminative process, the susceptibility to the disease is exhausted; and finally, that the severity of the disease

in each instance will depend, not on exterior circumstances, but on interior and personal conditions; not on any variation in the degree or amount of foreign infection (so long only as this has actually occurred), but on the patient's own possession, within the stream of his circulation, of a larger or less abundance of that specific material which, as I have argued, constitutes* on the one hand his susceptibility to infection, and on the other his power of expressing the disease. He must necessarily evolve symptoms in proportion to his richness in that which furnishes the material."

Having established this position by the argument of strong *analogy* with known facts, Simon ventures to suggest what that blood-material is which confers the susceptibility of infection; here also being guided by known facts appropriately applicable. Observing small-pox, it must be obvious that the material in question, though a *normal* constituent of the blood, cannot be an *essential* one; for the patient convalescent from this disease, and with no demonstrable trace of that constituent in his blood, returns to at least as good health as he ever enjoyed previously. Again, in a certain very small proportion of persons, this natural ingredient of the blood appears not to be *uniformly* present; for there are persons (apparently undefended by any previous occurrence of the disease or by other means) in whom your original inoculation would have failed; persons who would have shown a non-infectibility by the poison, and who therefore must (at least for the time) have been without that material in their blood which confers susceptibility to this disease.

These facts are also true of *all* infectious diseases. Their predisposing blood-constituents are not essential ingredients of the blood, nor uniformly present. Drawing nearer to the question, Simon continues: "What that material—the principle of infective disorders in the human subject—may originally have been, we are totally unable to say; but, whatever may have been its first

---

* Correctly speaking, *confers* his susceptibility. Nothing material can constitute—*i.e.*, be itself 'susceptibility.'—? F. J. G.

method of generation, we can now confidently speak of it as a possible product of the human body; we know that it is liable to develope itself out of some constituent of the human blood."

"What are these constituents? Observation and argument sufficiently show that the blood-corpuscles and albumen can hardly be the constituents in question; first, because, after death by zymotic disease, they are found without evident alteration, and no considerable change in them could escape notice; secondly, because they are indispensable to life, and their even temporary transformation (if complete) would of necessity be fatal; thirdly, because immunity could never be attained by one attack of any particular disease if it were requisite to exhaust these products, re-exposure to infection would ensure a return of the disease and a reappearance of its phenomena."

"For somewhat similar reasons, we may conclude that the salts are not the elements concerned. Fibrin, and the so-called extractive matters, are what remain; can these be the ingredients in question? Substituting for the chemical phrase, 'extractive matters,' the physiological one—'waste of the tissues,' I am strongly disposed to think an affirmative answer to this question; or, at all events, unhesitatingly to point here as the direction in which accurate pathological investigation may be made with most prospect of success." "For, in the first place, they are matters already in progress of decay, and therefore eminently susceptible of new modification; in the second place, they are inessential to the nutritive processes, and that removal of them from the system which would give immunity from reinfection, might be accomplished without withdrawing a vital ingredient from the blood; in the third place, only of such matters as these can it be said that some of them occur but once in life."

"In infancy, in early age, and till puberty, there are certain waste materials which never afterwards occur; the temporary cartilages have to waste away, the thymus gland has to decay, peculiar changes referrible to the sexual system have to be accomplished, and the effete products of these changes have to be eli-

minated from the system. And observe that the surfaces and organs most prone to affection in the diseases under consideration are those which are eliminative and defecating; those whose normal products can hardly be retained for any time within the body, much less out of it, without undergoing a fetid decomposition, which sufficiently stamps them with an excrementitious character. Bowels, skin, kidney, tonsils, are the favourite resorts of the several fever-poisons, just as they are the surfaces by which naturally the organic waste of the several tissues is eliminated. And it may not be amiss to notice that, whereas the normal and healthy discharge of these substances commonly tends to occur in the highest attainable form of oxidation; and whereas, under a variety of atmospheric circumstances interfering with their efficient oxidation, they must tend to accumulate in forms more susceptible of fetid decomposition; so it is peculiarly under such circumstances—where ventilation is defective—where human beings are unduly crowded—where the air is loaded with deoxidizing influences—that zymotic diseases tend to affect the system, either through a new generation of their poison, or through some vast increase of susceptibility thus engendered."

Such is Simon's theory of the blood-origin of infectious disease, in its widest sense. To illustrate how different *kinds* might thus arise:—"On inquiry, it might appear that the relations of infective material to these natural products are definite and constant; that one—let us, for instance, say syphilis—would stand in the particular relation to fibrin; it would be obvious that such an one would be of almost universal inoculativeness, and could only for a very short time, if at all, exhaust the patient's susceptibility to reinfection; and that a drug having certain relations to fibrin (mercury, for example) would interfere with the affinities established by this disease. It might appear that another material, having its origin in the organic waste of nervous substance, would constitute the liability, say, to typhus; such an origin would almost fix the circumstances increasing our proneness to that disease, as well as prefigure the symptoms attending it. Of

another material, it might appear that it originates in the infantile decay of temporary cartilage, or of thymus—a decay occurring only once in life; that such material would constitute the susceptibility to measles or hooping-cough, a single attack of which commonly exhausts the patient's susceptibility for ever. Of a fourth material, it might appear that it arises in those changes of blood which attend the inflammatory and reparative processes under direct atmospheric influence (as in open wounds, cutaneous or mucous), and that in such a product would consist the humoral liability to erysipelatous infection, and to puerperal fever."

This theory contemplates the evolution of 'infectious' disease in its most extended signification, as including those 'contagious' affections also which engender blood-disease rather than remain local affections. Itch is purely local; primary syphilis, erysipelas, and puerperal fever, thus rank with blood-diseases. But this 'waste-tissue' theory *more* particularly accords with those general facts which characterize 'eruptive fevers.' For, having the kind of blood-origin thereby assumed, the probability of these diseases being communicated by the breath, *i.e.* by infection, becomes apparent; so also, that they will occur, usually, but *once* in life; each at that period when the particular waste-tissue, predisposing, is present in the blood; that they will all *begin* with fever, and continue, each for its own *appointed period,* while that (particular) *débris* is being eliminated from the system.

These pathological peculiarities are likewise intelligible by the fermentation theory; and, in truth, there is more analogy between the theories of Simon and Liebig than at first sight appears.

Both theories presuppose an external material agent, and an internal material something mingled with the blood, on which the former acts *chemically,* and reproduces itself, more or less abundantly, at the expense of this blood-ingredient, which thence becomes exhausted. But the one theory regards this process as 'fermentation,' the other as 'conversion'; and Simon suggests that the blood-ingredient is possibly or probably 'waste-tissue' of various kinds.

I have dwelt the longer on this latter view, because it is an instructive example of that reasoning by which all theory should be cautiously constructed. This waste-tissue theory is also one of deep pathological interest in reference to the law of histological "structural retrogression" (p. 93). For if eruptive fevers do not represent rudimentary conditions of the blood, either by arrest or relapse of its own development, yet Simon's theory refers these diseases to the decay of various textures in the order of organic development; and considering the intimate *relation* of the blood thereto, it may, I think, be appropriately designated the theory of 'developmental blood-conditions,' and a further illustration of general *pathogeny*.

Chemical analysis has hitherto failed, for the most part, to detect the 'morbific matter' in the blood, or the altered relative proportion of its normal constituents in any one of these blood-diseases.

Respecting eruptive fevers, the following *general* conclusions issue from the investigations of Andral and Gavarret:—

In many exanthems, the composition of the blood differs but little from that of health; when, however, the disease is somewhat prolonged, the globules and albumen undergo a slight decrease. In a certain number of the pyrexiæ, especially in typhoid fever, and of adynamic type, the fibrin is considerably diminished in quantity; consequently, the blood becomes more fluid and less coagulable. 0.9 represents the smallest proportion of fibrin that Andral and Gavarret found under these circumstances. The quantity of fibrin is never increased in eruptive fever, unless complicated with one or other of the phlegmasiæ of a certain extent and severity. In some cases of eruptive fever the disease has an hemorrhagic character from its very commencement. This is due to a diminished proportion of fibrin. In such cases, the fluidity and diminished coagulability of the blood are likewise marked characteristics.*

Taking these diseases *individually*, what has chemical analysis

---

* Pathological Chemistry. Becquerel and Rodier, p. 117.

disclosed? Little, if anything, peculiar to any one—little, if anything, characteristic.

In Typhus, the blood undergoes no change which can justify us in terming this disease a *dyscrasia*. From the fifth to the eighth day, and therefore nearly as long as the typhus exanthem continues, the blood's composition is very similar to that of plethora, —*i.e.*, the corpuscles are increased, as also are the solid constituents of the serum, and especially the albumen; even the fibrin is generally augmented at this period. From the ninth day great changes ensue; the blood becomes lighter, chiefly from a diminished quantity of corpuscles; the residue of the serum also diminishes daily throughout the entire duration of this disease, with a rapidity proportional to the intensity of the intestinal affection. Such is the statement of Lehmann.* But, alluding, as he does, to an *intestinal* affection in typhus, obviously this disease is confounded with Typhoid fever.

Simon speaks of "Typhus abdominalis,"†—an expression which may, I think, fairly be paraphrased Typhoid Fever,—and of this disease, that its most characteristic blood-condition is a *decrease of fibrin*, proportionately to the violence of the attack; from which also is derived another character, an increased amount of globules. During the early period, this diminution of fibrin is only relative to the globules; as the disease approaches its height, the diminution becomes absolute. Simon much reduces the value of these results concerning typhoid blood, by adding, that "the statements regarding its qualitative and quantitative composition are still very contradictory; arising probably, in part, from its varying in different stages of this disease." The same is also true of "petechial typhus."

The following general conclusions are drawn by Becquerel and Rodier:—At the onset of Typhoid fever, rarely any modification of the blood is observed. Sometimes, however, in very severe cases, accompanied by hemorrhage and great prostration, there is a

---

* Physiological Chemistry. Translated by Day, 1853. Vol. ii.

† Animal Chemistry. Translated by Day, 1845. Vol. i., p. 288.

simultaneous decrease of the three principal elements of the blood, and especially of the fibrin. When the disease is fully developed, the globules and albumen diminish under the influence of low diet, and the evacuations,—diarrhœa, epistaxis, &c., which the patient usually experiences; the fibrin remains unchanged, or tends to diminish in proportion to the advance or aggravation of the disease. In very severe adynamic typhoid fever the fibrin is most diminished.

Respecting the blood in Typhus; experimental observations in six cases gave the following results:—Its specific gravity is generally much reduced. The proportion of globules, which in two cases (males) remained unchanged, was greatly diminished in two others; the same occurred in one case of a female. These differences must be explained by the previous condition of the patient, by his diet and regimen, and especially by the anæmic condition present; for this reduction of the globules is not due to the disease itself. The fibrin either remained unchanged in quantity, or fell below the limits of health—a tendency worthy of notice. The specific gravity of the serum was generally much diminished; this diminution being, in all probability, occasioned both by the disease and its dietetic treatment; it was, however, much more marked in one case than in others.

I may here advert to certain *physical* peculiarities which are said to characterize the blood in typhus and typhoid fever;—that it is semi-fluid, and that the slight clot which eventually forms is soft, friable, and of a black colour. It becomes putrid sooner than healthy blood. To the same effect Dr. Clanny long since remarked, that the blood, very dark, flows sluggishly when a vein is opened; that it coagulates slowly, and seldom acquires any buffy coat; that these characters become more marked as the disease advances; and that at last, in bad cases, the blood coagulates so loosely as to be tremulous and brittle, almost resembling ill-made currant jelly.

The blood in Relapsing fever has not, so far as I am aware, hitherto been subjected to analysis.

In Measles, Andral and Gavarret analysed the blood of seven patients. In all, the fibrin remained within its normal limits,—3·6 to 2·6. The globules were increased in four cases to 146·9, 140·6, 137·0, 137·1, and in another to 123·9. In two cases they were diminished to 118·6 and 116·1. The lowest proportions were found in those cases where the bleeding had been practised at a somewhat remote period from the commencement of the disease. Three analyses, however, made by Becquerel and Rodier, gave proportions nearly those of health.

In Scarlatina, the blood was analysed by Andral and Gavarret, in two instances only. The proportion of fibrin remained perfectly normal, while that of the globules was greater than in health.

In Small-pox and "varioloid disease," seven analyses were made by Andral and Gavarret. Of these, five were of the blood in small-pox (confluent), and two only pertained to the varioloid disease. The former showed no deficiency of the globules, except in one case. The amount of fibrin, which somewhat increased at the first bleeding, invariably underwent a decrease subsequently. Perhaps this primary increase of fibrin accompanied the inflammatory state of the skin.

In varioloid disease, no modification of the blood was discovered.

Varicella has not yet received chemical investigation.

Erysipelas has been thus examined, yet with little result. The blood is rich in fibrin, and poor in globules; but these alterations, together with others that occur, are common to all the phlegmasiæ. They are not peculiar to erysipelas, and therefore not characteristic. The circumstances of therapeutic treatment, age, &c., seriously affect the results of any analysis obtained in respect of this, as well as of *all* blood-diseases.

Hooping-cough and Influenza have both been quite overlooked by blood-pathologists; while Cholera, the last of the group we are

considering, has been examined by a host of scrutineers, and more satisfactorily than any other infectious disease.

Simon, who first analysed the blood in this disease, found the proportion of water diminished, and that the solid constituents had undergone a remarkable increase; at the same time, the proportion of fibrin was augmented, and the urea had accumulated. This latter change has not been verified by subsequent analyses.

Wittstock found the proportion of solid matters 137·5 in 1000.

Heller examined the blood after death from cholera, and found it rich in albumen, fatty matters, and urea. It contained also a large proportion of chlorides, but the fibrin remained unchanged in quantity.

Becquerel and Rodier conducted a complete analysis of the blood, in two very severe cases of cholera, and four analyses of the serum, under similar circumstances, with the following results:

The density of the serum is much greater than in health; the proportion of water is reduced,—that of solid matters, on the whole, much increased. Taking particular ingredients, the albumen varies but little,—if anything, it rather tends to diminish; chloride of sodium is considerably increased, insomuch that its proportion is nearly one-third more than usual; this ingredient, therefore, is concentrated in the blood; fatty matters are nearly doubled in quantity, and extractive matters are very abundant.

Analysis of the blood gave similar results, and a very large proportion of globules.

Guided by these results, and more especially when placed in juxtaposition with those supplied by analysis of the alvine evacuations and vomited matters in cholera; (it is justly argued* that) we can somewhat explain the phenomena of this disease. That, in fact, a portion of the solid matters of the serum, particularly albumen, is exhaled by the gastro-intestinal mucous membrane. The blood becomes less fluid also, and the dejections more watery;

---

* Pathological Chemistry, cit.

this efflux of water being necessarily accompanied with concentration of the globules, extractive matters, chloride of sodium, &c.

Analyses made by Dr. Dundas Thomson* exhibit the altered constitution of choleraic blood even more clearly. The relative composition of blood in cholera and in health may be stated as under :—

	Cholera.	Health (Lecanu).
Water	717·8	790·00
Fibrin	4·5	2·95
Globules and albumen .	268·8	199·55
Insol. salts	1·8	1·00
Sol. salts	7·1	6·50
	1000·0	1000·00

On comparing these columns, we gather the following conclusions respecting choleraic blood :—

The water is much diminished.

The total amount of other constituents, organic and inorganic, is increased.

The ratio of the inorganic to the organic is nearly that of health.

But of the former the ratio of the soluble to the insoluble salts is considerably disturbed ; so that although the total amount of all the saline matters exceeds that of health (in the ratio of 1·4 to 1), the quantity of soluble salts is less *in proportion* to the increase of insoluble salts.

The preponderating insoluble or earthy ingredients are phosphates of lime, magnesia, and iron ; the relatively deficient soluble salts are chlorides of sodium and potassium, triphosphate and sulphate of soda.

This deficiency, with the reduced proportion of water, together represent the disturbed equilibrium of the blood in cholera, as

* Chemical Researches on the Nature and Cause of Cholera. Med.-Chir. Trans., 1850.

compared with that in health. The proportion of constituents wanting is found in the alvine evacuations and vomited matter, both of which Becquerel and Rodier correctly describe briefly as being "nothing more than water, containing a small quantity of albumen, and a large amount of chloride of sodium."*

While, therefore, 'common salt' and the other soluble salts are undoubtedly *concentrated* in the blood, by draining off its water per alvine evacuations and vomiting; these salts, in solution, are thus actually washed away, leaving the blood *proportionately* deprived of their vital influence. And this *rationale*, guiding and regulating, as it does, the (rational) therapeutic treatment of cholera, singularly accords with that remedial measure which (therapeutic) experience has shown to be most valuable. The following life-restoring draught, first prescribed by Dr. Stevens, has been generally administered by other practitioners:—

℞ Carbonate of soda, ʒss.
Chloride of sodium, ℈j.
Chlorate of potash, grs. vij.
Water, half a tumbler.

This draught, repeated every half hour, more or less, according to the 'collapse,' restores that proportion of those ingredients which the blood has lost; and with such restoration the thick and heavy blood liquefies, its sluggish circulation quickens apace, the body regains its natural warmth, colour, and plumpness, and the nervous system revives. Nothing more like a resurrection can be witnessed.

But surely no one will imagine that any *altered proportion* of certain *natural* constituents of the blood, in cholera, is the *primary* and therefore essential change which the vital fluid undergoes in this disease. Diminished proportion of water,—increased proportion of such and such ingredients; can it be by virtue of any alteration in the *proportion* only of the constituents of healthy blood that cholera arises? Nay, more, that by so many alterations in the

* Op. cit., p. 458.

proportion of the *same* natural constituents the whole tribe of infectious diseases are evolved? Assuredly not. The blood-origin of all these diseases must be sounded with a deeper line than that by which it has yet been fathomed,—by a far more subtle and searching 'organic analysis,'—ere this grand consummation of blood-pathology can be attained.

For, to say nothing of the discordant results of analyses hitherto made by different observers, there are at least two general facts which clearly point to some primary and essential change in the blood's chemical composition and vital powers, as the ultimate source of all those (subsequent) phenomena that apparently constitute the particular disease. These general facts are—

1. The improbability that diseases, the obvious characters of which are so constantly distinct,—as those, for example, of cholera and small-pox,—should arise from conditions of the same blood, differing only in the *proportion* of its ingredients.

2. The impossibility of such alterations being the primary and essential change which the blood undergoes; for they are only detected *after* the particular disease is unquestionably declared by its own appropriate symptoms; cholera by its rice-water evacuations, small-pox by its eruption, and so on. The blood is examined *in* cholera, *in* small-pox, &c.; not *prior* to the manifestation of these diseases—*i.e.*, in the period of incubation, when indeed, being latent, no suspicion of their generation suggested such inquiry.

The altered proportions of the blood's natural constituents, in the various kinds of infectious disease, are therefore so many *symptoms* only of that primary and essential condition from whence these diseases arise.

Now this *essential something* has hitherto altogether eluded detection. As a blood-condition, it must be distinct and specific in each kind of infectious disease, and therefore peculiar to *each* kind; for the offspring bespeaks distinct species, the fruit distinct stock. Beyond this generalization, however, at present all is darkness.

Passing on to the question of Prevention—how far is the prevention of infectious disease now capable of being fulfilled; or, in a more advanced state of blood-pathology, may it yet be accomplished?

The importance of this question cannot be over-estimated. It is not merely that prevention is always better than cure, but that, as regards 'infectious' diseases, the former is possible, the latter is not. Each runs its own course, and all we can hope to accomplish by any therapeutic interference is to conduct the disease to a favourable issue, by obviating the tendency to death. Prevention, therefore, far surpasses cure, and its significance in relation to infectious diseases assumes the largest dimensions when estimated by their mortality. The prevention of these diseases becomes a matter of National concern, and of State importance.

What guidance, then, does Pathology afford?—how far does it suggest preventive measures?—and what are our future expectations from this source?

Adverting to the leading character of all 'infectious' disease,—that of self-propagation by 'inhalation,' and therefore by social intercourse,—this general preventive measure is at once suggested;—to *preclude* every mode of communication between the sick and healthy: removal therefore of infected persons, exclusion of the non-infected as much as possible, and total cessation of communication between them, or by any fomites, in the shape of clothing, &c. By observing this precaution infection may be arrested.

This preventive measure is recognised and fulfilled in various ways.

The removal of any infected member of a family to an attic, with exclusion of the other members, and of all inquiring friends—the temporary banishment of a schoolfellow from his playmates—the removal of infected sick to special institutions, *e.g.* special hospitals for fever, and of thus weeding out sources of infection from *crowded* neighbourhoods, and intercepting *close* personal communication. All these private and public regulations alike concur to prevent the spread of infectious disease among a com-

munity; while 'quarantine' is a similar preventive measure internationally.

These precautions should be *regulated* by the duration of the 'latent period' of the infectious disease.

Otherwise a person in apparent health may convey infection to another, and, in these days of rapid travelling, distant locality, without suspicion. Or again, the period for excluding suspiciously infected persons from communication with the healthy may be unnecessarily prolonged. The old regulations concerning quarantine blindly erred in this respect.

The period of latency, or incubation, varies in each kind of infectious disease; but the following averages are tolerably correct:

In typhoid fever, the latent period ranges from three to fourteen days; the medium duration of latency being ten days, according to Gregory's observations.

Respecting typhus, this period has not been distinguished from that of typhoid fever; these two kinds of fever having been formerly regarded as identical.

In relapsing fever, the period of incubation has not been observed.

In measles, this period generally extends from seven or eight to fifteen or twenty days.

In scarlet fever, from one to twenty or twenty-five days.

In small-pox, from six to twenty or twenty-one days; twelve days being its average duration (Gregory).

In erysipelas, latency extends from two to fourteen days (R. Williams).

In Asiatic cholera, from one to six days.

Equally important, in relation to prevention, is it to know the period during which an infected person retains the power of *communicating* his disease to others. This period is obviously uncertain, owing to the hygienic condition of the individual from whom the poison emanates, and that of the person to whom it may be communicated.

Probably, however, a fortnight after the commencement of

recovery from most infectious maladies, their infectious character ceases. But fomites, in some instances, remain infectious for a very long period. When speaking of scarlet fever, I gave an instance in which a piece of flannel retained the power of infection after the lapse of at least a year.

No less subservient to our present purpose is the 'infecting distance' of each of these diseases, especially in regulating the distribution of beds in hospitals and other institutions where persons are necessarily contiguous.

In typhoid fever, three feet around the patient's person may be said, under circumstances of ordinary precaution, to ensure exemption from infection (R. Williams). Perchance this statement has reference to typhus.

In relapsing fever, the infecting distance is unknown.

In measles, little is known accurately on this point, but the disease certainly spreads as widely and freely as scarlet fever; and this disease is disseminated extensively.

In small-pox, the infecting distance, although not absolutely determined, is probably very considerable, not less than from thirty to fifty feet.

In varicella, the circumferential range is probably very limited, for the extension of infection is easily controlled.

Erysipelas spreads around from twenty to thirty feet, and is therefore far more infectious than typhus (Williams).

In hooping-cough, the range is at least as extensive as that of exanthems.

There being, then, certain tolerably *definite* limits, beyond which infection emanating from one person as a centre loses its power over those around, are there any means by virtue of which the poison in this area can be so diluted or neutralized as to destroy its power also within the sphere of its operation? The question of 'disinfection,' in fact, is suggested, and it more immediately concerns those persons who are unavoidably brought near to the sick. Nurses and medical attendants, therefore, are thus concerned, and through them the community at large.

Now, *dilution* of the poisonous effluvia by free ventilation is one mode certainly of disinfection.

Any other means, the object of which is to *neutralize* or *kill* these animal poisons, are far less efficient. Of such means, heat (not warmth) and cold alike render these poisons inert. By a high temperature of 200 degrees, continued for at least an hour, the emanation from scarlatina loses its power.*

A host of *decomposing* and *deodorizing* specifics have been used to neutralize infection. Fragrant spices of the East, in times gone by, were employed to sweeten, if not to purify, the sick-chamber. Chemical disinfectants, as they are called, have been and are still appealed to. Chlorine was first introduced with this intent by M. Fourcroy in 1791. The Millbank Penitentiary was thus cleansed, under the sagacious directions of Faraday.† The irritating property of chlorine itself is now veiled by using it in the form of chloride of lime, and chlorinated soda, as first suggested by M. Labarrague.

But in whatever shape this and other reputed disinfectants of a similar kind are employed, their protective (and preventive) power is very doubtful. They certainly destroy or overwhelm noxious smells; yet probably, like the aroma wafted from burning spices —from the camphor bag, in domestic use, or from the time-honoured rue strewed about the dock of our criminal courts; all these vapours only delude the sense—they deodorize rather than disinfect.

More protective, I should say, is that fearless state of mind which accompanies the sense of duty in attending the sick; that calm feeling of resignation inspired by the confidence that all things happen for the best, whether to escape or to suffer any prevalent disease. This of course does not imply reckless indifference to the employment of those rational precautions for avoiding infection already adverted to.

---

* Phil. Mag. and Annals. Nov. 1831, and Jan. 1832.

† Journ. of Sciences and Arts, vol. xviii.

How far *these* preventive measures have proved efficient, can only be estimated by their results.

Take the aggregate mortality from infectious diseases, in each of a series of years, as compared with the *total* mortality from all other causes combined, in each year of the same period. For example, take the aggregate mortality from three exanthems—measles, scarlatina, and small-pox—in each of three consecutive years, as compared with the total mortality in England and Wales, in each of the same three years ; and the results of these statistics show a very large proportion of deaths by infectious disease. The following tabular view clearly exhibits this *aggregate* ratio, and also the ratio of these diseases, *severally*, to the total mortality :—

	Year 1838	Year 1839	Year 1840
Measles	6,514	10,937	9,326
Scarlatina	5,802	10,325	19,816
Small-pox	16,268	9,131	10,434
Aggregate Mortality . . .	28,584	30,393	39,576
Total Mortality in England and Wales	342,529	338,979	359,561

This fatality ranged, in London alone, as follows, during the same three years :—

Aggregate Mortality . . .	5,929	5,169	4,321
Total Mortality	52,698	45,441	46,281

Passing over a period of nine years, during which preventive measures, such as I have mentioned, had been duly recognised in this country ; do we find the number of deaths from infectious disease diminished, as compared with the total mortality ? Take again the three diseases just examined, and observe the proportionate mortality therefrom in 1850 and nine consecutive years, in England and Wales.

	1850	1851	1852	1853	1854
Measles ...	7,082 ...	9,370 ...	5,846 ...	4,895 ...	9,277
Scarlatina ...	13,371 ...	13,634 ...	18,887 ...	15,699 ...	18,528
Small-pox ...	4,665 ...	6,997 ...	7,320 ...	3,151 ...	2,808
Aggregate Mortality	25,118 ...	20,001 ...	32,053 ...	23,745 ...	30,613
Total Mortality ...	368,995 ...	395,396 ...	407,135 ...	421,097 ...	437,905

	1855	1856	1857	1858	1859
Measles ...	7,354	7,124	5,969	9,271	9,548
Scarlatina ...	17,314	14,160	14,229	30,317	19,907
Small-pox ...	2,525	2,277	3,936	6,460	3,848
Aggregate Mortality	27,193	23,561	24,134	46,048	33,303
Total Mortality ...	425,703	390,506	419,815	449,656	440,781

Guided by these statistical results, extending over a series of years, it must be acknowledged that the *proportionate* number of deaths from infectious disease is *declining;* and by observing the proportion which *each* of the three diseases tabulated bears to the whole, it appears that *small-pox*, more especially, is becoming *less* and *less fatal.* In fact, the diminished aggregate mortality from infectious disease, compared with the total mortality, is not so much due to a diminished proportion of deaths from measles, and still less the decline of scarlet fever, as that the proportionate mortality from small-pox is declining, and the absolute mortality from this disease is also declining.

Compare the number of deaths in the three years, 1838, 1839, and 1840, with those after an interval of nine years, in 1850, and also with those in the next nine years consecutively. A descending scale, with few exceptions, is observed.

Or again, place this scale in apposition with the yearly mortality from typhus and typhoid fever during the same period, say from 1850 to 1859 inclusive.

	1850	1851	1852	1853	1854
Typhus and Typhoid Fever...	15,374	17,930	18,641	18,554	18,893
Small-pox	4,665	6,997	7,320	3,151	2,808

	1855	1856	1857	1858	1859
Typhus and Typhoid Fever...	16,470	16,182	19,016	17,883	15,877
Small-pox	2,525	2,277	3,936	6,460	3,848

This series shows that the deaths from typhus and typhoid fevers have certainly fluctuated—rising and falling; but have never once fallen below the number in 1850, and concluded with very nearly the same mortality as in that year; while the deaths from small-pox have certainly declined—rising only occasionally with epidemic prevalence.

The persistence of an appalling yearly mortality from most other infectious diseases brings home their vast importance in a Preventive point of view, by which alone this sacrifice of human life can be mitigated, and possibly, at last cancelled from the Registrar-General's Annual Return.

The national importance of Prevention will be realized more fully by comparing the annual mortality from all infectious diseases, with the number of deaths annually from *all* causes *minus* infectious disease.

	1850	1851	1852	1853	1854
All causes	368,995	395,396	407,135	421,097	437,905
Infectious Diseases ...	78,280	90,002	96,107	89,448	117,088
All causes, less Infectious Diseases	290,715	305,394	311,028	331,649	320,887
	1855	**1856**	**1857**	**1858**	**1859**
All causes	425,703	390,506	419,815	449,656	440,781
Infectious Diseases ...	88,152	81,121	93,518	110,971	106,645
All causes, less Infectious Diseases	337,551	309,385	326,297	338,686	334,136

To compensate in some measure for this sad loss of human life—and of national productiveness therefore—year by year, is the cheering fact that *small-pox* is waning; and the decline of at least this one source of infection—this shadow of death quitting every home—is watched with even deeper interest, and assumes a more suggestive significance, by virtue of its *exceptional* character in the annals of infectious disease. The destroying angel has been expelled by Pathology. Yes; observation of the insusceptibility and immunity enjoyed by those persons who had (accidentally) undergone another disease, and which, therefore, apparently conferred that immunity,—this observation first suggested to Edward Jenner, of immortal memory, a method of prevention, the *principle* of which may eventually admit of far wider application. Small-pox has been vanquished by the *substitution* of another disease—vaccinia, irreconcilable, as it were, with the supervention of variola. The *susceptibility* to small-pox, in the same individual,

is thereby exhausted, for an indefinite period at least,—possibly, for there mainder of life. But the *practical* value and blessing of this discovery hinges on the fact, that this substitute—vaccinia—is a trivial and temporary indisposition only, in exchange for a foul and fatally prevalent disease. By the knowledge and wide-spread application of this pathological fact, small-pox is now being expelled from all civilized communities; and foremost among preventive measures—more protective than the preclusion of every mode of communication between those who are *already* infected, and those who may yet be healthy, ranks prevention by 'vaccination.'

The grand results of this anticipatory measure are precious records in the history of Preventive Medicine.

Simon's Report on Vaccination, by order of the General Board of Health, and presented to both Houses of Parliament by command of her Majesty—is the most noble triumphal arch that State Medicine has hitherto erected over the pathway of Human Progress. Let us, then, gather a few evergreen leaves from around the basis of this imperishable monument. And first of all, in honour of our own country.

"Dr. Lettsom, in his evidence before the Parliamentary Committee of 1802, stated reasons for estimating the small-pox death-rate of England at about 3000; and Dr. Blane's evidence was nearly to the same effect." Simon's statistical results lead him to believe that such an estimate by no means exaggerates our average losses *before* the discovery of vaccination.

In contrast therewith, Simon shows that for the years 1841-53, the average small-pox death-rate of England and Wales was only 304; in 1854, only 149; in 1855, only 132; and it is reasonable to believe that even these death-rates will be more and more reduced, as vaccination becomes more invariably practised.

Confining our attention to London alone, the following table exhibits the proportionate mortality from small-pox, in every one thousand deaths from all causes;—during fifty years, with no

protection; fifty years with inoculation; and forty years, with vaccination; each being estimated in successive decennial periods.*

Periods.	Small-pox Deaths per 1000 Deaths.	Comparative Numbers.	Description.
1650—1660	48		
1660—1670	36		
1670—1680	71	56	No Protection.
1680—1690	74		
1690—1700	51		
1750—1760	100		
1760—1770	108		
1770—1780	98	96	Inoculation.
1780—1790	87		
1790—1800	88		
* *	*	* *	* *
1810—1820	42		
1820—1830	32		
1830—1840	23	29	Vaccination.
1840—1850	18		

The decennial period 1800-1810 is omitted, as being one in which both inoculation and vaccination were practised.

From the same document we learn (p. 26) that the frequency of small-pox epidemics in London was—

Before Protection, as	42
During Inoculation, as	54
During Vaccination, as	14

Extending our view to foreign countries, and guided by Simon's Blue-book (p. xxiii.), we observe the *universal* decline of small-pox since vaccination has been *co-extensively* practised. Thus, its fatality in Copenhagen is but an *eleventh* of what it was; in Sweden, little more than a *thirteenth;* in Berlin, and in large parts of Austria, but a *twentieth;* in Westphalia, but a *twenty-fifth.* In the last named, there now die of small-pox only four persons, where formerly one hundred died.

These and similar results, on the largest scale, amply demon-

---

* Report on Small-pox and Vaccination, by a Committee of the Epidemiological Society. 1853.

strate the declining proportions of small-pox mortality since the practice of vaccination has prevailed.

But does this disease never occur *after* vaccination? Exemption for an indefinite period, possibly for life, is the rule,—post-vaccinal small-pox (modified small-pox, varioloid disease,) the exception; and when the disease does occur in this form, it is of shorter duration, without secondary fever, and far less fatal!

"Sufficient proof (of this position) is accorded by those public establishments—army, navy, and schools—in which it is the rule to vaccinate on admission all unvaccinated subjects who do not bear marks of previous small-pox." For example, "Dr. Balfour* shows that the small-pox mortality of the British Navy has not reached a third, nor that of the British Army a fourth, of the London rate; and that at the Royal Military Asylum, during forty-eight years—within which period 5774 boys were received for training—only four deaths by small-pox occurred, and *these all in non-vaccinated boys,* who probably had undergone the disease prior to becoming inmates of the school. In two severe epidemics of small-pox which prevailed in Malta, in the years 1830-1 and 1838-9, the death-rate of the general population was just twenty times that of the military population."†

The rare fatality of post-vaccinal small-pox is demonstrated on a still larger scale by "the Government Statistical Reports of the sickness, mortality, and invaliding among her Majesty's troops,"‡ extending from 1817 to 1836 inclusive. During this long period the Dragoon Regiments and Guards, with an aggregate strength of 44,611, and a total mortality of 627, lost only three men by small-pox. The troops at Gibraltar, with an

---

* On the Protection against Small-pox afforded by Vaccination, illustrated by the Returns of the Army, Navy, and Royal Military Asylum." Med.-Chir. Trans. Lond., 1852.

† See also Wunderlich's Handbuch der Pathologie und Therapie, vol. iv. p. 207.

‡ Edin. Med. and Surg. Journ., vol. lxiv., 1845. Inquiry, &c., by James Stark, M.D.

aggregate strength of 60,269, and a total mortality of 1291, lost only *one* by this disease. In the West Indies, the British troops, with an aggregate strength of 86,661, and a total mortality of 6803, had not even one death from small-pox; though several epidemics of this disease ravaged the islands in the period referred to, of twenty years. The native troops also, on the same station, with an aggregate strength of 40,934, and a total mortality of 1645, completely escaped: so likewise at Nova Scotia, at New Brunswick, at the Cape of Good Hope, and at the Mauritius, not one death from small-pox occurred during these twenty years; and even the white troops of Western Africa wholly escaped, while hundreds of the black *unprotected* population were dying.

In Malta, the British troops, from 1818 to 1836 inclusive, had an aggregate strength of 40,826, and a total mortality of 665; only two deaths, however, occurred from small-pox; but among the civil (unvaccinated) population, at Malta, the disease appeared as an epidemic in 1830, and continued during the whole of that and the succeeding year, carrying off no fewer than 1169 persons; in the proportion of 1048 deaths out of a total mortality of 3407 in the year 1830, and 121 out of a mortality of 2581 in 1831.

These facts plainly testify the *almost perfect* protection guaranteed by vaccination; for while small-pox was raging so fatally among the (unprotected) Maltese, that a *third* of the whole mortality was due thereto in 1830 alone, only two fell victims to its severity among the (protected) military during the extended period of eighteen years.

Equally conclusive evidence to the same effect is afforded by the statistics of small-pox included in this report, respecting Ceylon and Madras. In addition to these results of statistics, there is the evidence of individual testimony;—the evidence of persons of reliable credit. The protective value of vaccination has been sufficiently tried, and as sufficiently verified by this criterion also.

Simon issued a circular, containing *four* questions, addressed to five hundred and forty-two members of the Medical Profession, to

departments of the public service, and to foreign governments. An equal number of personal answers were received.

Here, then, were five hundred and forty-two independent witnesses, who voluntarily underwent examination; and their answers constitute a most remarkable body of evidence.

Question 1 was this—"Have you any doubt that successful vaccination confers on persons subject to its influence a very large exemption from attacks of small-pox, and almost absolute security against death by that disease?" This question, therefore, related to precisely that aspect of vaccination which we have been considering;—to the general exemption from small-pox conferred by vaccination; to the possibility, however, of post-vaccinal small-pox, and the extreme improbability of this subsequent disease being fatal. The five hundred and forty-two witnesses replied, as with nearly one accord, in the affirmative to the question respecting these particulars. Only two negatives marred the harmonious concurrence. Of these eccentric individuals, one distrusted vaccination, and "would gladly inoculate his own children with the small-pox;" the other disbelieved the virtue of both inoculation and vaccination.

If, however, guided by all that I have urged, we accept vaccination as a substitute for small-pox, and credit its protective power as a truth firmly established by the joint concurrence of very ample statistics and personal testimony; are there any *evils* possibly engrafted *thereby* or *therewith*, and evil results, therefore, possibly or probably accruing from the most liberal enjoyment of this otherwise inestimable blessing?

Questions 2 and 3, proposed by Simon, are respectively specially constructed with the view to the solution of these issues.

The former interrogatory (2), having reference to the possibility or probability of any disease being *superinduced* by vaccination, runs thus:—"Have you any reason to believe or suspect that vaccinated persons, in being rendered less susceptible of small-pox, become more susceptible of any other infective disease, or of

phthisis; or that their health is in any other way disadvantageously affected?"

The latter interrogatory (3), respecting the possibility or probability of any diseases being *imparted* by inoculation together with the vaccine virus, is expressed in these terms:—"Have you any reason to believe or suspect that lymph, from a true Jennerian vesicle, has ever been a vehicle of syphilitic, scrofulous, or other constitutional affection to the vaccinated person; or that unintentional inoculation with some other disease, instead of the proposed vaccination, has occurred in the hands of a duly educated medical practitioner?"

To both these questions, the most unqualified negatives, expressed or clearly implied, were returned by the five hundred and forty-two respondents.

As to any kind of disease being superinduced by vaccination; most of the replying Governments having made this a compulsory regulation within their dominions, shows that they at least have discovered no drawbacks to its unqualified advantage,—no vicarious diseases to set against the extinction of small-pox. And as to any kind of disease being engrafted by vaccination; among the five hundred and forty-two respondents, not a single one gives the slightest semblance of support to any contra-vaccinal doctrines: very, very rarely are they even referred to.

By question 4, the unqualified preventive value of vaccination is canvassed, so as virtually to embody each of the foregoing questions; the challenge of this one therefore re-elicited replies on the whole. "Do you (assuming due provisions to exist for a skilful performance of the operation) recommend that, except for special reasons in individual cases, vaccination should be universally performed at early periods of life?" To recommend—argues Simon—that, except for special reasons in individual cases, vaccination (skilful of course) shall be universally practised, is to imply that one's mind is made up on all those other matters we have been discussing. Regarding, then, the answers to this question, as for all practical purposes, summaries of opinion on the whole

subject of vaccination; observe, such was the recommendation, with only two personal exceptions—the unanimous recommendation—of every individual and of every Government in the series.

Indisputably established by all this weight of statistical facts and personal testimony, it only remains for me to notice certain particulars respecting the administration of vaccination necessary to ensure its practical efficiency as the most rational preventive measure against small-pox.

Firstly. Vaccination should be performed at an early period of life, not (without exceptional reasons in individual cases) later than the third or fourth month after birth (Watson).

Of the whole mortality from small-pox in England and Wales, no less than twenty-five per cent., or one-fourth, occurs in infants under *one* year old, and as much as eleven per cent. within the age of four months. Under the *fifth* year, this mortality rises to seventy-five or eighty per cent.

The inference from these statistical facts is twofold;—that vaccination should be performed at the earliest period compatible with the safety of the child; and delay being an error at the expense of human life, this safeguard should be enforced by law, rather than left to gratuitous administration. The practice of vaccination should be compulsory, and at a very early period of life, by legal enactment. So it is *now* under the provisions of a recent Act of Parliament, of which the Medical Profession are duly informed.

Secondly. The quality of vaccine lymph demands discriminative consideration.

Losing its protective power by transmission from one person to another through a series of individuals, the lymph should be reinstated from time to time, by fresh supplies procured immediately from its original source—the cow. As, however, this is a moot point, I refer the reader to Simon's report for convincing proofs of its importance. Perhaps the most convincing is this;—observing the declining protective efficacy of vaccination when the lymph has not been renewed, "it is difficult to conceive how

the infantine generations of a country could, crop by crop, successively derive less permanent constitutional impressions from vaccination, unless the efficient cause of those impressions—the vaccine contagion itself—had year by year undergone enfeeblement of its powers."

The test by which vaccination is proved to have been effectual at the time of its administration is simple but conclusive. It was contrived by Mr. Bryce. Having vaccinated one arm as usual, he vaccinates the other after the lapse of four or five days. If the first operation is effectual, the second vaccine vesicle will overtake the first, appearing in every respect similar, only a miniature representation, and both will subside and disappear together. If the first vaccination fails to induce its proper constitutional effect, the second vesicle will run its course, enlarging up to the eighth day, and altogether unaffected by its forerunner.

That the protective efficacy of vaccination *does decline* after a time, and moreover, that it does so in a fast ascending ratio, when the lymph has been borrowed from individual to individual, are facts alike demonstrated by the statistical results of revaccination in the Prussian army. To realize the force of these results, it is necessary to bear in mind, that in proportion as vaccination *takes*, so is the individual susceptible of small-pox. When this system of revaccination commenced in 1833, the proportion of successful results (of those who again took the disorder) was thirty-three in every hundred. Now the annual per-centages of successful results for the whole time during which revaccination has been practised in that army, run thus:—33, 39, 42, 46, 49, 50, 51, 54, 57, 58, 57, 57, 58, 60, 64, 64, 64, 61, 64, 69, 69, 69, 69, 70. The last proportion of success exceeds the double of that with which the series commenced. Supposing the first vaccination to have preceded the second by twenty years, the vaccinations of 1836, tested by eventual resusceptibility to cow-pox, were not half so permanently efficacious as the vaccinations of 1813.

Revaccination, therefore, and with lymph supplied fresh from its original source, is a regulation incumbent on all communities.

I have yet to describe the appearances of the vaccine vesicle when *protective*.

Vaccinia is a vesicular skin-eruption, resulting from the efficient inoculation of the vaccine lymph by puncture, as with a lancet charged with such lymph. Each point of puncture produces a single vesicle, as follows :—

On the third day after insertion of the lymph, the puncture is red and elevated. By the fifth day, the cuticle is elevated into a pearl-coloured vesicle, containing a small quantity of thin and perfectly transparent fluid. The shape of this vesicle is circular or oval, according to the puncture from which it has arisen. On the eighth day, the vesicle is fully ripe ; its margin is turgid and prominent, leaving a depressed centre ; it is *umbiliform*, like that of small-pox. It now measures nearly half an inch across. On close inspection, a cellulated structure is visible. Cells eight or ten in number together form but one. Yet they are distinct, for by puncturing one the rest do not collapse.

At this time also (eighth day) an inflammatory areola begins to extend around the base of the vesicle, or of each, if more than one ; the skin becomes red, tense, and painful ; and this areola enlarges during the ninth and tenth days. On the eleventh day it begins to fade,—leaving, as it declines, two or three concentric circles of a bluish tinge, accompanied with some tension of the corresponding portion of skin, for two or three days more.

The vesicle itself has now either burst, or been opened by the surgeon desirous of procuring a fresh supply of lymph. The remaining lymph dries up, forming a scab of a circular shape, and brown mahogany colour. This crust hardens and blackens, and at length, between the eighteenth and twenty-first day, drops off, leaving a cicatrix, of a form and size proportioned to the prior inflammation.

A perfect vaccine scar should be nearly half an inch broad, circular, having a defined margin, slightly depressed, marked also with radiations and indentations, the latter apparently representing the cells of the parent vesicle. This result is final evidence that

the inflammation was specific, and that it did not advance beyond that degree of intensity whereby the whole mass of circulating blood has become affected;—vaccinia substituted for small-pox. Protection is further assured by slight fever supervening about the eighth day. Many of the most perfect scars disappear entirely in after years.

Characteristic as is the vaccine vesicle, nevertheless it, like every other production in nature, may deviate from its type—such as I have described. How, then, can the formation of this vesicle be relied on as *the* proof of successful inoculation, and subsequent protection against small-pox? By a simple yet decisive test, invented by Mr. Bryce.

If the blood has been duly affected, the characteristic vesicle cannot be reproduced, at least for a considerable period, when the vaccine blood-condition may have become worn out. So that at the time of vaccination, let another part, say the other arm, be vaccinated four or five days after the first inoculation. If vaccinia be accomplished, the vesicle then induced will *overtake* the original one, and disappear with it. If this be spurious, the second will run its proper course of eight days, &c. This again may then be tested by the production of a third.

Respecting the number of vesicles requisite to ensure and prolong the protective power of vaccination; Mr. Marson's long experience at the Small-pox and Vaccination Hospital leads him to believe that four or more vesicles which have left good dotted cicatrices are sufficient.

A few particulars are essential to the successful application of this preventive measure. "Lymph"—says Mr. Marson—"for use, is in its best state on the seventh day of the vesicle—the day week after vaccination. It should be taken when the vesicles are plump, and just before the formation of the areola. Under no circumstances should lymph be taken for use later than twenty-four hours after the areola has begun to form.

"A serious error in vaccinating is the use of blunt lancets; it is impossible to have a lancet too sharp for vaccinating. The lymph should be introduced by a puncture of a valvular shape,

from above downwards, so managed that at each puncture it may gravitate into the wound.

"In this way the lymph may be introduced into five punctures —the number I recommend—from half to three-fourths of an inch apart, without recharging the lancet, care being taken that the punctures are not bruised.

"With good lymph, and the observance of all proper precautions, an expert vaccinator should not fail of success above once in one hundred and fifty times; yet a large number of those who take upon themselves the duty, think they do very well if they succeed, however imperfectly, five times out of six."

I have dwelt at some length on the protective power of vaccination, not only because of its own intrinsic importance, but also because it is scarcely consistent with analogy to suppose that small-pox is the *only* disease which would allow of being replaced by a *substitute* disorder, of such trifling character as not to merit consideration as a disease. This preventive measure is probably only *one* illustration of a *general* principle, which may yet be applied, in a more advanced state of Pathology, to the prevention of other diseases of blood-origin. Perhaps, some day, measles, scarlet fever, and the whole tribe of 'infectious' diseases, may be similarly anticipated, with an immeasurable saving of human life among those yet unborn, and in future ages. What a contemplation this for the medical philanthropist! what a triumph of *Preventive* Medicine!

Meantime, there is one course of investigation that cannot fail to approach this grand result. The unwearied prosecution of blood-pathology, by chemical analysis, more and more refined, will tend to reveal the essential blood-conditions which confer those specific susceptibilities that severally predispose each to its own peculiar kind of infectious disease. These predisposing conditions of the blood are, indeed, manifested by the actual occurrence of some kind of infective disease, when the individual susceptibility thereto is declared too late for prevention; and the appropriate preventive measure is discovered only by experiment, as the pro-

tective power of vaccination was discovered. But if the *essential blood-condition* predisposing to any given infectious disease were *itself* demonstrated by a more subtle and searching analysis of the blood in such disease, then an adequate preventive measure might be at once suggested, and applied with confidence, in all other cases of the same disease. This rational method of prevention would entirely supersede the experimental. For example, it may be that the blood in small-pox abounds with some particular material, the chemical relations of which to a certain antidote or neutralizing agent would be as well known, as that an alkali has the power of neutralizing an acid. Guided by this knowledge, the prevention of small-pox would have been at once secured (prior to the discovery of vaccination), as surely as the administration of potash or soda is known to neutralize the lithic acid—or essential morbid condition of gouty blood. By a similar application of chemical blood-pathology, other exanthems, &c. might be prevented. Suggestive, however, as is this analogy, it is at present only an hypothesis, but one that indicates the course of investigation which pathologists may henceforth most profitably pursue.

Certain blood-diseases are declared by local inflammations of the subcutaneous cellular texture.

Cellulitis stands at the head of this class.

Cellulitis has already engaged our attention, as an illustration of the converse Etiological Principle to that which we are now considering. Among constitutional, arising from local, morbid conditions, this disease is one of much interest; and in illustration of local proceeding from constitutional morbid conditions, cellulitis is of equal importance, although of less frequent occurrence.

In a few cases—observes Dr. Duncan*—this disease commences by *constitutional* symptoms, such as commonly indicate the invasion of typhus fever, or the appearance of an exanthematous eruption. After these have continued a day or two, intense

* Trans. Med.-Chir. Soc. Edin., vol. i., 1824, p. 591.

pain with diffuse swelling, and more or less redness, supervene in some part of the body, most commonly in the hand or arm. Thus in cases described by Duncan,—namely, 25 to 30 inclusive, and in case 34—no local cause was discovered; the disease, after fever, began directly, in the part affected, with diffuse inflammation,—which in cases 29 and 34 occupied the hand and whole arm, but in all others the axilla, shoulder, and breast.

Even *traumatic* diffuse inflammation of the cellular texture may be regarded as a local manifestation of a blood-poison; for the wound is often nothing compared with its constitutional consequences.

The textural changes wrought by this inflammation are these:—Speedily an effusion of serum engorges the subcutaneous tissue; this being soon exchanged for a purulent, bloody, sanious fluid. The cellular membrane dies rapidly, appearing in the form of shreds and skeins, and as mats of wet tow, or like large wads of wet shammy-leather, extending over a whole arm, a whole side, or over both, successively. An immense extent, therefore, of cellular texture is sacrificed, and this wide-spread subcutaneous slough passes in between the neighbouring muscles; but the fasciæ are singularly spared; thus the tendinous septa between the ribs are seen bared in places where the muscular substance itself and all other textures have disappeared.

This process of destruction *probably* involves the skin, and to a corresponding extent. Yet vesicles or bullæ seldom appear until the subcutaneous inflammation is very far advanced. These vesicles are in general solitary, sometimes remote from the cellular disease, of considerable size, and occasionally filled with bloody serum.

What, then, are the diagnostic characters of cellulitis? It might be *inadvertently* mistaken for erysipelas. The swelling is not unlike,—in both, diffuse and œdematous. But the pain is at once the earliest and most distinctive symptom; it is excruciating. This, without perhaps any, even the slightest, blush of redness, is characteristic of cellulitis. And should the skin become involved,

the cutaneous inflammation is always *secondary*, in all cases of true cellulitis, by its advance from below upwards, from the cellular texture to the skin. This latter texture remains uninflamed, or is not primarily and essentially affected. So much for diagnosis.

The fever which precedes (and accompanies) cellulitis, aids but little to establish an early and exact diagnosis. In those cases which Duncan observed, this fever had so much of the typhoid type, that it was scarcely possible to foresee which disease would be eventually declared. Nevertheless, the fever presented considerable varieties in respect of its symptoms and progress. It sometimes commenced insidiously, sometimes turbulently, but in most of the severe cases soon reached its height. Its chief peculiarities were, the supine position with depressed shoulders, in which attitude the patient almost always lay, without turning to either side,—the absence of coma, and the rare occurrence of delirium. The respiration was often remarkably embarrassed, owing to the inspiratory muscles,—pectorals, intercostals, &c., being the seat of cellulitis. In some cases dyspnœa was rendered more urgent by pleurisy. In others the respiration was itself much affected, especially when the disease began in the arm. Certain facts of apparently minor significance, because only occasional, are, a peculiar cadaverous smell emitted from the patient's body during life; in one case, a fetid and coloured sweat proved critical.

Cellulitis is the local manifestation of a blood-poison. Two facts at least point to this Etiological interpretation.

The fever being *typhoid*, resembles the working of some blood-poison analogous to that which is undoubtedly in operation when typhoid fever is engendered by 'infection;' and the local manifestation itself—cellulitis being a 'serpiginous' inflammation—extending continuously, and not confined to one spot, shows that some morbid condition of the blood disturbs the course of textural assimilation.

The nature of this blood-poison is wholly unknown in respect

of cellulitis, originating apparently *spontaneously*, and therefore the (rational) prevention of this disease, when it thus arises (as we are now considering), is equally unattainable in the present state of blood-pathology. Add to this difficulty the dilemma occasioned by the diagnostic fact, that, pending the local disease we would circumvent, the fever, which should be premonitory, is so similar to that of ordinary typhoid fever as to misguide the practitioner concerning the kind of disease about to be declared, and its prevention becomes doubly impracticable.

Carbuncle and boil are alike essentially inflammations of the subcutaneous cellular texture in various parts of the body; but the effusion is *circumscribed* and brawny, so that the imprisoned cellular tissue invariably sloughs. Both these lesions also are probably evolutions of morbid conditions of the blood, differing in respect of each, although their individuality has not hitherto been shown by chemical analysis. In some infectious diseases—as typhus, typhoid fever, and plague—inflammations of a carbuncular and furuncular character arising, are obviously due to blood-poisons; but in these and all other instances of such inflammation, chemical blood-pathology is still far in arrear. The rational prevention, therefore, of carbuncle and boil is still open to investigation.

There are yet two diseases that remarkably illustrate the Principles advanced in this chapter.

Gout and Rheumatism—each having its own special tissue of election—are alike manifestations of a blood-disease, different in respect of each. This Etiological doctrine is well established.

But the Prevention of these diseases can be accomplished, in the present state of knowledge, with very different degrees of success. As regards Gout—the morbid blood-condition having been discovered, its accessions, from time to time, can be prevented; while that of Rheumatism not having been detected, its accessions cannot be circumvented.

Taking in order, *first*, the etiological principle of 'blood-origin.' Observe the phenomena of Rheumatism :—An inflammation affect-

ing some portion of *fibrous tissue;* the ligaments and tendons around the joints, more commonly, sometimes the fasciæ, and probably the pericardium and endocardium. This inflammation is *specific.* It is denoted, just as common inflammation is expressed, by redness, heat, pain, and swelling; but rheumatic inflammation is characterized by not tending to the effusion of plastic lymph, nor to suppuration and gangrene; unless, indeed, some other texture besides fibrous tissue shares the inflammation, as synovial or serous membrane, when its products are the same as those of ordinary inflammation.

This local condition is *preceded* by and accompanied with inflammatory fever, in perhaps its highest degree,—contrasting, therefore, in every way, with fever of the typhoid type. A strong, rapid, hard pulse; headache without any delirium, excepting when pericarditis or endocarditis ensues; acid perspirations and urine: these are the chief phenomena of rheumatic fever. And this fever not only precedes the local inflammation; but possibly runs its course without any such (local) manifestation.

Rheumatic inflammation, whether exhibited by the joints, the fasciæ, or the heart, is evidently due to the operation of some morbid blood-condition; and for two reasons more especially.

The very fact of the same inflammation affecting, possibly, 'many parts'—*e.g.* many joints—simultaneously, points to the blood as its common cause. So also does the 'metastatic' character of this inflammation. Passing from one joint to another—from the shoulders to the elbows, or from the knees to the ankles, perchance back again to the joints first affected, and probably thence migrating to the heart; these and similar alternations of the same character of inflammation betoken some morbid condition of the *blood,* which, as a reservoir supplying in common all parts of the body, is turned on, as it were, more abundantly (by inflammation), now on this part, now on that.

The 'symmetrical' distribution of chronic rheumatism,* in many

* Med.-Chir. Trans., 1842. Communication by Dr. W. Budd.

cases, affecting as it does corresponding parts of either half of the body, is further evidence of there being a blood-disease in operation; while such distribution exhibits also the elective power of similar portions of the same texture.

But although the blood, vitiated in some way, is determined to the fibrous texture, by virtue of its elective power, we cannot say what particular ingredient, normal or foreign to the blood's composition, is appropriated thereby. The texture undergoing rheumatic inflammation selects something—but what?—from the blood.

Pending the discovery of this essential *materies morbi*, the *rational* prevention of rheumatism is impossible. Judging by the acid state of certain secretions—perspiration, saliva, and urine—during an attack of acute rheumatism, it would appear that an *acid* of some kind prevails in rheumatic blood; and, first suggested by Prout,* other authors,—Todd,† Fuller,‡ C. J. B. Williams,§ Headland,‖ &c.,—have since concurred in believing that this acid is *lactic* acid.

It is urged that, as the perspiration contains lactic acid, with lactates of soda and ammonia, and that exposure to cold, checking this secretion, is well known to be frequently followed by an attack of rheumatism, therefore this disease is due to an accumulation of lactic acid in the blood. But then, sufficient exposure to cold ought *invariably* to have this effect; or—making allowance for individual peculiarities of constitution—at least in *many* instances such would be the effect of exposure. Moreover, the not unfrequent *spontaneous* origin of rheumatism in hospitals, where patients are protected from exposure, is irreconcilable with the theory in question.

Again, it is alleged that primary mal-assimilation—dyspepsia, in fact, of some kind—produces lactic in excess, which accumulates

---

* Stomach and Renal Diseases, 1848, p. 84.
† Gout and Rheumatic Fever, 1843, p. 143.
‡ Rheumatism, Rheumatic Gout, and Sciatica, 1860.
§ Principles of Medicine, 1856.
‖ The Action of Medicines in the System, 3rd edition, 1859.

in the blood. But this theory also is not consistent with observation, so far as the absence of any symptoms of indigestion is significant.

Neither has it been demonstrated that lactic acid accumulates in the blood, as the product of secondary textural mal-assimilation; for chemical research has failed to discover any abnormal quantity of this acid in rheumatic blood. And this fact equally tells against the supposition of its accumulation by primary mal-assimilation, or by suppressed excretion of the perspiration.

Nor does *uric* acid superabound in rheumatic blood. Garrod's chemical analyses* establish this negative fact. In truth, rheumatic blood is decidedly alkaline.†

Under these circumstances it would be idle to imagine that the blood's composition can be rectified, and the recurrence of rheumatism prevented. Without knowing the essential morbid condition—which, in those subject to this disease, is ever in operation as the cause predisposing thereto,—its evolution, from time to time, cannot be averted; and as if to show how comparatively unimportant, apart from this knowledge, is that of knowing the exciting and reputed cause of rheumatism, it is useless to avoid exposure to cold, for this alone will never evoke rheumatism, and if the blood be charged with the unknown poison, rheumatism will arise spontaneously.

Contrasting favourably with our present knowledge of this disease in its most essential aspect, the blood-pathology of Gout is now well known, and equally, therefore, the principle of its prevention.

To this end let us first observe the more prominent phenomena of gout, chiefly as compared with those of rheumatism, for which they are apt to be mistaken.

Gout is manifested by an inflammation affecting the joints, and very commonly the first joint, or *ball*, of the great toe. Commencing usually when the individual about to suffer has re-

---

* Med.-Chir. Trans., vol. xxxvii., 1854. † Ibid.

tired to rest, and has enjoyed some hours perhaps of sleep, he is awoke with fixed pain in one of his feet,—mostly, as I have said, in the ball of the great toe, but sometimes affecting the heel, instep, or ankle. With this pain, cold shivering is generally experienced, succeeded by heat, as the pain—boring, grinding, and wrenching—fastens more and yet more firmly on the spot of its election. "Place your joint in a vice," said a witty Frenchman, "and screw the instrument up until you can endure it no longer. That will represent rheumatism. Then give another twist, and you will somewhat realize gout." The skin over this part is acutely tender, red, tense, and shining, encircled by some œdema, and by converging turgid veins. Much restlessness and excitement supervene. In vain the sufferer seeks to relieve himself of the weight of his bedclothes upon the part inflamed; in vain he shifts his foot from place to place in search of a cool and easy position. The pain, remorseless, grapples yet more tightly. At length, in the course of twenty-four hours or so, it loosens its hold gradually, perhaps suddenly. The sleepless excitement also then subsides, and the victim enjoys some temporary repose. He wakes again to undergo punishment. The toe-screw is reapplied, it may be with a turn or two less; and day by day, a slighter punishment is inflicted, until at length the full penalty has been paid.

Then the cuticle peels off the part affected, for gouty inflammation ends by resolution; it never terminates by the effusion of plastic lymph, suppuration, or gangrene. In these respects, this inflammation and that of rheumatism concur.

Eventually, after frequent attacks of gouty inflammation, the cellular texture around the joint usually becomes pervaded with a deposit of urate of soda, forming concretions, at first pultaceous, then 'chalk-stones,' of perhaps considerable size. The nodular fingers and toes of chronic gout are matters of common observation. The skin over these nodules being stretched, at length breaks, and the chalky concretions are laid bare. Urate of soda has been found infiltrating all the textures of one or several joints, in synovial membrane, cartilage, the heads of bone, and ligaments;

and usurping their place, the articulations are irreparably destroyed.*

Does any characteristic—*i.e.*, peculiar and constant, constitutional disturbance precede and accompany an attack of gout? Yes, verily—and its *premonitory* symptoms refer to the functions of the stomach and kidneys, more especially. Dyspepsia, denoted by inappetency, eructations, heartburn, and acidity of the saliva; together with scanty urine, clear, high-coloured, and containing less than the average amount of uric acid or none at all; these symptoms portend a fit of the gout.

The *blood superabounds* with *uric acid;* but its normal constituents apparently are not modified. "It is," writes Dr. Garrod,† "by the augmentation of those principles which exist in health, in such minute traces as to be detected with difficulty, that the peculiar alteration of the blood in this disease is manifested."

"The blood in gout always contains uric acid in abnormal quantities, and in the form of urate of soda, which salt can be obtained from it in a crystalline state."‡

Gout is, therefore, a blood-disease. And the fact first disclosed by Garrod's analyses, coupled with the known phenomena of arthritic inflammation, inducing the formation of urate of soda concretions,—to which I may add the fact of abundant deposit of urates in the urine; these facts taken together plainly declare the pathology of this disease. An attack of gout is an effort of nature —of the restorative power—to expel a poison, uric acid, from the blood. Dr. Watson§ well describes this struggle. "Morbific matter (it may well be called a *poison*) is generated, or detained, under certain circumstances, within the body, and silently collects in the blood; until, after obscure threats, perhaps, and prelusive mutterings, it explodes in the foot; and then the bodily economy,

---

* Diseases of the Joints, 1850, Ed. 5, Sir B. C. Brodie.

† The Nature and Treatment of Gout and Rheumatic Gout, 1859.

‡ Trans. Med.-Chir. Soc. 1848.

§ Principles and Practice of Physic, 1857, vol. ii., p. 762.

like the atmosphere after a thunder-storm, is for a while unusually pure and tranquil."

Chemical analysis having actually detected the presence of this poison in the blood, it is scarcely necessary to even glance at those characters in the history of gout which might otherwise be appealed to in support of its being a blood-manifestation. The absolute test of 'blood-disease' having been supplied in this instance directly (by chemical analysis), supersedes the occasion of any other evidence, such as I have hitherto resorted to, in the course of this chapter. But I might point to facts such as these :—Gout visits 'many textures' and parts of the body simultaneously or in 'succession.' Besides engaging many joints at once, or flitting from one to another; gout wanders about, disturbing the heart, the lungs, and the brain. Hence palpitation and syncope, hence dyspnœa, hence disturbed vision and hearing, with cerebral commotion, bordering on apoplexy and paralysis. This is known as irregular, lurking, or masked gout. Sometimes, however, having settled in the foot, it suddenly disappears, and migrates to the stomach, heart, or brain; *retrocedent* gout, as it is then called, being unlike the retreat of an ordinary foe—an assault on the very fortress of life. Less perilous migrations are witnessed, when gout betakes itself to the urethra, occasioning a scalding discharge; to the testicle, constituting one form of orchitis; to the eye, giving rise to ophthalmia. All these manifestations of irregular and migratory gout should be borne in mind; otherwise, the disease in some form might be overlooked by the preventive practitioner.

In whatever shape gout may have appeared, whether regular, disguised, or migratory, its decline is marked and measured by a flow of urine, surcharged with uric acid, thus relieving its accumulation in the blood, and plainly declaring the nature of the disease.

Viewed from the vantage ground of pathology, the prevention of gout resolves itself into this :—the prevention of uric acid accumulating in the blood. Therefore, whenever its *premonitory* symptoms arouse suspicion that gout is approaching, they can be verified directly by chemical analysis of the blood itself. The ready

method proposed and practised by Dr. Garrod for this purpose, and for which the abstraction of only a very small quantity of blood is requisite, he thus describes as the "uric acid thread experiment:"*—

"Take from one to two fluid drachms of the serum of blood, and put it into a flattened glass dish or capsule; those I prefer are about three inches in diameter, and one-third of an inch in depth: to this add ordinary strong acetic acid, in the proportion of six minims to each fluid drachm of serum, which usually causes the evolution of a few bubbles of gas. When the fluids are well mixed, introduce a very fine thread, consisting of from one to three ultimate fibres, about an inch in length, from a piece of unwashed huckaback, or other linen fabric, which should be depressed by means of a small rod, as a probe or point of a pencil. The glass should then be put aside in a moderately warm place until the serum is quite set and almost dry; the mantelpiece in a room of the ordinary temperature, or a book-case, answers very well, the time varying from twenty-four to forty-eight hours, depending on the warmth and dryness of the atmosphere.

"Should uric acid be present in the serum in quantity above a certain small amount, noticed below (*vide* work), it will crystallize, and during its crystallization will be attracted to the thread, and assume forms not unlike that presented by sugar-candy on a string. This may then be examined by a linear magnifying power of about fifty or sixty, procured with an inch object-glass and low eye-piece, or a single lens of one-sixth of an inch focus answers perfectly. The uric acid is found in *rhombs*, the size of the crystals varying with the rapidity with which the drying of the serum has been effected and the quantity of uric acid in the blood."

To ensure perfect success in this experiment, several precautions are necessary, for which the reader is referred to Garrod's work, pp. 111–113.

---

* Op. cit., 1859.

With a view to the prevention of gout, rather than to its diagnosis, when actually present, although perhaps in a disguised form, the uric acid thread experiment is not a very delicate test, and so far, therefore, its value is less. Dr. Garrod has ascertained that an amount of uric acid equal to at least 0·025 grains in 1000 grains of serum, in addition to the trace existing in health, must accumulate before this experiment gives indication of its presence.

When, however, *premonitory* symptoms, coupled perhaps with the signal given by this test, announce that gout is impending, the result of chemical analysis at once suggests adequate preventive measures.

It had long been a matter of familiar experience that certain habits of life predisposed to gout, and that certain other habits of an opposite character had an opposite tendency. Indulgence in animal food, more particularly, and stimulating drinks; generally, in point of fact, what is called 'rich living,' together with a sedentary, idle life; these are the acknowledged parents of gout; and that moderation in the 'pleasures of the table,' even abstinence, with a life of active exercise, has no such offspring. Luxury and ease have long since been mistrusted as unqualified advantages compared with the apparent hardships of daily bread, earned by daily labour; so much so, that Abernethy's pithy advice, "Live on sixpence a day, and earn it,"—pointing, as it does, to the two elements, food and exertion, in relation to gout,—has passed into a proverb.

The regulation of diet by exercise—of bodily supply by expenditure—is, therefore, the preventive measure furnished by *experience*. And is not this knowledge quite sufficient for practical purposes? Certainly not. It affords no clue to the right understanding of the physiological relation subsisting between food and exercise, nor of the pathological relation between these hygienic requirements which determines the rational prevention of gout. It is the peculiar province of Science to supply the interpretation and elucidate the significance of facts. Knowledge thus becomes appreciated, and *applied* because appreciated.

Physiological Chemistry having first demonstrated the fact that all kinds of animal food more especially furnish uric acid in their transit through the body, and that this metamorphosis is principally due to decomposition of the muscular textures; it became obvious that the balance between the production and elimination of uric acid can alone be adjusted and regulated by a supply of animal food *only* in proportion to its waste by bodily exercise.

Then Chemical Pathology contributed the additional and complemental fact, that uric acid existing as urate of soda in abnormal *excess* in the blood, is the *materies morbi* of gout. Obviously, therefore, the preventive measure which should be directed against such accumulation is this :—*To allow only that particular proportion of animal food which, with daily exercise in proportion, will preserve the blood free from uric acid, short of the trace existing even in health.*

Now this goes a step beyond mere experience. Guided by an exact knowledge of the *essential* morbid blood-condition from whence proceed all the phenomena of gout, we are enabled so to regulate the health as to prevent this disease. Exact information of this blood-condition, respecting any *individual* in question, can be readily obtained by abstracting a very small quantity of blood for analysis, or by examining the serum exuded under the application of a blister.

Moreover, by this *scientific* knowledge we are led to rationally administer certain medicines in aid of our hygienic preventive measures. Alkalies—of which the bicarbonate of potash is perhaps the most efficacious for prolonged use—may be administered daily, to neutralize any fresh accession of acid; and colchicum, as a diuretic, will aid the elimination of urate of soda by the kidneys. The mineral waters of Vichy, Wies-Baden, and other places of known repute, owe their virtue chiefly to similar qualities; but in speaking of the *principle* of prevention, I need not enter further into detail.

To illustrate the prophylactic management of gout, Dr. Garrod relates a case, on the authority of Sir H. Halford, in which col-

chicum with quinine was given, in moderate doses, daily for two years, and gout prevented, when previously scarcely two months elapsed without an attack.

This *régime*, practised ere chemistry had brought to light the true nature of gout, was a tentative method only of prevention, pursued by the blind gropings of experience. Yet it proved successful. How much more *surely* successful, then, may not it and other preventive measures become, when skilfully guided by the recent disclosures of chemical pathology, in respect of this disease?

Regarded, indeed, in all its bearings, the blood-pathology of gout is the most *unique* contribution which the 'chemical' method of investigation has hitherto made to Preventive Medicine.

Etiological Principle.

Local may proceed from Constitutional morbid conditions; *concluded* by Injuries and Diseases of the Central Nervous System, in relation to disorder of the Heart and circulation; the Lungs and respiration; the Chylopoietic viscera and the process of digestion; the Organs of Excretion—kidneys and bladder, skin—and the functions severally of these organs; diseases of Nutrition; organs of Reproduction and their functional derangements.

I insert the above to complete the *design* of this chapter. The pathology of the Central Nervous System represents those injuries and diseases which, from their predominant influence on all other organs and functions, may be aptly termed 'constitutional' morbid conditions; but the limits and surgical character of this work forbid me entering on a subject so extensive: it is here alluded to only in further illustration of that general Etiological Principle which has been already considered, and to fully realize which the pathology of the central nervous system must be applied.

CHAPTER IX.

CONSTITUTIONAL MAY PROCEED FROM LOCAL MORBID CONDITIONS.

This general Principle of Etiology, and the Principle of Prevention also, in a measure, illustrated by Injuries of Mechanism, and their constitutional effects —Shock (collapse, prostration), Reaction; Prostration with excitement, and Tetanus; respectively following Wounds, Burns, Fractures, and Dislocations. Preventive Measures.

In tracing the constitutional effects of all kinds of injuries, and those of many diseases of nutrition, we arrive by analysis at the converse Etiological Principle to that advanced in the preceding chapter. I shall, therefore, now consider the evolution of Constitutional from Local morbid conditions—local internal causes. And firstly, from injuries.

Clinical observation discovers no essential difference in the kind of constitutional functional disturbance induced by the various kinds of injuries. Pathologico-anatomically, they may be widely different; Pathologically, they all agree in provoking functional derangements of the Nervous System, known as shock (collapse, prostration), reaction; prostration with excitement, and possibly tetanus.

It matters little, then, what *kind* of injury we select,—whether a wound, burn, fracture, or dislocation, as our starting point in the history of shock and reaction, &c.; that special case will sufficiently illustrate the same general Etiological Principle—the Local origin of Constitutional functional disease or disturbance, in this instance, of the Nervous System. Take a common case, that of burn, superficial but extensive, and mark the kind of constitutional disorder it immediately induces. Many such cases are compared by Mr. Travers in his work on "Constitutional Irritation."

The muslin dress of a middle-aged lady, the mother of six children, became ignited as she lay upon a sofa beside the fire; and when the servants, upon hearing her shrieks, entered the

apartment, her person was literally enveloped in flames. Upon stripping her, almost the entire surface of the trunk of the body was denuded of cuticle. She shivered as if from a sense of cold, and from the moment of the injury her pulse was so much contracted as to be scarcely perceptible, and very soon ceased altogether. Her mind, although distracted, was under command till within a short period of her dissolution, which was preceded by a state of stupor, and took place twelve hours after the injury.

Here, then, we witness the phenomena which constitute the *shock* of injury—namely, failure of the heart's action, accompanied with a thready, feeble pulse, coldness and pallidity, with some cerebral disturbance, ending in stupor and a suspension of the cerebral functions.

Similar phenomena are observed after other kinds of injury. The charge of a musket, consisting of slugs, entered the thigh of a fine lad, thirteen years of age. The entrance wound was about two inches from the trochanter major, and the slugs passed obliquely across the limb. The femur was shattered. There had been no external hemorrhage, but the pulse could not be felt at the wrist. The boy's countenance was pallid, the surface cold, and the pupils as fully dilated as if under the influence of belladonna. He was perperfectly rational when roused, but strongly disposed to stupor; made no complaint of pain, but was troubled with insatiable thirst. He died nine hours after the injury. The hip-joint was found to be uninjured, but the trochanter and upper part of the femur were shattered to fragments, the surrounding muscles extensively lacerated, and the artery torn across. The effusion of blood was inconsiderable, the torn ends of the vessel having contracted and receded to a distance of nearly two inches. The contents of the head, chest, and abdomen were perfectly natural, and free from any morbid appearance.

This case, then, illustrating the phenomena of shock proceeding from a lacerated wound and comminuted fracture, shows that such constitutional disturbance is appropriately named the shock of *injury*, without specifying any particular kind of injury as being more especially its cause.

The history of other cases exhibits additional derangements of function, which, although exceptional, are Constitutional in the sense I use that term. For example, hiccup, obstinate vomiting, convulsions, occur in some cases; and these, again, are disturbances of the nervous system. Suppression of urine sometimes occurs, and this also implies the failure of a predominant function, that of excretive secretion, in its most important mode of operation.

These and other symptoms of similar import are familiar to the clinical observer in cases of shock; but the more prevalent condition is the impression made on the 'nervous,' and *thence* on the 'vascular systems,' as denoted by failure of the heart's action, with a cold, pallid skin, cerebral syncope, and soft, sighing respiration. From this state of collapse or prostration the patient may never rally, or a natural restorative effort may ensue in a period varying from a few minutes to thirty-six or forty-eight hours, or more.

This *reaction*, as it is termed, of course implies the revival of those functions which have been temporarily suspended. Its phenomena will therefore almost be presumed. The balance of the circulation is regained, and with it warmth returns, and the colour of the living body reappears. The cerebral obscurity clears off. If revival be incomplete, then a mixed state of prostration with reaction obtains. The pulse is rapid, but weak and liquid; the mind becomes excited and bewildered; the manner agitated and tremulous. The patient now dozes off, then flickers up, as it were, suddenly, only again to wander, and again arouse. At length frenzy possibly, subsiding in coma, ends in death; or ultimately, reaction prevailing, the pulse regains its force, loses its frequency, and health is re-established. Mr. Travers, who first named this ordeal *prostration with excitement*, drew attention to it by cases, since corroborated by the experience of every surgeon, and of which the following one shows the general course of the symptoms:—

A stout lad of fourteen had his leg jammed by a piece of timber falling upon it. The tibia was comminuted rather above its middle. The integuments on the fore and outer part of the leg were separated from the bone and muscles from a little below

the tubercle nearly to the outer malleolus, and the extensor muscles considerably lacerated. He was pale and sleepy from a free hemorrhage. Amputation was performed at midnight, about an inch below the tubercle. During the operation he became very faint, the pulse extremely quick and sometimes almost imperceptible. A small quantity of wine and twenty drops of laudanum were administered, but rejected before removal to his bed.

Five A.M. the next morning.—Has been restless and slightly delirious; frequent muscular catchings. Seven A.M.—Has vomited again, and is disturbed by the convulsive action of the muscles. Laudanum, twenty-five minims, given. In ten minutes a tranquil sleep; pulse sunk to ninety-four, and proportionately fuller. Two P.M.—Disposition to coma; rather incoherent, but can answer rationally when roused. Five P.M.—Lips parched, skin hot and dry, pulse increasing in fulness and frequency. Six P.M.—Has had a convulsive attack, apparently epileptic; pulse now very rapid, eyes fixed, profuse perspiration, breathing quick and laborious. Nine P.M.—Comatose, does not answer questions, impatient of being disturbed. Pulse one hundred, soft; skin moist. Bowels not relieved since the operation, although castor oil and an enema have been administered. After sleeping ten or fifteen minutes, he suddenly starts up, bawls vehemently, and then falls back on his pillow. Six ounces of house physic given as an enema.

Following day, nine A.M.—Continued very restless until six A.M., when his bowels were relieved and he became composed. Opium one quarter of a grain, and castor ten grains, as a bolus every fourth hour. Two P.M.—More quiet since the medicine, but when awake is some time before he can give a rational answer. Three copious pale evacuations. Ten P.M.—Sleeps soundly for half an hour together, and answers readily.

Next day, eight A.M.—Passed a comparatively good night, and is perfectly sensible, makes no complaint. Pulse one hundred, tongue less coated, no evacuations since yesterday afternoon, subsultus has entirely ceased. Two P.M.—The enema again given, followed by two copious evacuations. At intervals during the

day he slept soundly. Pulse one hundred, full and soft; skin still hot and dry, tongue much cleaner, no anxiety of countenance.

Eight A.M. on the following morning.—Had a natural motion, and passed a comfortable day.

Seven A.M. the next morning.—Stump examined, and no adhesion about any part, but suppuration profuse and fetid; edges of the wound are sloughing.

The constitutional disturbance more and more subsided, and in the course of a week the slough had all separated, and the entire surface was granulating kindly. In another week the shell of bone was found not yet detached, and the granulations were exuberant. After a month more, the flake of bone was broken off with forceps, and a healthy surface of bone exposed. The wound now healed rapidly, and very soon the boy left the hospital (St. Thomas's).

Such are the ordinary phenomena of shock followed by reaction, and such the phenomena of that concurrence known as prostration with excitement—a condition in which sometimes prostration is the prevailing element, sometimes excitement—delirium traumaticum —very much resembling the excitement of delirium tremens, as exhibited by the young and vigorous.

Now these are illustrations of a general Etiological Principle;—the Local origin of Constitutional functional disease, or disturbance; in the cases adduced of the Nervous system: and the important question is, whether, and how far, such disturbance can be Prevented.

For this purpose it should be borne in mind, that although shock follows injury so immediately as not to allow of the prevention or mitigation of its *instant* severity; yet that the chance of restorative reaction is regulated not only by the primary severity of shock, but also by its subsequent *duration*.

Experience further shows that the latter element is, in its turn, regulated chiefly by the *persistence* or otherwise of the 'cause in operation,' which first gave the shock. Bad news will cause the susceptible to grow pale and faint in a moment, soon perhaps to rally; while a depressing passion, such as grief, is a rooted

sorrow which permanently weighs upon the heart, thus precluding reaction. Of the various kinds of injury also to which the mechanism of the body is exposed, some, such as wounds and burns, not subsequently exposed to the air, do their work at once; others, such as unreduced fractures and dislocations, continue to operate, by laceration and pressure, on the nervous system. Shock is maintained, and reaction is much postponed. The shell of bone, in the last case, during the slow process of its separation, postponed reaction, which, however, eventually became victorious; and daily experience supplies similar instances of persistent prostration, by virtue of its causes being persistent in operation.

The question of reaction therefore, as regulated by the *duration* of prostration, refers us to the cause or causes still in operation.

But the efficacious removal of any (persistent) cause of (continued) prostration, presupposes its prior detection, at the earliest period, if possible, of its operation;—implies the earliest, as well as the most exact, diagnosis.

Unfortunately, however, those injuries only which immediately produce their *full* measure of shock—not therefore to be prevented or mitigated—are open to detection,—such are wounds and burns not subsequently exposed to the air; while those injuries, the prostrating effects of which accumulate, such as *unreduced* fractures and dislocations, are less open to detection. Nevertheless, the tactus eruditis will mostly prove sufficient to meet any difficulty of diagnosis; so that, with rare exceptions, *continued* prostration is not, or need not be, due to the kind of injury remaining undiscovered.

Whether, when discovered, the cause or causes in operation can be *removed*, is another question, and the one immediately bearing on the prevention of continued prostration. But it will be answered most fully by considering the etiology of what appears to be another phase of this constitutional disturbance.

Tetanus is manifested by certain phenomena of the 'nervous' and 'muscular systems,' which are apparently allied to those of

shock, consequent on injury. This latter condition ordinarily denotes a failure of the heart's action, with perhaps a suspension of consciousness, together with a loss of sensibility and the power of voluntary motion; but in some cases involuntary excito-motion is exhibited, by hiccup, vomiting, and even *general convulsions*. *Tetanus* (τείνω, to stretch) signifies a violent and involuntary contraction of the voluntary muscles, attended with severe pain and rigidity. The spasmodic contractions having commenced, never entirely relax throughout the course of the disease; they are *tonic*, thus distinguishing them from ordinary convulsions, as in hysteria; and consciousness is retained to the last, thus distinguishing the tetanic from epileptic convulsions, and from those of hydrophobia.

Tetanus approaches and proceeds by the following series of spasmodic contractions, each persisting in the order of their succession:—Stiffness is first experienced about the root of the tongue, which is usually retracted. Articulation therefore is imperfect. A sense of painful rigidity in the posterior muscles of the neck, and some difficulty in moving the jaw, are soon experienced. If the jaw be fixed and tetanus proceeds no further, it is known by the name of *trismus*, or lock-jaw. Generally speaking, other sets of muscles become involved. Deglutition is accomplished with great difficulty, and fluids are convulsively ejected when any attempt is made to swallow them. So far tetanus resembles hydrophobia. Even the sight of fluids may occasion dread,—another point of resemblance. Yet in no other respect is there any similarity between these diseases. No foaming at the mouth of a thick mucus, with constant movement of the jaw to extricate it, is ever witnessed, as in hydrophobia. Other peculiarities supervene. Pain strikes through the body from the ensiform cartilage backwards to the spine, and being accompanied with intense spasm of the diaphragm, occasions agonizing dyspnœa, which has been compared to that of hydrophobia. Very soon the muscles of the back contract, and with such violence that the body is drawn into the form of an arch resting on the head and buttocks (opis-

thotonos); or the abdominal muscles contracting, become as hard as a table, and draw the body forward (emprosthotonos). During these spasms, the rectus muscle has been torn with the violence of its contractions. The body is sometimes bent to one side (pleurosthotonos). Next in order, the muscles of the lower extremities are involved; and lastly, those of the upper, excepting the fingers, which generally remain movable to the last. The tongue also is rarely involved, although it may be thrust violently against the teeth and be much lacerated.

Such is the ordinary course of events in *tetanus*. They constitute one continued manifestation of spinal excito-motion, without suspension of the cerebral functions; but, as would be expected, the tetanic spasms—violent and continuous—provoke general disturbance of the *organic* functions. Thus, the respiration being much embarrassed, the heart beats more quickly and forcibly, giving to the pulse similar characters; but they are eventually succeeded by feebleness and irregularity. The urine is perhaps scanty and high-coloured, and is sometimes retained or voided in small quantities, while the skin pours forth abundantly a sour-smelling sweat. Lastly, obstinate constipation is a constant and significant symptom throughout this disease; and should an evacuation occur, it is singularly offensive. "I remember," says Abernethy, "on one occasion, asking an old nurse what sort of evacuations had come from a tetanic patient, who for a week had no relief from the bowels; 'Lord, sir,' she replied, 'they are not stools, they are sloughs.'"

Fortunately, from such misery the sufferer is released by death within one, two, three or four days, or it may be in about a week, although in some cases much later. The tetanic contractions may subside and life be spared; and considerable experience shows that this happy issue is mostly granted to the few who survived the ninth day.

Now the question of Prevention presses; for *tetanus* is rarely curable or recoverable.

Certain causes of *tetanus* are beyond our control. This disease is more prevalent in warm climates and in marshy districts,

also near the sea-coast, than inland. No period of life is exempt, although perhaps infancy and middle age are most subject; while, as to sex, males are most liable. Under these circumstances, tetanus arises from other causes than injury; and the origin of this *idiopathic* form of the disease is obviously, for the most part, beyond human anticipation.

Traumatic tetanus, resulting from various kinds of injury, is at once an illustration of constitutional disturbance arising from local causes, and of causes the *continued* operation of which can be prevented. But the etiology of traumatic tetanus is a subject of wide extent. No kind of injury is without some tetanic danger;—from the most trifling scratch to the most terrible laceration tetanus may ensue, yet not with equal probability. What, then, are the injuries from which this disease occurs most frequently? They are those which, as regards their nature, extent, and situation, are most conducive to the continuance of *shock*. This, at least, is the conclusion to which my own observations point. Tetanus and persistent shock, terminating in convulsions, are alike induced by whatever continues to irritate any portion of the Nervous System.

Sometimes trismus and general tetanic twitches supervene on continued prostration, and remaining associated with it, constitute a variety of 'prostration with excitement.' The excitement is *tetanic*. Such were the phenomena I observed during the course of a severe burn, lately under the care of Dr. A. Marsden in the Royal Free Hospital. The burn extended over the toes and instep of the right foot and lower part of the leg. Its depth was to Dupuytren's fifth degree. The annular ligament was destroyed, the interior of the ankle-joint exposed, as also were the carpal bones, and the nails were burnt away. An attempt was made to save this foot, which would, I believe, have proved successful, with the removal of portions of bone; but the patient, a middle-aged woman, was fat and flabby, and a hard drinker. Prostration continued, and in a few days partial trismus, with tetanic twitches of the arms and hands, set in. The patient's mind wandered; she frequently raised her head, uttered a few words, and then dropped on to the pillow,

turning her eyes right and left with restless agitation. I suggested to Dr. Marsden, who was good enough to ask my opinion, that amputation might be deferred, and that a grain of opium, with ten grains of castor, be given every four hours. The trismus never became complete, nor the tetanic twitches more general; but this condition lasted for more than a week, with continued prostration, when the woman died exhausted.

In other cases of continued irritation *pure* tetanus supervenes.

A man died of universal tetanus in a few hours after an oblique fracture of the thigh-bone, which penetrating the rectus muscle, was continually playing through its belly with a see-saw* motion.

A man having a simple fracture of the femur, and who appeared to be doing well for four days, was seized on a sudden with lock-jaw, and died in three days of acute universal tetanus. Examination showed that the upper fragment of the bone, obliquely fractured, had perforated and left a detached spiculum of considerable size transfixing the vastus internus muscle.†

The alliance of tetanus and continued prostration is thus indicated by clinical observation, and also the origin of both from continued nervous irritation. A further investigation of the known causes of traumatic tetanus shows the influence of *texture*, together with that due to the *kind* and *extent* of injury.

Burns, we have seen, induce tetanus. Wounds also, whether unhealthy, healthy, or healed, tend to induce this constitutional disturbance. In one case, related by Hennen, cicatrization was completed on the same day that life terminated; and Dr. Elliotson observes, the disease has sometimes declined and ceased, although the wound daily grew worse. The size of the wound is immaterial. Large amputations, or the removal of a breast or testicle, are known causes; so also the operation for fistula in ano, the irritation of a seton in the neck, or extraction of a tooth, have occa-

---

* Constitutional Irritation. A further Inquiry, 1835. Travers, p. 292.

† Ibid, p. 315.

sioned tetanus. Nay, more, a scratch of the thumb, made by a broken plate, proved fatal in a quarter of an hour; and in some cases a slight blow, as by a whip-lash under the eye, although the skin may be unbroken, has been followed by tetanus.

The kind of wound and the textures involved are all-important circumstances for consideration. Punctured, lacerated, and contused wounds, more especially of the hand or thumb, of the sole of the foot, or of the toes, have a tetanic tendency. Unyielding fibrous textures are peculiarly revengeful in this respect. Compound fractures and dislocations, rather than simple, are threatening complications; and they are more so if the fracture be oblique and playful among nerves and muscles, or if the dislocation be that of a ginglymoid joint, as of the thumb.

These are the more frequent causes of traumatic tetanus, and, observing their general character, they *all* concur in producing considerable nervous prostration as the *immediate* effect of their operation. So far tetanus cannot be prevented. But this first impression is (necessarily) aggravated by *continued* irritation, should the cause, whatever it be, continue to operate. Witness the see-saw action of fragments of bone in the two cases I have mentioned.

Fortunately, some time usually elapses between the occurrence of the local injury and the commencement of actual tetanus, and this probationary period has its average limits of duration. In one thousand cases, collected by Sir James Macgregor during the Peninsular War, none occurred after the twenty-second day.* No case after the fifteenth day, was the observation of Larrey, during the French campaign in Egypt.† These were the limits of probation. Therefore, knowing the kinds of injury which most frequently induce tetanus, here is an interval, variable in duration it is true, but still a period between the local injury and the commencement of tetanus, during which *opportunity* is afforded for

---

* Med.-Chir. Trans., vol. vi., p. 453.

† Mém. de Chir. Militaire, t. i., p. 263.

removing any cause in operation, provided only it be detected, and sufficiently early.

Now, excepting the influence of constitutional predisposition, which is beyond our control, all *local* causes can, in most cases, be readily *discovered* and *removed*. Wounds may be thoroughly cleansed of foreign bodies, and the parts adjusted. Swelling, under tight, unyielding textures, may be relieved by incisions. Burnt surfaces may be effectually protected from the influence of the air. Most compound fractures and dislocations can be soon detected, reduced, and kept in position.

I subjoin four cases, two of incipient trismus, and two of incipient general tetanus, which seem to me to convey this instruction. They occurred in the practice of Mr. Travers.

A stableman had a tense circumscribed hypogastric swelling, corresponding to the sheath of the right rectus abdominis muscle. It followed the kick of a horse in the right groin a few days previously. The general health remained undisturbed, fluctuation could not be detected, and some doubts were entertained of its being a ventral rupture, when he was suddenly seized with trismus. The swelling was immediately punctured, and then freely incised, whereby half a teacupful of pus escaped, the spasm subsided, and left him entirely after a day or two.

A stout man, aged twenty-seven, received a lacerated wound of the hand. In forty-four days he was seized with trismus, which ceased with the discharge of a deep fascial abscess of the fore-arm.

A man, aged twenty-eight, received a gunshot wound of the hand, with fracture and dislocation of the wrist and metacarpal bones; he became tetanic on the fourth day. Complete relief of the symptoms was obtained by incision of an abscess of the fore-arm at the end of eighteen days.

A farm-servant was attacked with tetanus, threatening suffocation from spasms of the diaphragm, in consequence of a wound four inches long in the right buttock. Tetanic symptoms commenced in twelve days, and the disease yielded to extensive

and repeated divisions of the integuments beneath which matter had collected.

These cases actually *overprove* the *preventibility* of tetanus, by removing the causes in operation: they prove also the possibility of recovery from this disease when its causes are promptly removed. Are there any other causes possibly in operation besides the local injury? I have said that constipation is constant and significant; significant, partly because constant, and also because of the peculiar matter voided when an evacuation does occur. More than thirty years since, Abernethy proposed for the consideration of surgeons this question—whether the disordered condition of the digestive organs, established during the irritative state of the wound, may not be the occasion of tetanus, when that irritation has itself ceased? If, he adds, this proposition be established, the very important conclusion follows, that by preventing the disordered condition of the digestive organs, you would prevent tetanus. What, then, is this condition, as disclosed by post-mortem examination?

In several cases the intestines have been found much inflamed, and in two a yellow, waxy fluid, of a peculiar offensive odour, covered their internal surface.* The pharynx and œsophagus may be much contracted, and contain a viscid reddish mucus. (Larrey.)†

Inflammation apparently extends to the cardiac portion of the stomach; but this is not peculiar to tenanus; it has been found also in cases of hydrophobia, as well as in other diseases. The veins within the vertebral canal are sometimes turgid with blood. In a case of strangulated femoral hernia, on which I operated, all went on favourably for a week, and the incision had nearly healed, when suddenly the most violent tetanus set in, beginning with lock-jaw, proceeding to appalling opisthotonos, and terminating fatally on the fourth day, by which time the wound had reopened and become gangrenous. I carefully examined the cerebro-spinal axis throughout its whole extent. The intra-spinal veins were

---

* Med.-Chir. Trans., vol. vii., p. 475.

† Mém. de Chir. Militaire, t. iii., p. 287.

engorged with blood, but not those of the cord itself. Hydrophobia has the reputation of being associated with congestion of the vessels of the spinal cord.

In conclusion; our present knowledge of the pathology of Tetanus being fairly interpreted, points to the continuance of nervous irritation, and thence to the kind and extent of the local injury, and to the textures involved as the immediate cause of *traumatic* tetanus. Coupled therewith is the peculiar constipation which perhaps precedes, and certainly accompanies, the disease. This local and this general condition admit of timely intervention, and so far our etiological knowledge may be preventive, or at least anticipatory, of the full development of tetanus.

These considerations are most important, for I am sure that every practical surgeon will concur in acknowledging the hopelessness of all known medical treatment when tetanus is fully developed, and the equal uselessness in such case of any surgical operation to relieve the symptoms of, much more to cure, this deadly disease.

CHAPTER X.

THE ETIOLOGICAL PRINCIPLE ADVANCED IN THE PRECEDING CHAPTER FURTHER ILLUSTRATED BY DISEASES OF NUTRITION, THOSE OF OTHER FUNCTIONS, AND BY CONTAGION; SEVERALLY, IN RELATION TO THEIR CONSTITUTIONAL CONSEQUENCES.

Various (Constitutional) Disturbances of the Blood, Nervous, and Vascular Systems, *i.e.*, Fevers—inflammatory, hectic, and gangrenous typhoid—arise from inflammation, suppuration, and mortification, respectively.

Various (Constitutional) Blood-diseases arise from other perversions of Nutrition besides that of inflammation; also from those of the Digestive process, Excretion, and Respiration, respectively.

Various (Constitutional) Blood-diseases arise from Contagion, illustrated by—

Contagious matter derived from Animals:—Hydrophobia, Snake-bites, Malignant Pustule, Glanders, Vaccinia.

Contagious matter derived from the Human Species:—Hospital Gangrene, Puerperal Fever; Primary Syphilis—Chancres and Buboes.

The Pathological Principle which guides the Preventive View of these Diseases. This presupposes a sufficiently early and exact detection and discrimination—Diagnosis, of each one.

Preventive Measures.

THE Local origin of Constitutional Disease is further illustrated, as a General Principle of Etiology, by Diseases of Nutrition and the Constitutional Disturbances they induce.

Three 'constitutional tissues,' the blood, nervous, and vascular systems, in conjunction with all other tissues, co-operate to fulfil the compound function of healthy (textural) nutrition; and due knowledge of this department of Physiology will enable us to interpret rightly the phenomena of Inflammation—the most frequent departure from, and perversion of, the process of textural assimilation.

Observe, then, the process of nutrition, so far as it can be traced in those tissues which, being situated on the surface of the body, are open to observation. Epidermic tissues, such as scarf-skin or cuticle,

hairs and nails, are thus patent. The scarf-skin, for example, is a layer of cells overlying the sensitive and vascular true skin, from which a nutritive plasma perpetually exudes, in which the cells are formed. Let a portion of cuticle be removed by a blister or a slight burn, and the exposed surface of true skin is found to be constantly bathed with this nutritive fluid. If sponged dry, the surface immediately perspires, as it were, and moistens again. The cells first formed in this fluid are soft and round, as seen under the microscope in the newly-formed cuticle. I have observed similar cells in the thin new cuticle which accompanies the formation of true skin around the margin of a healthy healing ulcer. These round, soft cells give place to others underneath; and as the former pass up from the true skin, they become dry, hard, and flattened, and acquire the characters of ordinary scaly epithelium, which eventually disintegrates and desquamates from off the surface of the body. This process is always going on, and the cuticle coming off more or less perceptibly, by the decay, death, and shedding of the cuticular cells; they having previously served the function of protecting the subjacent true skin. Death, decay, and disintegration, by the exercise of function, may be inferred, if not seen, to be proceeding in all other tissues.

But constant waste, consequent on the exercise of function, implies constant repair; and the reparative material can only come from the blood. This vital fluid must therefore present an *appropriate composition* for the repair of each component tissue of the body, and must also be supplied in *adequate quantity* thereto, as all are undergoing incessant destruction.

Assuming this twofold condition of quality and quantity due to each of the tissues, they, on their part, severally *select* and *secrete* from the blood-vascular system, as the common reservoir, that kind and amount of plasma which is appropriate for their *individual* nutritive maintenance. The blood is thus left *reduced* in *quantity*, and deprived of those constituents which have entered into the formation of the tissues. But observe, as each tissue draws its own particular nourishment, the residual mass of blood

(in circulation) becomes *relatively adapted* for the proper nourishment of those tissues which have dissimilar composition. The tissues severally act (functionally) as 'excreting' organs. The blood of the part, reduced in quantity, but improved in quality relatively for the nourishment of other tissues, is also maintained by the co-operation of other functions—digestion, secretion, and respiration—while its circulation is regulated by the agency of the nervous system. Again and again is this fluid replenished and renovated, distributed to every part of the body, each tissue claiming and retaining that quantity of the common pabulum which may be necessary, and selecting those ingredients which enter into its own formation, to repair its waste and thereby maintain its substance.

It would, therefore, be anticipated that any *increased demand*—*i.e.*, beyond that of mere 'maintenance,' as by 'growth' and 'development'—necessarily implies at least an *increased supply* of blood to the part undergoing such changes, and perhaps, also, a *different quality* of blood supplied to meet these additional requirements. Indeed, ever moving and vibratile as are the leaves of an aspen tree on a breathless summer's day, so likewise changeful is the nice adjustment and equilibrium of nutrition. The balance of maintenance could never be preserved from hour to hour, scarcely from minute to minute, without a corresponding capacity in the circulatory system to satisfy variable demands. And not a sensation thrilling, not a thought inspiring, nor an emotion agitating ever so gently, but forthwith there is a flush of arterial blood with life-giving energy, manifesting the wide-spreading influence of the nervous system. In some instances this rush of blood is visible —in blushing, for example. By analogy, is it not probable that any act of *volition* in like manner relaxes and enlarges the blood-vessels of the muscle or muscles to which it is directed, through the medium of those nerves that are distributed thereto? The flow of nervous influence, or *vis nervosa*, which excites contraction of the voluntary muscular fibres, simultaneously relaxes the involuntary fibres of the smaller vessels, not to say the capil-

laries. The waste of muscular tissue incurred by each momentary movement, and therefore more so by continued exercise, is thus repaired by an adequate flow of blood. Adequate, because proportionate to the act of volition, as is the exercise and consequent waste, proportionate also (to the act of volition) is the enlargement of the vessels and consequent flow of blood. 'Determination' of blood it may be called. But this influence through the agency of the nervous system by the act of *volition* is probable only, not actually demonstrated.

It would appear that *emotional* influence is not limited to apportioning the supply of blood, and thence the quantity of growth; the *quality* also produced is in a measure regulated thereby. For example, a fatty tumour was removed by Mr. Lawrence, several years ago, from a woman's shoulder; and when the wound had healed soundly, she imagined the tumour to have been a cancer, and that it would return. By accident, Mr. Paget saw her some months afterwards, and she had a large and firm painful tumour in her breast, which was not removed, for its nature was obscure and her general health unfavourable. Some months subsequently, having become Mr. Paget's patient at the Finsbury Dispensary, her health had much improved, but the hard lump in her breast still remained, as large as an egg, and just like a portion of indurated mammary gland. She was assured by Mr. Paget that her supposed cancer would disappear; and it did become very much smaller, without any help from medicine. As this tumour had arisen under the influence of fear, so it very nearly subsided under that of confidence. The patient was lost sight of before the tumour had been completely absorbed.

In this summary of the process of Nutrition, we recognise a mutual relation subsisting between the 'blood,' its 'vessels,' the 'nervous system,' and the 'textures,' in respect of the quality and quantity of blood supplied to the various parts of the body for their nutritive maintenance; and we further recognise adequate provision for the extra demands of growth, whereby blood is duly determined to parts growing.

Different periods of life therefore imply different degrees of activity in the function of Nutrition; and, when the body is growing, there is a corresponding liability to overflow of nutritive material, accompanied with constitutional disturbance, as exemplified by croup, scrofulous and tuberculous deposits in the lungs and mesenteric glands.

Inflammation.—The transition from 'determination of blood' to 'inflammation,' is happily *introduced* by the original observations of John Hunter.

"The very first act of the vessels when the stimulus which excites inflammation is applied, is, I believe, exactly similar to a blush. It is, I believe, simply an increase or distension beyond their natural size. This effect we see takes place upon many occasions: gentle friction on the skin produces it; gently stimulating medicines have the same effect. A warm glow is the consequence, similar to that of the cheek in a blush; and if either of these be increased or continued, real inflammation will be the consequence, as well as excoriation, suppuration, and ulceration. This effect we often see, even where considerable mischief has been done; and I believe it is what always terminates the boundaries of true inflammation. A musket-ball shall pass a considerable way under the skin—perhaps half-way round the body—which shall be discovered and traced by a red band in the skin, not in the least hard, only a little tender to the touch; and it shall subside without extending further. This appearance I shall term a blush; for although this may be reckoned the first act of inflammation, yet I would not call it inflammation, having produced no lasting effect: I should rather say that inflammation sets out from this point, and that afterwards a new action begins, which is probably first a separation of the coagulating lymph, and the throwing it out of the vessels."*

Hunter froze the ear of a rabbit and thawed it again: acute inflammation supervened, with increased heat, and considerable

* On the Blood. Inflammation and Gunshot Wounds, 1794, p. 279.

thickening of the part. The rabbit was killed when its ear was at the height of inflammation. The head was then injected, and the two ears were removed and dried. The uninflamed ear dried clear and transparent; its vessels were distinctly seen ramifying through its substance; but the inflamed ear dried thicker and more opaque, and its arteries were considerably larger.

I gather from these observations that Hunter considered incipient inflammation to be a flow of blood, persisting,—determination of blood, persisting, as a blush of redness, with heat, not to say pain; and that the turgid vessels relieve themselves by an overflow of nutritive material—lymph. The exalted nutrition of the part inflamed is denoted by *swelling*—a symptom distinguishing inflammatory from temporary determination of blood.

Accordingly, the definition of inflammation is, an over-supply of arterial blood, *persisting*, with an overflowing exudation of lymph, the motion also of the blood being increased beyond the average force and rapidity of its transmission to, and through, the part.

The latter items of this definition are established by further observation of any part in the state of inflammation. The arteries towards that part throb with increased pulsation, while the veins therefrom are turgid. It may be objected that the former are beating with blood apparently rebounding from some obstruction in advance, and that the latter become turgid with stagnant blood. But experimental observations meet this objection, and demonstrate the accelerated rapidity and force of the blood's motion *through* an inflamed part.

Let an artery toward a part inflamed be divided, and the blood is seen to be ejected to a much greater distance than that from an artery of the same size and distance from the heart, but not contiguous to an inflamed part.

This increased propulsion was noticed by Dr. John Thomson,* when the arteries of a finger were divided in whitloe, and when those of the prepuce, much inflamed, were cut in the operation for

* Lectures on Inflammation, 1813, p. 67.

phymosis. More exact, because comparative and otherwise complete, was the observation of Mr. Lawrence.* One hand of a patient being inflamed, venesection was performed, at the same time, and in a similar manner, in both arms: the vein from the inflamed hand yielded about three times more blood in a given time than the vein from the uninflamed hand. The blood's motion through the part was increased.

Inflammation, so far analyzed, is too much arterial blood in a part, persisting, with motion of that blood *increased*, and an increased exudation of lymph.

To this condition of active hyperæmia has been added, by some authorities, motion of the blood *partially diminished* in the inflamed part. Dr. C. J. B. Williams maintains *this* view,† and also that the resulting compound—Inflammation—may originate either in determination of blood or in congestion; the former implying enlargement of the arteries, the latter that of the veins and capillaries.

To illustrate congestion *following* determination of blood, Dr. Williams adduces observations made under the microscope on the web of the *frog's foot*. If, says he, a strong irritant, as a grain of capsicum or a minute globule of essential oil, be applied to the web, all its blood-vessels speedily become enlarged: those most irritated are very large and red, and the blood in them is stagnant and coagulated; contiguous vessels are also very large, but less red, and the motion of the blood in them is slow, and often in pulses or oscillations; whilst in vessels beyond, the enlargement of the capillaries is less considerable, but that of the arteries is obvious, and the current of blood is very rapid.‡

Congestion, associated with determination of blood, as seen under the microscope, by over-irritation of the web of a frog's foot, may *possibly* also occur in the *human* subject, thus fulfilling the definition of Inflammation laid down by Dr. Williams; namely—"too much blood in a part, with motion (of that blood) partially increased, partially diminished." But in *other animals* (than

* London Medical Gazette, vol. v., 1829, October 24.

† Principles of Medicine, 1856, p. 320, *et seq.* ‡ Ibid, p. 323.

the frog) congestion occasioned by venous obstruction, produced artificially, actually averts determination of blood: therefore, experimental observation equally justifies the inference, that diminished motion of blood cannot be, in the *human* subject, an *essential* part of inflammation—a process which certainly implies 'determination.'

Thus, the experiments of Macartney appear to me conclusive evidence that determination of blood is prevented in rabbits by congestion arising from venous obstruction. The most remarkable circumstance, observes this authority, with respect to congestion, and the one which has not hitherto been described, is that arteries found in a congested part are *smaller* than their natural size.

Both the jugular veins of a rabbit were ligatured; the animal died apoplectic, and upon examining the vessels of the ears, the veins which lie towards their outer edge were found greatly enlarged and gorged with dark blood; but the artery which runs in the centre of the ear was reduced very much below its natural size, so that it appeared as a mere line.

Another experiment showed the instantaneous effect of arresting the venous circulation. The mesentery of a young rabbit was exposed, and the trunks of several mesenteric veins having been tied, their corresponding arteries contracted immediately in the most palpable manner and to a very small size, as if, significantly adds Macartney, taught by their organic instinct that blood should not be permitted to go where it must immediately return.*

I may here advert to a question of great practical importance. Does not the distinction between 'congestion' and 'inflammatory determination' of blood afford a rational explanation of the inutility, and perhaps the evil results, of blood-letting in certain inflammations? Take, for example, pneumonia. I have briefly noticed the pathology of this disease at page 476, by which it will be evident that pneumonia arising from exposure to cold should be regarded as a natural effort, prolonged *immoderately*, to restore the balance of the blood's circulation. If, then, under these circumstances,

---

* On Inflammation, 1838, p. 141.

blood be taken from the venous system, the purpose of Nature is temporarily facilitated, but the heart's action, upon which restoration of the circulation depends, is proportionately weakened; so that the former advantage, incomplete and temporary at the best, does not compensate for the permanent disadvantage of an enfeebled heart when this vital organ is required to perform more than ordinary duty. Rather should stimuli be administered, and perhaps freely, in aid of Nature's effort to

> "Cleanse the stuff'd bosom of that perilous stuff
> Which weighs upon the heart."

In support of this view, I may add that Dr. Hughes Bennett, the chief opponent of blood-letting, and ex-champion of the stimulating plan of treatment in (all?) cases of inflammation, draws his conclusions *exclusively* from statistical results of fatal cases of *pneumonia*, in which blood-letting* was practised, and from clinical observations of this congestive kind of inflammation, or rather congestion and reaction, after the administration of stimulants, and recovery. All Dr. Bennett's illustrative cases of success in acute pneumonia, from Case cxxvi.† to cxxxi. inclusive, originated from exposure to cold, damp, or both. With these remarks on congestion, I resume the pathology of inflammation.

Equally untrustworthy as are experiments on the frog's foot, the bat's wing, and other transparent parts of *animals*, to determine whether congestion be an element of inflammation in the human subject, are similar observations to ascertain the condition of the capillary vessels and of their contents in the state of inflammation.

Assuming the experimental observations of Dr. Williams on the web of the frog's foot to be accurate, the dilatation, lengthening, and tortuosity which he describes in respect of these vessels, are at the best but data for conclusions, indirectly—grounds of probability only—as to the condition of the capillaries during inflammation in any part of the *human* body. No one has ever

---

* Clinical Lectures on the Prin. and Prac. of Med., 1858, p. 281 *et seq.*

† Ibid., p. 629 *et seq.*

seen the appearances of the capillaries in the human subject, either during inflammation or in the healthy condition; and although this objection may be urged against much of *human* Physiology,—which, for the most part, is purely *inferential* from observations and experiments on the lower animals,—yet this extension of an erroneous method only further illustrates the force of our argument in the present instance.

Nor can we follow with faith in the assumption that because red blood-discs become impacted, and white blood-corpuscles are apparently more abundantly developed and more adhesive than usual, clinging and lingering round and round on the walls of capillary vessels in the web of the *frog's* foot; *therefore* there is a similar blockade of these discs and waltzing of these corpuscles in the capillaries—say of the conjunctiva of the *human* eye when inflamed. In point of fact, the capillary circulation of a warm-blooded animal under the same circumstances does not present these appearances. Paget and Wharton Jones both watched and failed to discover any undue display of white corpuscles in the bat's wing, and the former authority attributes the large proportion of these corpuscles noticed in the frog to an unhealthy condition of this animal.

Rejecting the results of observation with the microscope on behalf of inflammation, it is unnecessary to refute the mere *hypotheses* of certain writers. Boerhaave's *viscidity* of the blood and *error loci* of the corpuscles; Cullen's *spasm,* and Wilson Philip's debility of the capillary vessels—*cum multis aliis,* are alike purely gratuitous assumptions.

Seeing, then, that Inflammation signifies persisting determination of (arterial) blood to and through some texture or organ, accompanied with increased effusion of lymph, what are the outward and visible signs of this hyperæmia? 'Redness,' of course, proportionate to the number of vessels and the determination of blood through them, of a florid hue, shaded by any intervening texture, or altogether unseen through the depth of integuments concealing the engorged vessels. But in some superficial textures the very arrangement of the smaller vessels can be readily per-

ceived, and various distinctive terms are commonly used to describe the appearances of such vascularity. The skin, conjunctiva, and orifices of the mucous canals, are open to inspection, and various forms of inflammatory hyperæmiæ—as punctiform, stellate, arborescent, maculiform, and uniform blush-redness—are frequently recognised. These distinctive terms sufficiently describe the particular arrangement of the vessels in the part inflamed, and they are as permanent as the determination of blood is persistent.

Redness, such as I have described, is the *earliest* announcement of inflammation, and its *most exact* indication, being invariably present, even if unseen, a sign of no other kind of hyperæmia, and the measure of its own. Redness from new vessels *developed* in an inflamed part is not an early occurrence during inflammation.

'Heat' is said to be another local sign of inflammation; and truly the same persistent flow of arterial blood, which presents a florid redness, will also occasion some increase of temperature above that which the part previously possessed; and this increase will, like the redness, be proportionate to the *number* of vessels and the *determination* of arterial blood, but perceptible only according to the superficiality of the inflamed part and the facility with which heat can be felt through the intervening textures.

Is heat actually *developed* by inflammation? The flow of arterial blood, and therefore of red blood-corpuscles charged with oxygen, may possibly generate heat. Yet the experiments of John Hunter* show that the temperature of an inflamed part never rises more than two or three degrees, and scarcely, if ever, above the average heat of arterial blood—say, from ninety-eight to one hundred degrees Fah. On the other hand, the more recent "thermo-electric" observations of Mr. Simon† and Dr. E. Montgomery are apparently conclusive on this point,—that an inflamed part is no mere passive recipient of heat, but is itself actively calorific. For among the observed results are these:—

---

* Op. cit., p. 293 *et seq.*

† System of Surgery, edited by T. Holmes, 1860. Vol. i., Inflammation, by J. Simon, p. 42.

"That the arterial blood supplied to an inflamed limb is found less warm than the focus of inflammation itself.

"That the venous blood returning from an inflamed limb, though found less warm than the focus of inflammation, is found warmer than the arterial blood supplied to the limb; and,

"That the venous blood returning from an inflamed limb is found warmer than the corresponding current on the opposite side of the body."

Granting, then, that the inflammatory process unquestionably involves a local production of heat; "to interpret this fact," adds Mr. Simon, "is perhaps, in the present state of physics, not possible."

Whatever be the heat of inflammation, it continues (with the inflammation) unlike the transient warmth of blushing, or other uninflammatory determination of blood. So also this (increased) heat is cotemporaneous in point of origin with the redness, and therefore an equally *early*, if not an equally *exact*, sign of inflammation.

More important, however, are the changes which inflammatory hyperæmia induces in the *blood*, by increasing the proportion of fibrin to its other constituents, and favouring the coagulation, separation, and contraction of the fibrin. The phenomena known as the *buffy coat* and *sizy blood* are due to these changes in the blood's constitution and vital endowments; and this 'constitutional disturbance,' thus manifested, will be more clearly understood by first observing the coagulation of healthy blood.

Let a pint of fresh-drawn blood be exposed in a shallow basin; immediately a vapour, having a faint odour, arises, which (*halitus*) was first noticed by Haller. In about four minutes a pellicle appears at the edge of the vessel, soon extending over the surface of the blood and down the sides of the vessel. It pervades the whole in about eight or nine minutes. The fluid blood is thus converted into a jelly. But in a variable period, from seventeen to twenty minutes, or much later, this jelly begins to shrink away from the sides of the basin, and the colourless, transparent serum exudes—a process which, continuing for several hours, or even days,

at length leaves a blood-red clot, floating about in limpid serum. And what is this clot? The *fibrin* of the liquor sanguinis, which has spontaneously solidified into fine homogeneous filaments, interwoven like felt, and which, as a mesh, has caught and involved the red corpuscles. They also have spontaneously aggregated, their disc-shaped surfaces cohering side by side, and forming *rouleaux*, like piles of money, which in their turn connected themselves into an irregular network; the shrinking of *this* mesh, intertwined with that of the fibrin, expressed the serum, and thus aided the solidification of the clot. Two constituents, therefore,—the fibrin and red discs,—together spontaneously aggregate to form the clot, which consists of woven filaments, involving the mesh of red discs. The expressed serum is structureless. The pale or colourless corpuscles are irregularly distributed throughout the clot and serum.

These changes are represented in the following table.

Fluid Blood.	Liquor Sanguinis.	Serum.		Coagulated Blood.
		Fibrin.	Clot.	
	Corpuscles.			

The experimental observations of Dr. B. W. Richardson* apparently demonstrate that this process of coagulation is essentially associated with the elimination of ammonia—a most significant fact. More recently, however, the observations of Professor Lister† tend to negative this conclusion.

If the blood, freshly drawn as we have supposed, be *inflammatory*, its coagulation then presents a clot, the upper portion of which is pure fibrin, of a tawny-yellow colour, and known as the *buffy coat*. This, therefore, consists of a portion of fibrin, which has coagulated apart from the mesh formed of red discs. And what is the *immediate* cause of this kind of clot? Obviously, that the red discs separated and subsided from the liquor sanguinis *before* the fibrin began to coagulate. How does such isolation *arise?* Possibly in either of two ways, or by a concurrence of both. The fibrin may coagulate so *slowly* as to allow

---

* The Cause of the Coagulation of the Blood, 1858.

† Lecture before the Roy. Soc. Lond., Lancet, 1863, Vol. ii., Nos. vi. vii.

time for the blood-discs to separate and subside. But Dr. Stokes* watched the coagulation of inflammatory blood in twenty-seven cases. In fifteen of them the buffy coat formed; in the remaining twelve it did not. In four of these twelve samples of ordinary coagulation, it began only at the end of eight minutes after venesection, and in other three of this series not until after twenty to forty minutes had elapsed—making a range between the two extremes of from eight to forty minutes. This delay of coagulation gave ample opportunity for the red discs to escape from the fibrin during its solidification; yet they did not subside, and the usual red clot formed. On the other hand, in twelve of the fifteen samples of buff-forming coagulation, the yellow clot of pure fibrin formed in only five minutes, and in the remaining three it was delayed only to ten minutes; so that, during this comparatively short period of five or ten minutes, the red particles had separated and settled down, leaving the buff-coloured fibrin free and floating. Slow coagulation, therefore, does not explain the production of the buffy coat.

The only other active element in coagulating blood is the mesh-forming discs; and how do they behave in freshly-drawn inflammatory blood? They individually possess *undue* power of aggregating (H. Nasse), and the net thus wrought has also *undue* power of contracting (W. Jones), whereby the serum is more effectually expressed from its meshes. This fabric, therefore—the component particles of which have individually greater specific gravity than the serum—has now even greater weight, bulk for bulk, and being formed earlier than usual, subsides in the serum *before* the fibrin has fairly solidified, or perhaps before this more essential element of the clot has begun to coagulate. John Hunter seems to have anticipated this view of buffy blood, and Schroeder Van der Kolk, with other observers, have corroborated it.

Diminished density of the serum of inflammatory blood—according to Becquerel and Rodier—aids the subsidence of the

---

* Brit. and For. Med.-Chir. Rev.

mesh of discs, and would also appear to account for the difficulty with which the buffy coat forms, when the blood, escaping drop by drop, coagulates.

Combining all these observations—formation of the buffy coat seems to imply an increased separation and contraction of fibrin in a free state, rather than its increased *power* of separation and contraction, and that the blood-discs are the initiative and active element in the process of buff-forming coagulation.

Slowly coagulating fibrin will, however, favour this result, by allowing more time for the gregarious blood-discs to flock together and exercise their function as a contracting mesh. If, therefore, the blood be *artificially* preserved in a fluid state, by adding serum to above its proportion, thereby delaying coagulation; the red discs aggregate and subside, and the buffy coat is presented.

Probably all the causes I have mentioned concur to produce this result, and that as they prevail more or less during the act of coagulation, so is the buffy coat of pure fibrin more or less completely established.

The earliest intimation that blood is about to undergo this kind of coagulation, is the appearance of a violet tint, not unlike the bloom of black Hamburg grapes, on the surface of the exposed blood. This appearance was, I conceive, noticed by Hunter,* and regarded by him as due to the red particles shining through a thin layer of buff-coloured lymph, just as blood in the veins gives a similar tint when viewed through the skin. This tint will therefore vary as the layer of fibrin becomes thicker. Coagulation proceeding, if the mesh of red particles separate and subside from the fibrin, but yet slowly and incompletely, and if the free fibrin but imperfectly solidifies and contracts, then a loose *sizy* clot is produced, resembling a solution of isinglass, attached to the sides of the vessel, and scarcely trembling when shaken. If, again, the separation and contraction of pure fibrin be more complete, a flat, yellow-buff-coloured cake is produced, swimming in serum; but

---

* Op. cit., p. 43.

the under portion of this clot is red as usual by admixture of the red discs. If, again, the separation and contraction of fibrin be still more complete, the fully-formed, solid, and buff-coloured clot is presented, withdrawn from the sides of the vessel, and probably concave or *cupped* on its upper surface: the lower portion of this clot having formed more slowly, has therefore contracted more strongly, and drawn down the central part of its upper aspect. The blood, or rather clot, is buffed *and* cupped. Even in such case the red particles and fibrin do not completely separate. In thirty samples of buffed blood, carefully inspected by Dr. Richardson,* he never failed to find red discs in the lower portion of the clot, and in many instances this red lower portion had the consistence of ordinary coagulum.

Besides these deviations in the process and product of healthy coagulation, inflammatory blood is found to have undergone certain alterations in respect of *chemical* composition. Its constituents, no less than their properties and endowments, are perverted. Becquerel and Rodier enumerate the following alterations in the blood of acute phlegmasiæ:—

1. An increased proportion of fibrin.
2. A decrease of globules.
3. A decrease of albumen of the serum.
4. An increase of fatty matters.
5. A decrease of soda and soluble alkaline salts.

In point of practical interest, the importance of all these chemical changes appears to concentrate in the influence they exercise on the separation of the fibrin, and its coagulability in a free state.

Alkaline salts in excess are well known to retard coagulation, and a decrease of the soda and soluble alkaline salts will have the opposite effect. Less time than usual is then allowed for the blood-discs to subside, and so far the formation of the buffy coat is *not* promoted.

---

* Op. cit., p. 335.

An increased proportion of fatty matter will probably favour the early separation of pure fibrin, by inducing it to rise with such matter to the surface (of the blood drawn), and leave the red discs below.

A decrease of the albumen of the serum will have a similar effect, by directly diminishing the specific gravity of this fluid, so that the blood-discs sink more readily. The mean specific gravity of the serum in the phlegmasiæ generally, is estimated by Becquerel and Rodier at 1027·0; and although, according to Nasse, that is about the average in health, yet the specific gravity of serum in inflammations frequently declines below the *mean* of 1027. And this is due to the proportion of albumen being reduced below the healthy average of 80 parts in 1000, to 73·35, and even as low as to 64·84. It was formerly stated by Gendrin* that the albumen rose to about twice its proportion above the standard of health. The decrease noted is in a direct ratio to the increased proportion of fibrin.

So also the blood-discs decrease in quantity proportionately to the increase of fibrin (Simon), and this reduction will facilitate their complete separation,—the more so, since, by undue aggregation of the discs, their combined specific gravity preponderates even more than they do individually.

Lastly, the increased proportion of fibrin above the average of two to two and a half parts in 1000 of blood, contrasting as it does with the reduced proportion of blood-discs, is the culminating point in favour of a clot being formed of *pure* fibrin; and this increase, and corresponding formation, of the buffy coat was noticed by Andral and Gavarret to rise as high as ten parts in 1000 of blood drawn, in acute articular rheumatism, and in pneumonia.

The source of this additional proportion of fibrin is doubtful.

Simon suggests that the blood-discs are transformed into fibrin, and in conformity with an acknowledged physiological law,

---

* Hist. Anat. des Inflammations, 1826, t. ii.

that as textures waste in proportion to their functional activity, so therefore the blood-discs disintegrate more abundantly in inflammation,—owing to their function as bearers of oxygen, to the various textures being overtaxed, in the more frequent transmission of these discs through the lungs, by acceleration of the blood's circulation. The flotilla of oxygen-laden cells perishes *seriatim* from overpressed service, and their wrecks are converted into fibrin. Simon's statement, that the discs decrease in quantity proportionately to the increase of fibrin, harmonizes with his theory; but, against it, Becquerel and Rodier urge that this destructive change ought to take place whenever the circulation is accelerated, and therefore whenever fever exists. Yet an increased proportion of fibrin is not found in other fevers accompanied with an accelerated circulation.

Is the excessive fibrin transformed albumen? Probably similarity of composition allows of such transformation; and certainly, as the albumen diminishes in quantity, so does the proportion of fibrin increase.

Of this metamorphosis we know neither the cause nor the mechanism (Becquerel and Rodier).

Summarily, the coagulation of inflammatory blood—when drawn and fresh—amounts generally to this;—the blood-discs having an undue tendency to aggregate, and the mesh they form an undue power of contracting, more speedily sink in serum, normally of less specific gravity than the discs individually, of still less than the shrunken net of discs; and this separation of the discs (before coagulation of the fibrin) is facilitated by their reduced number, and by diminished specific gravity of the serum itself. The fibrin then coagulates free of blood-discs, at least in its upper portion, and rises to the surface of the serum, its ascent being probably aided by admixture with the free and floating fat.

Such is the coagulation of a sample of inflammatory blood, such the process that sample after sample of inflammatory blood undergoes.

It might therefore be inferred that the whole mass of blood,

when subject to the influence of inflammation, is ready to undergo these changes, and that it would present the same alterations of chemical composition if withdrawn from the living body. But does the exalted tendency of the blood-discs to separate, and consequently of the fibrin to coagulate in a free state, prevail *in* the *living* body, and affect the whole mass of blood? Does the blood circulating in the system undergo any disturbance, which would then be a 'constitutional' one, and proceeding from a local morbid condition—inflammation of some part?

This question is highly important; for the *nutrition* of every part of the body, beyond that which is inflamed, must be modified by excess of fibrin in the blood—hyperinosis, and by its undue separation and coagulation. A blood-crasis bordering on the inflammatory encourages nutrition; a further degree of the same condition of blood encumbers this process with superfluous material. The former is a salutary provision to meet the exigencies of nutrition,—in growth and repair; the latter, an oppressive compulsion to overgrowth, or at least to an overflow of the redundant nutritive material. Behold, then, a reparative power; behold an impending evil.

The question proposed is at present open to much, very much, further inquiry.

Hitherto, I have analyzed the pathology of inflammation,—respecting its kind of hyperæmia (persistent determination of arterial blood), and the local signs—redness and heat; their diagnostic value; lastly, the question whether there be any disturbed condition of the blood circulating in the system,—a constitutional disturbance, and depending on the local hyperæmia.

I now proceed to consider the intimate nature of another element of inflammation; namely, effusion of the liquor sanguinis, and the accompanying 'swelling;' its diagnostic significance; lastly, inflammatory fever, and the origin of this constitutional disturbance from inflammatory swelling.

The *persistence* of inflammatory determination of blood implies effusion of lymph and serum, and consequent swelling, more or

less; unlike the issue of that temporary distension of the vessels, which ordinary determination of blood denotes.

For, from this persistent increased flow of blood, the surcharged vessels are gradually relieved by effusion of the liquor sanguinis.

Virchow* ventures to advance the hypothesis, "somewhat bold perhaps, though perfectly able to sustain discussion; that, fibrin generally, wherever it occurs in the body external to the blood, is not to be regarded as an excretion from the blood, but as a local production. Nobody"—he alleges—"has ever been able by the production of a mere change in the force of the current of the blood to induce the fibrin to transude directly, as it is wont to do in certain inflammatory processes; for this some irritation is always required." Accordingly, in respect of inflammation,—Virchow regards fibrin, not as an exudation or effusion from the vessels by persistent (inflammatory) *determination* of blood, but as "an educt, from the vessels, in consequence of the *activity* of the histological elements themselves."† Professor Bennett‡ suggests that the tissues attract the fibrin, which, however, pre-exists in the blood.

The engorged vessels themselves occasion some degree of swelling of the part; an increase proportionate to the vascularity of the part, and to the degree of turgescence of its vessels. Full-blooded internal organs become most swollen in this way, such are the lungs, liver, spleen, and kidneys when inflamed; but with persistent engorgement further enlargement ensues by effusion, and the situation, size, shape, and physical characters generally of this swelling, will, for the most part, depend on the kind of structure in which it takes place.

Liquor sanguinis is readily effused into the constituent cellular texture of the organ, or part inflamed, and therefore most readily into the substance of loosely parenchymatous organs;

---

* Cellular Pathology. Trans. for Syd. Soc. by F. Chance, 1860, p. 162.
† Ibid., p. 386. ‡ Clin. Lectures on Medicine, 1858, p. 133.

frequently, moreover, the most vascular, such as those just enumerated. Lymph and serum overflow into cavities, *e.g.*, into serous membranes and synovial sacs. These and similar structures solicit the overloaded vessels to relieve themselves, and their *size* becomes enlarged, in a corresponding measure, by inflammation. Witness hepatized lungs from pneumonia in its second stage—immense enlargement of the liver from chronic inflammation; enormous increase of the spleen, forming the *ague-cake* by an analogous process; and the immense size to which the kidneys attain by chronic nephritis ending in Bright's disease. Phlegmonous erysipelas—engaging, as it does, the subcutaneous cellular tissue deeper and deeper—is characterized by considerable swelling. Witness the opposite results in tight unyielding textures—such as effusion beneath fasciæ and in fibrous textures generally, and an abscess formed in the substance of bone, but unattended with any perceptible swelling, and suspected only by the intense and unremitting pain it occasions. Sir B. Brodie made some instructive observations* on this point. Certain textures allow of an intermediate amount of swelling between the extremes presented by cellular and fibrous tissues. Such are the degrees peculiar to the skin and mucous membranes. Of the latter I may mention pulpy thickening of the large intestine in cases of chronic dysentery, while swelling in some measure of the skin is one feature of most of its eruptions, however otherwise diversified their appearance may be—as rashes, scales, papules (pimples), vesicles, pustules; and most conspicuously this is the character of solid tubercular swellings, such as occur in secondary syphilis.

The *shape* of inflammatory swelling is also a mixed result, principally due to the kind of structure into which effusion takes place; partly, however, to the kind of matter effused. If thin serum, the swelling will be fluid, fluctuating, and diffused; if coagulating lymph be poured out, it will be more solid and circumscribed. I shall not attempt to describe the various degrees

---

* Diseases of the Joints, 1850, p. 288.

of *density* which inflammatory swelling presents, as resulting from the combined influence of the kind of matter effused, and that of the receiving structure. It would, morever, be useless to enter generally into details which can only be appreciated by personal observation, and I shall have occasion to add sufficient when describing the characteristic swellings presented by acute and chronic synovitis, and by scrofulous caries, in the next Chapter.

The diagnostic value of inflammatory 'swelling' is not equal to that of 'redness.' Regarded as an *exact* sign, although some degree of swelling invariably follows inflammatory determination of blood, and although the nature of an obscure swelling is assured, if not by its physical properties, at least by puncture, and if necessary by examining with the microscope the material of the swelling; yet these guarantees of identity are the only unequivocal advantages of this sign. Unlike redness, it is no *measure* of the degree of inflammation. The most intense may produce a trivial swelling in an unyielding texture, and a trivial degree of inflammation will soon exhibit considerable swelling in a loose tissue. Then, again, this is necessarily a *later* sign than redness, which always precedes effusion by an important and often appreciable period of time. Iritis is announced by injection of the ciliary arteries and a zone of redness around the iris, *before* the perilous effusion of lymph. Erysipelas spreads with a red blush *before* the disorganizing engorgement of the subcutaneous cellular tissue.

Subject to these disqualifications, 'swelling' is the more valuable sign, *practically* speaking, of inflammation. It can be discovered when the redness cannot be seen. In all superficial textures and parts, swelling can be readily detected. The skin, cellular texture, muscles, periosteum, bone, blood-vessels, and lymphatics, and the component tissues of the joints severally present each a characteristic swelling when inflamed. Certain internal organs are also open to examination, *e.g.*, the pelvic viscera, excepting the bladder. Thus, inflammatory enlargement of the prostate, or the uterus, and thickening of the rectum, can be

felt, and possibly seen. Certain other organs are indeed beyond the reach of vision and the direct application of the hand, yet the ear can then detect effusion and swelling by means of percussion, as of the liver, gastro-intestinal canal, and spleen; and this may be aided by auscultation, as of the heart and lungs. Under other circumstances the verdict of inflammation must rest on the evidence of functional derangement, as of the kidneys and bladder, and more particularly of the brain and spinal cord.

This leads me to another accredited local sign of inflammation—'pain.'

Observe, then, the influence of swelling. No sooner has the first contribution toward swelling been made by persisting distension of the *vessels* of the inflamed part, than pain, or at least exalted sensibility, is induced by the blood's influence on the nerves of that part. The *degree* of pain from this cause will be regulated by both the elements which determine the amount of hyperæmia, that is to say will be proportionate to the over-supply of blood, and the number of vessels in the part; but the number of sensory nerves affected will further apportion the degree of pain, and as swelling ensues from effusion, the same conditions which represent the degree of tension, will also measure the intensity of the pain. Thus a more *solid,* and therefore circumscribed effusion of coagulating lymph, underneath an *unyielding* texture, such as a fibrous membrane, *e.g.*, the fascia-lata, or a fluid similarly circumstanced, as an abscess in the substance of bone, are aggravating conditions inducing the most severe and unremitting pain; while a more fluid serous effusion into a loose texture such as the cellular tissue, say of the arm-pit, allows of a considerable accumulation without much pain, and will then be more tolerable.

The *character* as well as the degree of pain accompanying inflammation, is equally diversified. Erysipelas and certain other inflammations of the skin, occasion a burning pain, whence the popular name of this disease—St. Anthony's-fire. Inflammation of the rectum occasions a scalding pain during evacuation of the fæces. Chronic rheumatism and lumbago are attended with the

dull aching pain of inflamed fibrous and muscular tissues; gout, with a wrenching pain; abscess in bone, with an unremitting burrowing pain; inflammation of the dental periosteum—periodontitis, with a throbbing pain. In other parts this character of pain is the known forerunner of suppuration. Parts endowed with but little sensibility in health, generally become acutely sensible when inflamed. Inflammation of fibrous textures and of bone, including the teeth, exemplifies this change; so likewise does the pain of enteritis, and, in a minor degree, the stitch of pleurisy, which is also the pain of other inflamed serous membranes. Parenchymatous organs likewise acquire exalted sensibility, as manifested by the heavy, oppressive pain of pneumonia. Organs of special sense are for a time quickened by inflammation, and convey their own sensations, but too keenly. The ear becomes too susceptible of sound, and iritis begets intolerance of light.

Reflected pains in distant parts are not uncommon symptoms of inflammation. Pain in the inner side of the knee may emanate from inflammation of the hip-joint; in the glans penis, from cystitis; in the testicle, from nephritis; under the right shoulder-blade, from hepatitis; and under the left scapula from gastritis. Reflex motions may be excited in like manner: Sneezing, by catarrh; coughing, by bronchitis and pneumonia; vomiting, by gastritis; and (reflex?) micturition, by cystitis.

Respecting the diagnostic value of 'pain,' the reader is referred to Chapter II., on Functional Symptoms. Suffice it to say that the pain of inflammation being chiefly due to swelling, is scarcely an *earlier* sign. It may also be absent in true inflammation, and present without; and is rather a measure of the kind of swelling than of the degree of inflammation. By itself, therefore, pain has little diagnostic importance; yet, viewing inflammation as an "internal cause" in operation, we can from this point trace the origin of 'inflammatory fever,' and thus further illustrate the "local origin of constitutional disease."

Now the phenomena of inflammatory or symptomatic fever are well known. The heart's action is excited, the pulse becomes

more forcible and frequent than usual, and in some cases less compressible; the skin is dry and hot, the urine scanty and high-coloured, the bowels probably constipated, the fæces dry and hard; thirst and inappetency, weakness, with general nervous excitement,—restlessness, sleeplessness and hurried respiration, are also primary accompaniments of this fever. Here, then, we observe the 'vascular,' 'secretory,' and 'nervous systems' together engaged in a constitutional disorder—symptomatic of inflammation.

But if the constitutional symptoms of inflammation be familiar, *how* do they arise from this local morbid condition? What is the theory or interpretation of inflammatory fever?

Hunter apparently was inclined to attribute this excitement of the circulation and its concomitant (constitutional) symptoms to the previous excessive formation of *fibrin* in the blood. He adduces the case of a man* who was stabbed in the loins, and who, from the subsequent symptoms, was most probably wounded or hurt in some abdominal organ. At first there were no symptoms, but simply pain in the part; venesection therefore was practised by way of precaution, and the blood then drawn was perfectly natural; in less than a quarter of an hour after constitutional symptoms supervened, such as rigor, sickness, &c., and on opening the same orifice the second quantity of blood was very thickly and firmly *buffed*, having all the appearance of highly inflammatory blood; and throughout the constitutional symptoms, which lasted some time, the blood continued in this state, as proved by subsequent bleedings.

This case appears to me inconclusive, even so far as a *single* case can be conclusive. The rigor and sickness *may* have been due to nervous *shock* rather than inflammation with buffy blood; and Hunter's record does not state whether other unequivocal symptoms of inflammatory fever were present. Then, again, the same authority narrates a case in which the blood was *not* buffed, or sizy, and yet inflammatory fever was undoubtedly present. A young

* On the Blood and Inflammation, 1794, p. 314.

woman being attacked with a violent cough, oppressive breathing, quick, full, and hard pulse, bleeding gave her ease, the blood was then sizy; the symptoms returned, a second venesection also relieved her, and the blood was more sizy than before; so far all the symptoms agreed; they again recurred, and more violently; a third venesection again brought relief, but this blood was not in the least sizy, although it came from the vein very freely.

These cases are irreconcilable with the theory that inflammatory fever is due to an excess of fibrin in the blood, so far as such excess is denoted by buff-forming coagulation.

The results of chemical analysis also indicate that inflammatory fever may possibly be absent with *hyperinosis*, or present without it; and they certainly prove beyond doubt that the degree of inflammatory fever cannot be measured by the amount of hyperinosis. MM. Becquerel and Rodier estimate the *increase* of fibrin in various diseases to range from the healthy average of three to ten parts in one thousand of blood; and that a slight increase from three to five takes place in chlorosis, in certain cases of scurvy, more especially when it assumes the chronic form during pregnancy, and in erysipelas of the face; yet surely the two first mentioned diseases are not inflammatory, nor are the ordinary constitutional symptoms of pregnancy those of inflammatory fever. On the other hand, a *diminished* proportion of fibrin below the average of three in one thousand was noted in scarlet fever, small-pox, and measles; but the ordinary type of these fevers is inflammatory in a high degree. Lastly, when present, the degree of this fever does not correspond with the amount of hyperinosis. A great increase of fibrin up to ten in one thousand was noticed in acute articular rheumatism, in pleurisy, and pneumonia; and a proportion varying from five to ten was also found in peritonitis, bronchitis, and severe erysipelas of the face—diseases which are accompanied with at least as high a degree of inflammatory fever as pleurisy or pneumonia.

The foregoing facts and considerations compel us to attribute the accompanying inflammatory fever to some other source than

the blood; and the only other bond of sympathetic connexion between the heart and inflamed part is the 'nervous system.' Many years since, Abernethy suggested this channel of communication without, however, specifying his "strong reasons for believing that the inflammatory fever—the state of vigilance and delirium, convulsions and tetanus, that arise in consequence of injuries of the limbs, are produced by irritation imparted to the brain, which, by a kind of reflected operation, occasions a greater disorder of some of the organs of the body than of others, and thus gives a character and denomination to the disease."* Subsequently, Travers pointed out the agency of the nervous system, and drew the distinction between nervous excitement alone and inflammatory fever. Although he attributes this fever to excitement of the circulation from *hyperinosis*, yet he observed that the *first* morbid impression was upon the nervous system, and transmitted by the nerves of the part injured or inflamed to the nervous centre, and thence to the organs of circulation. In proof thereof, Travers urges the priority of nervous excitement in the development of inflammatory fever. "The *premonitory* symptoms—viz. headache, lassitude, disquietude, nausea, chilliness, and rigor are indications of the more or less troubled condition of the nervous centres; to these the alterations in the measure and force of the circulation, the permanent and sensible changes upon the internal and external surfaces, and their secretions *succeed*—viz., quick pulse, hot skin, dryness of the mouth and fauces, furred tongue, vitiated and scanty secretions,"† &c. I have italicised two of these words in order to bring out the force of this paragraph.

Nervous excitement may stop short of inflammatory fever, or may be followed by, and remain associated with, excitement of the sanguiferous system, and either element may then predominate. Thus, "irritation may be a symptom of fever, as fever may be an effect of irritation; but they are originally and essentially dis-

---

* Constitutional Origin, &c., of Local Diseases, 1824, p. 3.

† Physiology of Inflammation, 1844, pp. 62-63.

tinct forms of disease, and either may exist in the absence of the other."*

At page 145 of the work referred to will be found cases of "inflammation following injuries and operations;" and I think they fairly bear this construction. Some of them illustrate nervous excitement alone, following inflammation. Such, I conceive, are the histories of Mosely (p. 146), and of Woodcock and Laurisson, which immediately follow. Others, again, exhibit nervous excitement, followed by that of the sanguiferous system—inflammatory fever. Of this character is the history of Fuller (p. 152); and in some cases, as in this last, the nervous excitement ending in prostration, actually outlived the inflammatory fever, and the patients died from exhaustion.

My own clinical observations, I believe, justify me in enlarging the inference I have drawn from these cases; and, that while inflammatory fever is invariably and immediately preceded by nervous excitement, more or less, so also it never arises under any other circumstances. In short, nervous excitement is not merely a cause of this fever, but *the* only cause of it. But to prove this proposition by a general review of the clinical history and etiology of inflammatory fever would occupy too large a portion of this work.

Certain of the nervous phenomena which precede and accompany inflammatory fever are difficult of explanation. I allude more particularly to *inappetency* and *thirst*. Healthy hunger and thirst are now generally allowed by physiologists to be sensations expressing corresponding requirements of the 'system,' rather than proceeding from conditions of the stomach. "These sensations," observes Dr. Carpenter,† "bear no constant relation to the amount of solid or liquid aliment in the stomach, whilst they do correspond with the excess of demand in the system over the supply afforded by the blood; and they abate by the introduction of the requisite material into the circulating blood, even though this be not accomplished in the usual manner by the ingestion of food or

* Constitutional Irritation, 1826, p. 493. † Principles of Human Physiology.

drink into the stomach." Agreeably to this physiological provision, inflammatory fever should be attended with hunger, rather than inappetency. Albumen is the *pabulum* most extensively demanded by the tissues for their support, and its proportion in the blood declines considerably during inflammation; yet this deficiency is accompanied with the loss of appetite. Again, the secretions are suppressed, and water, therefore, retained in the blood; yet this excess is attended with incessant thirst. Further investigation is needed to clear up these anomalies. All the other phenomena which I have enumerated as emanating from the local irritation spreading through the nervous system, are symptoms of excitement mingled with exhaustion. The *sympathetic* fever—not inaptly so called—thence arising, is said to have similar *types;* the *sthenic,* denoted by forcible action of the heart, and a strong, hard pulse; the *asthenic,* pronounced by feeble and rapid action of the heart, and a quick, weak, perhaps irregular pulse. But, after all the distinctions that have been drawn between these types, they are seldom well defined in Nature. Daily circumstances will modify the character of inflammatory fever during its course in the same individual.

Does the *temperature* of the body rise during this fever? Dr. Thomson states that it ranges from the low extreme of 94° up to 107° Fahrenheit. This is partly due at least to suppression of the perspiration, and, therefore, the retention of that heat which would otherwise pass off by evaporation from the skin. But the excited nervous system probably contributes to the production of heat. Sir B. Brodie's experiments* show that if the encephalon be removed, the body speedily loses its temperature; a doctrine subsequently confirmed by the experiments of MM. Le Gallois and Chossat. Certain pathological observations also lead us to infer that the loss of nervous influence in any part is accompanied by a loss of temperature there.†

---

* Physiological Researches, 1851, repub. from Phil. Trans.

† See Med.-Chir. Trans., vol. vii., H. Earle; also, Human Physiology, Dunglisson, 7th edit., vol. ii., p. 238.

If, then, such be the physiological influence of the brain and nervous system in relation to the ordinary temperature of the body, may we not infer that the (pathological) influence of nervous *excitement* will be to evolve the heat of fever?

The secretions are arrested and their composition perverted probably by the same influence; for they also are regulated by the nervous system in health.

Febrile urine is at first characterized by its deep red colour, strong urinous smell, super-acid reaction, high specific gravity, and scanty quantity secreted in a given time. This deficiency is chiefly owing to a reduced proportion of water, rather than of solid constituents, in the urine; which has thus become concentrated. Dr. Day* adds, that the constant characters of febrile urine are the diminution, both relatively and absolutely, of the inorganic salts, especially of the chloride of sodium, with augmentation of the uric acid and urates. Even when febrile urine does not deposit urates, it always contains an excess of uric acid. Urea is increased in some cases, and probably diminished in others. Heller observed the greatest quantity of urea in meningitis, the whole urine solidifying in a few minutes into a crystalline magma, on the addition of concentrated nitric acid. Excess of urea was also found during exudation, but diminished during resorption, in pneumonia, in pleuritis, and in acute rheumatism, especially if endocarditis be simultaneously present. In the beginning of typhus, urea is increased, but not so much as in the diseases before mentioned. Extractive matter is generally increased in febrile urine, and lactic acid is often present. Occasionally, a small quantity of albumen is found, but only for a short time. (Becquerel and Rodier.) As inflammatory fever declines, the urine deposits plentifully a lateritious or brick-dust coloured sediment, consisting of urate of ammonia.

The quantity of sweat is much diminished during inflammatory fever, but its chemical composition at that time is not well under-

---

* Op. cit., p. 343.

stood. Ulcers, also, which have been discharging freely become dry. The flow of saliva is less free, and the tongue *furred.* This appearance arises from a material of whitish yellow or brown colour and firm consistence, overlaying the posterior and middle portion of the tongue on its upper aspect, and adhering closely. It cannot be removed altogether by scraping, but as the fever declines it is shed spontaneously. If, says Dr. Thomson, this fur arose from the nature of the saliva secreted, then, instead of being found only on the upper, middle, and posterior parts of the tongue, we should find it incrusting the whole internal surface of the mouth. It is probably secreted from the papillæ to which it adheres. A similar appearance arises from irritation of the stomach, without any fever at all. Discrimination, therefore, is necessary, by considering whether other symptoms *concur*.

Thus far respecting the pathology of inflammatory fever, and its origin from the local irritation of inflammatory swelling.

But does the sympathetic fever arise in every instance from inflammation? It has been alleged that febrile symptoms may *precede* the local, as in erysipelas, small-pox, measles, and all exanthemata. All internal inflammations arising from external cold are also said to be preceded by inflammatory fever, which cannot, therefore, in such cases, be termed 'sympathetic.'

To determine this question, take, for example, pneumonia. In most cases, inflammation of the pulmonary texture is consequent on the intropulsion of blood by exposure to cold. The superficial blood-vessels contract under the influence of cold, and becoming comparatively empty, the blood retires from them, and the area of the 'systemic' circulation is diminished. More than an appropriate share of the whole mass of blood is now circulating through the systemic vessels, and in due course the 'pulmonic' system receives an extra portion of blood (from the systemic veins), thereby relieving all the systemic vessels, and re-adjusting the balance of the blood's circulation. But the larger vessels affected by exposure to cold are *veins,* they being superficial; they are, therefore, most

excluded from the systemic circulation, and accordingly the venous half of the systemic circulation is comparatively more surcharged than the arterial portion. The deeper systemic veins being thus engorged, convey their load to the right side of the heart, thence through the pulmonary artery, to accomplish the readjustment of the circulation by distributing their (venous) blood throughout the lungs. These organs become much congested. The heart, overtaxed, fails, for a time at least, to propel the blood through distal pulmonary vessels, as it also fails to force the blood through the more distal systemic veins leading to the surface of the body; the pulmonary veins become engorged with blood, motionless, or nearly so. By-and-bye, the heart arouses, as it were, from its lethargy, and makes an effort to restore the systemic circulation, and to clear the pulmonic veins of their superfluous blood. This is *reaction*, attended with a glow of returning warmth and colour; and, simultaneously, determination of blood through the lungs. Reaction does not precede the determination of blood, and if it advance only so far as to *restore* the average force and frequency of pulse peculiar to the individual, cannot be called fever; and if beyond this standard, and the determination of blood be also persistent—in fact, inflammatory—even then inflammatory fever arises simultaneously with inflammation, and does *not precede* it.

Bearing in mind the pathology and origin of inflammatory fever, as now traced in its clinical history, aided by experimental observations, it will be obvious that we cannot regard 'swelling' by effusion as a "termination," often so-called, of inflammation. On the contrary, from the *tension* wrought by effusion, proceeds, through sympathy of the nervous system, the full development of inflammatory or sympathetic fever.

Inflammatory hyperæmia may indeed subside by the timely removal of the cause of local irritation, and the vessels regain their normal dimensions before the supervention of much effusion, if any, and perceptible swelling. The redness fades away, the part loses its exalted sensibility, and the incipient fever passes off. This is *resolution*.

But with the supervention of much effusion and swelling, an internal cause, and one therefore less easily removed, comes into operation, and the concomitant fever becomes fully established. I shall hereafter illustrate the diagnostic value of swelling, by the diagnosis of inflammatory diseases of the joints. It offers *the* opportunity for remedial measures to anticipate and prevent the supervention of constitutional disturbance.

A correct knowledge of the pathology of inflammatory *effusion* will much facilitate the earliest and most exact diagnosis of this 'swelling;' and this knowledge will moreover supply certain guiding principles in aid of removing the effused products, whereby the probable consequences of effusion may be *prevented;*—suppuration with its wasting hectic, and sloughing or mortification probably, with its typhoid type of fever. On these grounds I resume the pathology of inflammatory effusion.

Analogy might lead us to expect a general resemblance between the processes of normal healthy nutrition and inflammatory effusion;—that the 'composition and vital endowments of the blood' predispose to the formation of certain products, and that this process is affected by the 'degree of inflammatory hyperæmia,' and by the state of the general circulation; moreover, that it is regulated by the 'nervous system,' and completed by the 'secretory power of the inflamed structure.'

How far does this anticipation accord with known facts?

Take the condition of inflammatory blood, and of the blood-vessels in an inflamed part. Serum exudes from the engorged vessels, and fibrin is effused in a fluid state. But inflammatory blood superabounds with fibrin, and therefore tends to deposit it more abundantly. The *liquor sanguinis* effused is, for the most part, fluid fibrin, and this character of *plasma* represents the first product of inflammation before the formation of filaments of exudation or pus-corpuscles. It is that which appears in the first instance on blistered surfaces, as demonstrated by Mr. Paget's observations.*

* Op. cit., vol. i., p. 338.

The *quantity* of liquor sanguinis, and therefore of fibrin, effused, is further regulated by the degree of hyperæmia, aided by the force of the general circulation.

Virchow denies this proposition. He argues* that nobody has ever been able to prove the affirmative experimentally; that "nobody has ever been able, by producing a mere change in the force of the current of the blood, to induce the fibrin to transude directly, as it is wont to do in certain inflammatory processes; for this, some irritation is always required." I shall presently recur to the latter clause in this paragraph; but the want of experimental proof that fibrin is produced in an inflamed part, by the force of the blood's current, is, I think, compensated by familiar observations quite as conclusive as any experiments. When the pulse is strong and hard the blood flows forcibly through unyielding vessels, and tells on an inflamed part; then fibrin is produced abundantly in that part: when again the pulse is rapid rather than strong, and compressible, the blood is misdirected; then less fibrin is found in the part inflamed. The former condition is illustrated by common phlegmonous inflammation, with *much* fibrin; the latter, by erysipelas, with *much* serum. This large proportion of fibrin, or of serum, does not, it is true, necessarily accompany only those states of the local and general circulation to which I have referred. The same results may possibly proceed from, and denote an excess of fibrin, or of serum, in the *blood;* the presence of the blood's circulation in an inflamed part may not be *the* (only) cause of a serous or fibrinous effusion: but whenever the circulation is active and tense, then there is a tendency to the effusion of fibrin; whenever the circulation is feeble and lax, then a tendency to the effusion of serum. The inference obviously suggested by this invariable sequence is, that a mere change in the force of the blood's current is *a* cause of fibrinous effusion. It is recognised as such in practice. Phlegmon and erysipelas are regarded as representing almost opposite conditions of the circulation.

---

* Cellular Pathology, p. 163.

The *sthenic* and *asthenic* varieties (not types) of inflammation have long since been recognised, and they suggest respectively a phlegmonous and erysipelatous character of inflammation. Other varieties proceed rather from the *quality* of the fibrin effused. *Scrofulous* inflammation produces much fibrin, yet the hyperæmia is asthenic; the fibrin being thin easily transudes, and coagulates imperfectly, forming a flaky and curly clot, with much serum and pus. *Diphtheritic* inflammation also produces a fibrinous effusion, nevertheless the hyperæmia is asthenic: but the lymph is far less tenacious than in health, and forms slimy pellicles which scarcely hold together. *Hemorrhagic* inflammation is likewise asthenic, but it denotes a disintegration of the blood-corpuscles, their thin colouring matter readily transuding and tinging the loose and sizy clot. Purpura and scurvy are familiar examples of this diathesis.

Thus, then, we may ascribe the ordinary varieties of inflammation to different conditions of the blood and circulation, or to both. Beyond these sources of lymph-effusion, it is possible that the character of this deposit may be influenced by the nervous system; but the share of its influence, if any, must be determined by future inquiry.

Lastly, the structure in which inflammation takes place, may itself exercise some power, either by selecting the fibrin from the blood, thereby inducing its effusion, or by producing it in the inflamed part through metamorphosis of the material effused. In other words, an inflamed structure may possess either a 'secretory,' or a 'plastic' and 'metabolic,' power.

Virchow adheres to the latter view, and maintains by experimental observations, that irritation induces the effusion of a *fibrinogenous* substance,* which can be converted into fibrin. In proof of the self-sufficiency of irritation, Virchow adduces the operation of a blister; that firstly, serum only is yielded, but if the irritation

---

* Op. cit., pp. 159, 60.

be more violent, a fluid which coagulates. The general doctrine advanced is thus stated: "A patient who produces at a certain point a large quantity of fibrin-forming substance, much of it passes from that point into the *lymph*, and finally into the blood. The exudation may therefore in such cases be regarded as the surplus of the fibrin formed *in loco*, for the removal of which the lymphatic circulation did not suffice. As long as the current of lymph does suffice, all the foreign matters which are formed in the irritated part are conveyed into the blood; but, as soon as the local production becomes excessive, the products accumulate, and in addition to the hyperinosis, a local accumulation of fibrinous exudation will also take place."*

In objection to this theory, I would urge the very facts by which its author endeavours to support it. Virchow has unhappily referred to the products formed by the operation of a blister, in proof of the plastic power that an irritated structure possesses. But the products of a blister are formed on the immediate *surface* of the true skin; and surely the cuticle possesses no degree of plastic power. I see nothing more in the process of blistering than an increased and persistent flow of blood to the part—inflammatory hyperæmia—and therefore an overflow, first of the serum, and then of the liquor sanguinis. The vessels from which this effusion comes are disposed on the surface, and similar exudation takes place on other *surfaces* as on serous membranes, without the intervention of any structure to elaborate the fibrin. The effusion is first fluid, then more solid, spontaneously assuming the characters of fibrinous deposit.

More consistent with known facts is the operation of a 'secretory' power, by which structures select this or that constituent of the blood as it passes through them, and by which the effusion of that particular constituent is determined. An approach to the proof of this theory is, I conceive, the behaviour of the *same* blood in *different* textures. Blood, having the same composition

* Ibid., pp. 165, 166.

and properties, deposits in one inflamed part much fibrin; in another, rather albumen in a third, more fatty matter. In pleuro-pneumonia, for example, *fibrin* and a large proportion of serum are effused from the inner surface of the pleura; an *albuminous* matter is deposited in the lung-parenchyma; and pus, in which *fat* abounds, is more readily secreted from the (bronchial) mucous membrane. These products are known ingredients of the *same* blood, flowing alike to each of the three structures mentioned, and yet they severally receive a different kind of deposit. The inference is, that the particular deposit is selected or secreted by the particular kind of texture. How far this secretory power is a vital property; how far it results from the physical construction of the texture, is an open question. Assuredly the physical consistence and permeability of a part will very much affect its capability of receiving this or that kind of effusion.

Probably *all* the causes of effusion *co-operate*. The 'composition and properties of the blood,' the 'hydraulic state of the circulation—both local and general,' the 'secretory power of the structure,' and perhaps 'nervous influence,' are each engaged; and sometimes serum is the prevailing product, sometimes fibrin, as I have explained. This latter soon coagulates, and the effusion, at first fluid, assumes the physical character of a mixed product—partly solid, partly fluid, varying in this respect as either fibrin or serum prevails.

The *kind* of 'swelling' consequent on inflammatory effusion will be recognised in many cases by these *à priori* considerations; but if necessary, the diagnosis of a chronic swelling of doubtful character can be completed by puncturing its substance, and examining with the microscope the very matter effused.

Inflammatory effusion undergoes *plastic* changes beyond coagulation of the fibrin. Plastic or fibro-cells may form by the elongation of cells which assume an oat-shape; elongating more and more, they become attenuated into filaments. This fibro-cellular or connective tissue is the highest product of inflammation. Usually granules appear, and exudation-corpuscles are formed, apparently by their coalescence into little round irregular masses,

each of which sometimes acquires an investing cell-membrane. (For a descriptive account of this fibrinous deposit, these cells and corpuscles, see the introductory "Elements of Pathological Anatomy.")

They are not the only products evolved in inflammatory effusion. Pus-corpuscles may also form, apparently by a modification of the same process of cell-development which evolves exudation-corpuscles; and pus may be, or become, the prevailing product.

The process of pus-cell formation probably resembles that of exudation-corpuscles. Granules aggregate and constitute a compound granular mass. An investing cell-membrane gathers around this as a nucleus, which nucleus, by liquefaction of its peripheral granular matter, is left free, yet imbedded slightly in the cell-wall. Such, apparently, is *pyogenesis*. (The structure, properties, and composition of pus are described in the introductory "Elements of Pathological Anatomy.")

These plastic changes, viz., coagulation of the fibrin, and the formation of exudation and pus-corpuscles, are exhibitions of an *inherent* plastic power, which *all blastema* possesses in a greater or less degree. I shall not pause to examine the arguments in favour of this view of coagulating lymph; but the operation of a plastic power can, I think, be demonstrated by the circumstances under which pus is sometimes produced. At least, such is the construction I put on certain experiments made by Dr. Wood. He laid small plates of mica on a healthy granulating sore, which had been previously sponged dry. They became damp in about two minutes, and when examined the blastema was found pellucid and structureless, or only granular. In a few minutes more, another plate showed flattened epithelial cells; and in ten minutes a third plate presented the same cells, with also well-marked pus-corpuscles. Here then was a *spontaneous* evolution of pus-corpuscles. I therefore pass over the untenable hypotheses* which have from time to time been advocated to explain the production of pus by dis-

* See works by Boerhaave, Gendrin, Carswell, Donné, Gorter, Quesnai, De Haen, Sir J. Pringle, Gaber, B. Bell, Hoffman, Grashuis, Kaltenbrünner, Dupuytren.

integration, or by chemical transformation—either of the blood-corpuscles, lymph, or of the textures involved by inflammation.

Pus is a *new* product; but its evolution appears to be governed by conditions analogous to those which regulate the formation of other inflammatory products, and those of nutrition in general. 'Secretory power' is in operation, regulated probably by 'nervous agency,' and determined by the 'condition of the blood;' possibly also by the 'flow of that blood to the part.'

Pus has been termed a secretion; and its constant production in connexion with certain textures, rather than others, although the blood be the same, plainly indicates that such parts possess and exercise some secretory power.

The readiness with which mucous membranes suppurate is well known. Bronchitis, enteritis, and cystitis are prone to induce purulent sputa, fæces, and urine, respectively; indeed, pus has been found on a bougie five minutes only after it was introduced into the urethra. Other textures are far less prone to suppurate. An incision through the skin and subcutaneous cellular tissue probably unites by adhesion, without any suppuration, or pus is not produced for two or three days.

Observations such as these tend to establish the theory, first advanced by Simpson,* that pus is a secretion. Certainly it is as much so as cuticle and all other tissues. They are evolved from a blastema, effused from neighbouring vessels; and pus is also the product of a blastema, but which is effused by inflammatory hyperæmia. The doctrine of pus-secretion was likewise suggested by De Haen; and a few years afterwards, Dr. Morgan† fully discussed the whole question. Brugmann‡ followed with a similar view; and at length Hunter§ adopted and supported this secretion theory, without, however, having first suggested it, as is sometimes asserted.

---

* Dissertationes de re Medica, 1722.

† Puopoises sive Tentamen Medicum de Puris confectione, 1763.

‡ Thesis de Puogenia, 1785.

§ Blood and Inflammation, 1794, p. 417 *et seq.*

The pus-forming power of textures is probably regulated, like other secretory power, by the nervous system. In paraplegia, cystitis usually ensues, and this paralytic cystitis produces purulent urine in a more marked degree than cystitis arising from other causes. Injury of the fifth pair of nerves is followed by suppuration of those parts which they supply. Such cases show the effect of intercepting the nervous influence to a part; but the nervous system exhibits its influence by inflammation and suppuration of a part through *sympathy* with some irritation in another and perhaps distant part. Mr. Paget refers to a specimen* where extensive deposits of lymph and pus were found in the testicle of a man whose urethra contained a portion of calculus impacted after lithotrity. Analogous cases are on record. The influence of mental emotion in producing inflammation with speedy suppuration is manifested by the following case,† and of which there are similar ones. A lady was watching her little child at play, and she saw a heavy window-sash fall upon its hand, cutting off three of its fingers. In a short time the mother also had inflammation of the corresponding three fingers of her own hand, and in twenty-four hours *pus* was evacuated by incision.

Pus-production is determined very much by the condition of the blood. One would suppose so, judging from the analogy between pus-secretion and other efforts of the secretory power. Certain experiments made by Mr. Paget supply apposite illustrations. They show that the same tissue, inflamed by the same stimulus, and as near as possible in the same degree of inflammation, yields, in different persons, and in whom therefore the blood may be considered dissimilar, different forms of lymph. The inference is obvious, that blood-conditions determine the kind of product which shall be formed, or, as we may say, secreted. I have elsewhere in this work noticed this doctrine and these experiments. Blisters raised by cantharides in thirty patients gave

---

* Museum of St. Bartholomew's Hosp., Ser. xxviii., No. 55.

† Pathology and Treatment of Hysteria, Carter, 1853, p. 24.

sometimes a fibrinous, sometimes a purulent product. It was found that in cases of purely local disease, in patients otherwise sound, the lymph formed an almost unmixed coagulum, in which, when the fluid was pressed out, the fibrin was firm, elastic, and apparently filamentous. Whereas, in cases at the opposite end of the scale, such as those of advanced phthisis, a minimum of fibrin was concealed by the crowds of *corpuscles* imbedded in it. Mr. Paget therefore concludes that the highest health is marked by an exudation containing the most perfect and unmixed fibrin; the lowest, by the most abundant formation of corpuscles, and their nearest approach, even in their healthy state, to the characters of *pus-cells*.

Has continued determination of blood any influence in producing pus? It has been alleged, that "such a result is most likely to ensue in complex and highly vascular structures, where the effused matter is retained in intimate contact with the blood-vessels; hence intensity and continuance of inflammation in the true skin, cellular textures, glands, and most parenchymatous organs, pretty surely lead to suppuration."* And in explanation of this process, it is suggested by the same authority, that, as under the exaggerated "influence of the red-corpuscles (which convey oxygen) on the liquor sanguinis, more of its *protein* passes into the state of solid deutoxide,—a material fitted for organization and reparation; so we may infer that the excessive degree or continuance of the same action may overdo this change, give chemical properties an ascendancy over the vital powers, and by turning the most recently-formed solid into a fluid tritoxide, may effect a work of separation and destruction, involving the blood in the obstructed vessels, and extending to the albuminous matter of the containing texture."

This chemical theory needs no further refutation than the now acknowledged fact, that protein itself is a chemical myth, having no existence, and of course therefore incapable of forming a

---

* Principles of Medicine, C. J. B. Williams, M.D., 3rd Edit., p. 364.

deutoxide, and then a tritoxide. But what influence does persistent determination of blood exercise, under the circumstances of pus-formation, in complex and highly vascular structures? If an incision be made when pus is about forming in a large boil, an abundance of solid lymph is seen agglutinating the cellular texture. Pressing as this does upon the vessels which it encompasses, they become, at least partially, occluded. Inflammation being far advanced, 'obstruction' is *now* added to 'persistent determination' of blood. And this obstruction—this solid lymph and agglutinated cellular texture, may extend for some distance, giving the external swelling a broad base. Around the periphery of the solid swelling blood plays freely, yet without pus forming there; whereas, in the *centre*, first appears a white spot of pus, far removed from the circumferential determination of blood. This afflux of blood goes on depositing fresh lymph, thus enlarging the swelling; while in the midst of the solid lymph and *occluded* vessels, more and more removed from all such external influence, suppuration proceeds. Persistent determination of blood, therefore, only prepares a structure for suppuration; suppuration itself is an independent process. Consequently, we are not surprised to discover pus in a texture where determination of blood cannot have availed much; as in the centre of a large fatty tumour, itself but ill-provided with vessels for its own supply of blood; and the freest afflux of blood without suppuration, as in a gouty toe.

The pathology of *suppuration* throws some light on the nature of its local signs and symptoms; moreover, their diagnostic value can thus be estimated.

With each stroke of the heart arterial blood is propelled against the surface of the solid lymph, through all the vessels which surround it at the seat of inflammation; and in each vessel the blood rebounds, so as to occasion a *throbbing* or pulsating pain. The heat of the part is somewhat increased, and the bright tinge of arterial blood overspreads the swelling.

Now, these signs are usually said to betoken approaching suppuration. But, proceeding as they do immediately from deter-

mination of blood—a condition not essential to suppuration—these signs are not invariably present or trustworthy. They may occur without suppuration ensuing. Inflammation in an unyielding texture is accompanied with throbbing pain; toothache being a familiar example. Again, in loose parenchymatous textures which admit of a more free flow of blood, extensive suppuration may have taken place without any previous throbbing, and be altogether unsuspected by the individual. Under these circumstances I have not unfrequently opened an abscess in the ischio-rectal fossa, and given vent to several ounces of pus which had formed there without the accredited premonitory signs of suppuration. Perhaps the diagnostic value of these symptoms is fairly represented by saying, that when suppuration *does* occur in unyielding textures, it is invariably preceded by throbbing, with some increased heat and redness; otherwise they are not constant signs of approaching suppuration. Some inference may be drawn from the *size* of the swelling. As the afflux of blood increases towards the period of suppuration, the swelling enlarges with fresh deposits of lymph; but this sign, like those before mentioned, will be modified by the kind of texture in which suppuration is about to commence. In yielding textures, such as the loose cellular tissue around the anus, considerable effusion and swelling may supervene without suppuration; while in unyielding textures, such as around the fangs of a tooth, an abscess will form, without an increasing swelling of the gum. No connexion, therefore, necessarily exists between suppuration and this symptom; yet here again, when suppuration *does* occur in a loose yielding texture, it is perhaps invariably preceded by some increase of swelling.

But the physical properties of pus confer certain distinctive characters on any soft part in which it is formed. Just as the first effusion of lymph is recognised by a semi-solid swelling, arising from the products—fibrin and serum—into which that lymph has become resolved; so now another product—fluid, but of creamy consistence, and slightly viscid—being diffused, imparts a *more elastic* feel to the swelling. It is the sign of *diffused* sup-

puration, and this condition may continue. Or fresh lymph may be deposited so abundantly round about the focus of suppuration as to effectually circumscribe the matter and imprison it. Simultaneously with this process of construction, destructive operations are going on within the barrier of lymph. The encompassed tissues are resigning for ever their vital powers, and are yielding to the dominion of chemical and physical forces. Deprived of blood, the textures enclosed with lymph, die, disintegrate, and dissolve; and thus prepared for, are then removed by, absorption. The new product—pus, mingled with some *débris* of the old textures —now occupies their place. Suppuration has become victorious over healthy nutrition, and the suppurative process is established. A pus-containing cavity has been constructed, the walls of which are lymph, supported by the textures around. An *abscess* is formed.

This circumferential lymph at length assumes the structure somewhat, and the characters, of a mucous membrane, and acquires a secretory power. It becomes pus-forming, as well as pus-containing. It is a *pyogenic* membrane. Richly provided with blood-vessels, and well organized, this membrane, like other structures of equal organization, is not formed at a very early period of the abscess; and when formed, is fashioned gradually. A distinct membrane was found lining an abscess in the lung of a person who died of pneumonia after seven days' duration (Grisolle). When perfectly formed, the pyogenic membrane absorbs old pus, as well as secreting new; and the abscess is gradually concocted, or ripens.

Pathology, as usual, supplies a rational explanation of the concomitant 'signs,' which are now those of *abscess*.

The throbbing pain, heat, and redness—in fact, all the signs of previous active hyperæmia, are mitigated by the yielding resistance of pus as *compared* with that of solid lymph; unless the pus be confined under any unyielding texture, such as a fascia, or within the substance of bone. Then indeed this fluid resists with hydraulic pressure against the force of the afflux of blood, and these

local signs are aggravated with each stroke of the heart. The swelling assumes characters agreeably to the physical properties of pus, circumscribed. A *fluid* and *fluctuating* swelling, therefore, is now presented, rather than one having the solidity of effused lymph, or the more elastic feeling of pus diffused.

Like any other structure, the pyogenic membrane may lose its *functional* power, or possess it unimpaired, or acquire a higher degree of that power. If the former, then pus-secretion failing, while absorption continues and prevails; the abscess dwindles and eventually disappears. If secretion and absorption be equally balanced, the abscess is maintained in a stationary condition, possibly for some time. Should secretion prevail, as usually it does, and pus accumulate, the pyogenic membrane grows also, and enlarges in a corresponding measure to accommodate the encroaching secretion of pus. Moreover, this membrane changes its characters in becoming hypertrophied with the more active exercise of its functions; in a *chronic* abscess of three or four months' duration, it appears as a soft spongy enclosure of lymph, slightly mammillated, and of a greyish or reddish-brown colour after death. This sac is attached to the surrounding textures; sometimes closely, sometimes loosely. They however are invaded by the expanding abscess, and being absorbed to make room, the area of the fluctuating swelling enlarges.

At length, though at a variable period—usually before the abscess becomes chronic—absorption of the pyogenic membrane itself begins under this expansion by constant fluid pressure. The abscess is ready to burst, and generally, in the direction of least resistance, the abscess *points*. Macartney states that on the side of the abscess opposite to this thinning portion, the pyogenic membrane actually grows thicker, and contracts so as to exercise some degree of expulsive power. The circumferential tissues are absorbed more and more in the direction of the pointing, for the expanding force there gains advantage. A prominent point appears, over which the skin assumes a *dark livid* tint, and the cuticle desquamates in eccentric rings.

Here fluctuation is most readily felt. Soon this point cracks, and pus issues forth. The distended abscess is relieved; and if the aperture be sufficiently free and dependent, or made so, then, as the pus drains away, coagulable lymph is effused instead, which, forming granulations, contracts towards this opening, and gradually closes the cavity of the abscess. Around the opening also granulations of lymph spring up, which contract. In fact, the abscess is now a healing *ulcer*, and the process of reparation is completed in a period varying from, perhaps, twenty-four hours to days, weeks, or months.

Sometimes reparation is incomplete; the sac of the abscess is brought together only here and there, leaving intervening cavities in which pus is reproduced, forming a *multilocular* abscess. Or the abscess contracts into a narrow channel or *fistula*, characterized by having a perfect pyogenic membrane throughout its course, and an obstinate indisposition to heal. It is convenient to distinguish a *sinus* by the absence of these characters.

Sometimes the process of reparation is unhealthy and without tendency to heal. The sac secretes a fetid sanious pus, induced possibly by the admission of air, or by some constitutional disorder affecting the pyogenic membrane, and the healing process is delayed. Or, at a later period, when the abscess is reduced to an ulcer, it, like ulcers otherwise produced, may exhibit an indisposition to heal. From constitutional causes chiefly, this ulcer may become indolent, irritable, inflamed, phagedænic—differences of character which relate rather to 'the Constitutional origin of Local disease.'

The situations where abscess may be *formed* are regulated very much by the different kinds of texture. Abscess is rarely, if ever, formed *in* fibrous or cartilaginous tissues, and *within* any serous membrane is almost unknown. The situation where abscess may be *found* is quite another question. An abscess may be extended from where it forms to another locality. "Abscess by translation," as this is termed, somewhat inaptly, is favoured by the difficulty with which pus produced in certain parts finds its way to the surface; by the

comparative facility with which it travels to other parts; by the slow progress of the abscess, giving opportunity for its extension; and by special circumstances conducing, which are peculiar to each case. Thus, a psoas abscess, formed in the lumbar vertebræ, will travel by extension to distant parts, and ultimately is found pointing in the thigh, having been guided down by the sheath of the psoas muscle; or at the obturator foramen, having been guided by the obturator vessels and nerve; or at the vagina; or at the sacro-sciatic foramen; or at the external abdominal ring. In one case, which occurred at the Royal Free Hospital, I found a psoas abscess pointing midway between Poupart's ligament and the umbilicus, about three inches to the right of the middle line. The patient, a groom, had strained his back some months previously, while riding a horse he was training. When admitted to the hospital, the tumour subsided in the recumbent position, and disappeared under pressure. It fluctuated, and pressure with one hand communicated a sense of fluid resistance to the other hand, placed over the right flank and just above the ileum. It was unaffected by coughing. Mr. Fergusson, who, among others, saw this interesting case, was clearly of my opinion as to its nature, and the abscess was relieved by a valvular opening with a bistoury.

Abscess by translation is further illustrated by the elbow giving issue to an abscess originally formed in the shoulder-joint; and that which originated in the hip-joint may present at the knee. An instructive case came under my care at the hospital, in December, 1856. A man was admitted with a considerable swelling of the right knee. Neither the shape nor character of this swelling was peculiar, but it disappeared entirely when he laid down. In that position, I noticed a slight fulness appear a little below the great trochanter, and by gentle palpation to and fro, as in the last case, I felt a fluid communication between this upper part and the knee. Pressure above partly reproduced the swelling about the knee. The man told me that formerly some matter had been let out from below the trochanter, and that ever since he had experienced no pain or inconvenience; but that subse-

quently the knee became swollen, of which he now complained as being painful and disabled in walking. This history completed my diagnosis.

The general physical characters of an abscess such as I have described are obvious. A fluid fluctuating swelling, like that of an ordinary abscess *in situ*, while its concomitant redness, heat, and pain are *absent*. Some degree of inflammation may, indeed, ensue from the weight of pus on the most dependent portion of the abscess; and pain also may be experienced, owing to pressure on nerves in the neighbourhood. Nothing, however, beyond a careful examination of the swelling can ensure an early and exact diagnosis. And this leads only to the discovery of a 'translated' abscess; the *more early* detection of the original abscess—*i.e.*, in the first situation of its formation—is necessary in order to prevent its subsequent extension to another locality.

Of analogous importance are the earliest and most essential signs of suppuration and of abscess, in relation to the constitutional disorder thence arising; which, when fully developed, is known as 'hectic fever.' The earliest symptoms of this fever may partly be inferred from the pathology of 'inflammatory fever.'

Hectic, as a consequence of inflammation, derives its origin from *relaxation* of that tension which inflammatory effusion had previously exercised; and this relaxation is occasioned by the formation of pus. Commencing at a variable period in the course of inflammation, the first symptoms of hectic are those of prostration with excitement. The patient feels as if some source of irritation had been removed, or at least relieved; and therefore its constitutional effects—nervous excitement and inflammatory fever—subside also. A sensation of chilliness and a shivering fit—in fact, *rigors*, more or less severe—announce this consummation. Still nervous excitement prevails. The patient continues restless and sleepless, and the pulse retains its frequency, although losing its force and hardness; the heat and flush of skin subside, and the body becomes swathed with a cold, clammy sweat; the urine is pale, abundant, and deposits a pinkish sediment of lithates. Other

secretions, and the digestive process, are less restored. The tongue loses its brown fur, and assumes a bright scarlet hue at the tip, but is otherwise white and pasty; the appetite is capricious, sometimes absent, with loathing of food, and even vomiting; while profuse watery diarrhœa succeeds to the previous constipation. Progressive emaciation and loss of strength bespeak the failure of nutrition, and the general course of the symptoms at length proclaims the victory of exhaustion.

Hitherto the battle has been uninterrupted; inflammatory fever has *continued* to subside, perhaps without intermission; if, however, the struggle be prolonged, it becomes *intermittent*—exhaustion still, but with recurring efforts of excitement, particularly of the circulation, followed by increased secretions from the skin, kidneys, and bowels. That is to say, towards evening, after a day of dread exhaustion, and when the blood has quite forsaken the skin, a chill is felt; soon after the pulse acquires some force as well as increased frequency, rising, perhaps, from 100 to 130 beats, or more, per minute. Then the hollow cheeks are crowned with a tint of crimson hue, and the sunken eyes are lustrous, giving to beauty a complexion and an expression of intelligence which are not of this world. The palms of the hands and soles of the feet feel hot and dry. During the night, or towards morning, this fitful fever abates by profuse perspiration, urgent liquid diarrhœa, and probably diuresis. The sufferer welcomes daylight, but only with feelings of greater prostration. Such is 'hectic' fever. It recurs at variable periods, frequently as a quotidian, sometimes as a tertian or quartan. Hence the approach of hectic is often very insidious, more so since the symptoms themselves are equivocal, resembling those of other intermittent fevers. It is, therefore, an important matter to distinguish them. To this end, Dr. Thomson observes that the return of a hectic paroxysm is not so regular as that of a true intermittent. It seldom takes place for more than three or four paroxysms at a period perfectly regular. The paroxysms then begin to recur at irregular intervals, or perhaps disappear entirely

for ten or twelve days. Now, this is unlike the course of ague. Moreover, the intermissions of hectic are not perfect, it is rather a remittent fever; during its remissions, also, the pulse still beats above *par*, and is very easily excited. Thus, hectic may recur two or three times every day; and a very slight degree of emaciation, a pulse a little quicker than ordinary, with a slight increase of heat, particularly after meals, are the first symptoms of hectic. *Rigors*, upon which so much stress has been laid, is not necessarily the earliest and most constant constitutional indication of suppuration and of approaching hectic. This symptom may be present without suppuration, as the harbinger of an ague fit, or from the mere introduction of a bougie into the urethra, or as arising during inflammation itself from exposure to cold. Then, again, hectic may come on stealthily without previous rigors.

It is necessary to bear in mind the characters of hectic as contrasted with those of inflammatory fever, in order to clearly identify the kind of constitutional disorder we would prevent, if possible, by a sufficiently early and exact diagnosis of suppuration; but this implies the *causative* relation of it to hectic.

Does, then, suppuration induce hectic? It is alleged that pus is absorbed, contaminates the blood, and thence the symptoms of hectic fever. This theory is irreconcilable with known facts. Pus often exists in the body without hectic supervening; as, for example, in chronic diseases of the joints, and in psoas abscess. Probably, however, in such cases absorption is not very active. In other cases, pus is readily absorbed, yet without inducing hectic. A large bubo will sometimes subside in a few days without any symptoms of hectic, and every pus-secreting ulcer is liable to absorb its own secretion, and yet hectic is an unusual concomitant. Purulent absorption, therefore, is not a cause of hectic fever. On the other hand, I am not prepared to assert that hectic ever occurs without suppuration. Assuredly the quantity of pus formed is no *measure* of the degree of hectic which shall ensue; for, after amputation, say, of a compound fracture, a much larger quantity of pus may form eventually, before convalescence, than had been

secreted during the period prior to amputation, and yet the hectic shall subside.

Recovery follows *the removal of the disorganized part.* In some way, therefore, a causative relation exists between the disorganization of a part, and the supervention of hectic. It cannot be that the progressive destruction of nerves by suppuration, and the injury thereby continuously inflicted on the nervous system, gives rise to hectic, for the symptoms are not those of nervous irritation, and moreover, they are *intermittent,* or at least remittent. This certainly looks as if some noxious matter was gradually absorbed and accumulated in the blood, until thrown off by a hectic paroxysm, again to reaccumulate. The matter in question is not pus, or if absorbed it does not infect: by exclusion we are led to infer that the morbid matter is probably the *débris* of the disorganized textures. Clinical observations and examinations of the blood in hectic are wanting to confirm this view; meanwhile, it is a significant fact, that the urine deposits *lithates* during the *decline* of *each* hectic paroxysm.

This interpretation of hectic indicates the kind and degree of relationship existing between the progress of suppuration and itself.

If hectic arises from absorption of disorganized textures; then, if this type of fever may possibly arise independently of suppuration, it *must* follow suppuration, with progressive accumulation of pus: not that pus, in such case, itself infects, but because its accumulation necessarily *implies* the concurrent and corresponding absorption of the encompassing textures, and by thus poisoning the blood, induces hectic fever. Suppuration and hectic are, therefore, related indirectly, as cause and effect; and, moreover, the quantity of pus accumulating actually becomes a fair measure of the degree of hectic.

Thus the value of the earliest and most exact diagnosis of suppuration is obvious; and the general remarks I made respecting the *kind* of 'swelling' which characterizes diffused suppuration and abscess respectively, apply with full force to the 'rational' prevention of hectic.

Furthermore, certain acknowledged principles of preventive treatment admit of explanation by the theory I have advanced. Absorption of the textures is checked when the progressive accumulation of pus is prevented: hence the importance of making an early, free, and dependent opening; for this equally allows any *débris* of the tissues to escape. Again, absorption of disorganized tissues is altogether prevented by removing them: hence the necessity for amputation when their destruction is extensive and beyond hope of recovery to a healthy condition. Amputation under these circumstances is often followed by surprisingly beneficial results. As Hunter originally remarked, a hectic pulse at one hundred and twenty has been known to sink to ninety in a few hours after the removal of the hectic cause. Persons have been known to sleep soundly the first night afterwards, who had not slept tolerably for several preceding weeks. Cold sweats have stopped immediately, as well as those called colliquative. Purging has immediately ceased, and the urine begun to drop its sediment.

All these facts and considerations harmonize with the theory, that hectic arises from absorption of disorganized tissues. Pus itself is sometimes apparently absorbed as pus; but this produces very different symptoms and results: I mean those of 'pyæmia' followed by *secondary* abscesses.

I shall presently notice more particularly the circumstances under which pyæmia arises. The *symptoms* are still those of a blood-poison, and of typhoid character; but the exhaustion induced is more overwhelming,—*more typhoid;* and the poison is either not thrown off at all, or insufficiently to allow of marked intermissions. The fever, so to speak, is *more continued;* in this respect also contrasting with hectic.

Suddenly a wound, for example, ceases to secrete pus; then follow violent concussive *rigors,* lasting probably for some minutes, with prostration; the face looks haggard, alarmed, and vacant, as if the individual were conscious of some vital injury; and so it is, the whole nervous system responds by a prolonged shudder. This is not necessarily accompanied with a sensation of cold. Rapid

sighing completes the picture of prostration. At length the heart arouses, and the pulse rises, perhaps to 150 in a minute, beating feebly and irregularly; soon a scorching heat of skin is experienced; flooding perspiration succeeds. The paroxysm is over. In some cases a succession of similar paroxysms occur at periods varying from twelve to thirty-six hours;* in other cases they recur about the same hour for three or four days, in others at irregular intervals.† Probably, in most cases, rigors occur once for all, then *absolute* prostration, from which no perceptible reaction ensues. Whether prostration be continued, or relieved by fits of reaction, other significant symptoms supervene. The skin becomes overcast with a dirty yellow tinge, and the abdomen, tympanitic. All the secretions show signs of a blood-poison being ineffectually eliminated. Rank perspiration, often profuse, ammoniacal urine, and a putrid diarrhœa, alike compete incessantly to evacuate it; but these drains only help to complete the exhaustion. The breath has been noticed by Bérard and Mr. Gamgee to have a very peculiar odour, compared by the latter to that of sweetish liver. In one well-marked case of pyæmia this odour was absent; and it was present without pyæmia in two other cases—one of prostatic abscess, the other, extravasation of urine after lithotrity. The diagnostic value of this symptom is therefore equivocal. Eventually the tongue and perhaps the lips are dry and brown, or black. Sometimes a leaden hue overspreads the face (Gamgee). No time is allowed for progressive emaciation, as with hectic, prostration is so overwhelming:

> "the life of all his blood
> Is touched corruptibly; and the pure brain
> (Which some suppose the soul's frail dwelling-house)
> Doth, by the idle comments that it makes,
> Foretell the ending of mortality."

Pyæmia is not always fatal. Sédillot‡ and Vidal§ have re-

---

* On Pyæmia, J. S. Gamgee, 1853.

† Inflammation of the Veins, &c., H. Lee, 1850, Case xxxvii.

‡ De l'Infection Purulente, ou Pyémie, 1849.

§ Traité de Pathologie.

corded cases of recovery. Nélaton* dissents, and affirms that death is inevitable.

Post-mortem examination reveals numerous abscesses in one or more organs and textures of the body; and they are not inaptly termed *secondary,* because subsequent to an abscess, or at least to suppuration, in some other part. Secondary abscesses most frequently occur in the lungs and liver; and their general characters are these:—In several portions of the organ affected, black blood has accumulated; such portions are indurated, but brittle, and easily break down under slight pressure with the finger;—the texture is congested and disintegrated. Some of these disintegrated portions present a yellow spot of pus in the centre of the black mass; in other parts pus has altogether supplanted the blood and disintegrated textures; and these *pus-deposits* are tolerably circumscribed; in fact, abscesses are formed, each surrounded with a dark margin of congested texture. Mr. Lee† states that the central spot of each affected portion at first consists of lymph, which gradually extends towards the circumference. If the disease continue, these spots suppurate, the affected portions having previously softened and disintegrated in the same order in which they were primarily solidified. Secondary abscesses are usually very numerous. They are not confined to any one organ; for although most commonly found in the lungs and liver, other organs and tissues are not exempt. They occur—approaching the following order of frequency—in the spleen, brain, kidneys, heart, skin, mucous membranes, within serous and synovial cavities, in the muscles and cellular tissue, and in the eye; in the prostate (Gamgee). It will be observed, that the chosen seat of an ordinary or primary abscess—the cellular texture—is not the usual habitation of secondary abscesses; and I may add, as another point of difference, that usually there are no local symptoms—no pain, or functional derangement, although some parts—as the lung,

---

* Elémens de Pathologie Chirurgicale, p. 167.

† Op. cit., p. 52.

pleura, peritoneum, and intestinal canal—will not tolerate secondary abscesses without some local symptoms of disaffection.

The blood itself has undergone serious changes of chemical composition, probably, of physical properties and vital endowments, certainly. It has less plastic power, coagulates imperfectly and less readily. Being more fluid, minute hemorrhages take place in various textures, and the gastro-intestinal mucous membrane is stained with a reddish tint; but the pathology of pyæmic blood is almost a *terra incognita.*

Such are the leading facts relative to pyæmia as at present known, and its local consequences. To what theory or explanation do they point—what cause or causes in operation, anticipating which, by due precautionary measures, we may reasonably trust to prevent secondary abscesses?

I have just alluded to certain changes which pyæmic blood is found to have undergone after death. Now, the circumstances under which pyæmia arises lead to the *inference* that during life *pus* itself, as *bonâ fide* pus, has entered the circulation, either by suppurative inflammation of the veins—occasionally of the lymphatics, rarely of the arteries; or by absorption through the veins, aided by the lymphatics, laid open and exposed by mechanical injury, as by wounds, or rapid sloughing of the suppurating part: and that the pus thus produced within vessels, or introduced from without, possibly circulates with the blood, inducing secondary deposits. This is a long sentence, and each clause must be submitted to the test of clinical observation.

Without doubt, *suppurative phlebitis* induces pyæmia, and thence secondary abscesses. Hunter* originally observed, that "in all cases where inflammation of veins runs high, or extends itself considerably, *it is to be expected* that the whole system will be affected. For the most part, the same kind of affection takes place which arises from other inflammations, with this exception, that

---

* Trans. of a Soc. for the Improvement of Med. and Chir. Knowledge, 1793, vol. i., p. 18.

where no adhesions of the sides of the vein are formed, or where such adhesions are incomplete, pus, passing into the circulation, may add to the general disorder, and even render it fatal." Hunter, therefore, clearly recognised this mode of origin, and accurately described the circumstances under which suppurative phlebitis is inevitably followed by pyæmia. But Hunter did more; he suggested the probable mode in which the local disease,—suppurative phlebitis, operates so as to induce the general disorder since denominated 'pyæmia'—viz., by "pus passing into the circulation"—acknowledging, however, that he had not been able to *determine* the particular event which occasions *death*; "it may either be that the inflammation extends itself to the heart, or that the matter secreted from the inside of the vein passes along that tube in considerable quantity to the heart, and mixes with the blood."

All subsequent observers have confirmed Hunter's observation as to the fact that suppurative phlebitis is frequently *followed* by pyæmia; some, moreover, have adopted his theory, that pus *entering* the circulation is the immediate cause, and have extended this view to interpret the *formation* of secondary abscesses; while no one, in my opinion, has ever disproved it.

In 1815 Hodgson* supported the Hunterian theory of pyæmia; and although Travers† alleged that suppurative phlebitis was comparatively harmless, and the adhesive fatal; yet the influence of his opinion was soon reversed, the potency of pus-forming phlebitis re-acknowledged, and Hunter's theory re-adopted. Carmichael,‡ ever foremost in the vanguard of truth, contributed his observations, and his concurrence with the view of our immortal *confrère*, if not countryman. Abernethy§ followed, rather as a disciple than as an original observer, in this department of Pathology. Then the

---

* Diseases of Arteries and Veins, 1815, pp. 511 and 518.

† Wounds and Ligatures of Veins. Surg. Essays, Cooper and Travers, 3rd Edit., 1818, vol. i., p. 286.

‡ Obs. on Varix and Venous Inflammation. Trans. of King's and Queen's Coll. of Phys., Ireland, 1818, vol. ii., pp. 355 and 368.

§ Essay on the Occasional Ill-consequences of Venesection. Surg. Works, 1823, vol. ii., p. 150.

French school instituted investigations to discover the immediate cause of pyæmia from suppurative phlebitis. Their results were, with scarcely an exception, in favour of Hunter's suggestion. Bouillaud* contributed his clinical researches, ascribing the constitutional consequence of phlebitis to pus in the blood. Cruveilhier† likewise came to the conclusion, that pus is transmitted from the veins in which it is formed; and that, being *arrested* in the capillaries of various organs, it induces secondary suppurative phlebitis of the smaller veins. This view of the process by which secondary abscesses are formed will be considered in connexion with the issue of pus in the blood.

Respecting the *transmission* of pus as the *immediate* cause of pyæmia, Cruveilhier's observations of the stages of suppurative phlebitis tend to establish two facts. Firstly, when pus produced within a vein is excluded from the general circulation, as by a barrier of coagulated blood, or by adhesion and obliteration of the vein, no symptoms of pyæmia ensue; secondly, when the obstruction is removed, the symptoms of pyæmia immediately commence. The unavoidable inference is, that pus is carried from within the suppurating vein into the general circulation.

It would appear that pus is first formed, not between the vein and the clot, but in the very *centre* of the coagulum, which soon blocks up an inflamed vein. The symptoms of pyæmia do not arise *then;* but the proportion of coagulum diminishes, while that of the pus increases, and this may take place here and there in the course of the vein, as the inflammation is more or less advanced, thus presenting adhesive, alternating with suppurative, phlebitis. Usually, adhesive phlebitis limits and *circumscribes* the suppuration; and *then,* also, the general mass of blood remains uncontaminated.‡ Pyæmia does not arise under

---

* Recherches Cliniques pour servir à l'Histoire de la Phlébite. Revue Méd., Juin 1825, p. 424.

† Anatomie Pathologique, 1829–35, tome premier, liv. xi., Phlébite et Abcès Viscéraux; also Dict. de Méd. et de Chir. Pratiques, art. Phlébite.

‡ Anat. Pathol., liv. xi., pl. 1.

these circumstances. A woman, soon after delivery, had inflammation of one of the superficial veins of the breast. It presented a large, hard, painful cord, running in a transverse direction immediately below the nipple, and fluctuation could be felt at the deep end of this cord. A puncture being made, and pressure from without inwards, the vein was emptied, and then appeared as a furrow. Pyæmia did not attend this condition.

As phlebitis advances, the distended vein becomes knotty at the various portions where pus has accumulated; eventually the vein bursts, pus is deposited in the surrounding cellular tissue, and abscesses are formed. Pending this issue, when the dyke made by the *clots* is *broken,* and removed by absorption, typhoid symptoms immediately commence, announced by violent rigors, soon succeeded by death. Frequently a patient, who overnight exhibited no constitutional symptoms of pyæmia, is found next morning in a dying state, and perhaps almost the moment when pus entered the circulation can be noted.

Duly considering these facts, it is scarcely necessary to bring forward the corroborative testimony of other observers. The student may advantageously consult memoirs by Dance* and Blandin,† both of whom attribute pyæmia to the transmission of pus from suppurating veins. Mr. Arnott‡ was led by his observations of pyæmia, in seventeen cases of fatal phlebitis, to conclude that the entrance of pus into the circulation is its principal cause, "a similar influence being, perhaps, also possessed by any inflammatory secretion from the vein." Andral§ promulgated this doctrine, and Carswell‖ likewise; alleging, moreover, that with all the facts before us—namely, the existence of suppurative

---

* Archiv. de Méd., 1828–29. De la Phlébite Uterine, et de la Phlébite en Général.

† Journ. Hebdom. de Méd., 1829, tome ii. Sur quelques Accidens, &c., à la suite des Amputations.

‡ Path. Inq. into the Secondary Effects of Inflammation of the Veins, 1829, p. 61.

§ Pathological Anatomy. Trans. 1831, vol. ii., pp. 419 and 422.

‖ Elementary Forms of Disease, 1838, art. Pus and Purulent Deposits.

inflammation, the presence of a greater or less quantity of pus in the veins, evidence that the pus so situated is the product of inflammation of these veins, and of this morbid product being carried into the blood by the collateral venous circulation —it appears that a satisfactory explanation may be given of the formation of those anomalous collections of pus which take place in remote parts of the body. Dupuytren, Bérard, Vidal* (de Cassis), and Sédillot† concur in the doctrine of pus-transmission; the latter observer maintaining that pus can actually be seen in the blood when examined under the microscope. Remembering the great difficulty of clearly distinguishing pus-cells from the pale corpuscles of blood, I cannot accord much weight to this argument.

The evidence worthy of credit is purely circumstantial; nevertheless the inference is conclusive, short of ocular demonstration, and perhaps incapable of direct proof. Certain observers, therefore, have been content to record the important fact that suppurative phlebitis very frequently induces the constitutional disorder known as 'pyæmia,' without specifying its immediate cause. Ribes‡ does not assign the transmission of pus nor any other explanation of this question; neither does Breschet§ nor Guthrie.‖

Others have denied the possibility of pus entering the circulation under the ordinary circumstances of suppurative phlebitis. Tessier¶ alleges that at *all* stages of venous inflammation the pus is *enclosed* in the cavity of the vein by clots or false membranes, and that at no period of the anatomico-pathological existence of phlebitis is its entrance into the blood possible.

---

* Traité de Pathologie Externe, et de Médecine Opératoire, 1846, tome deuxième, pp. 82–87.

† De l'Infection Purulente, ou Pyémie, 1849.

‡ Exposé Sommaire de quelques Recher. Anatom., Physiol., et Path. Mém. de la Soc. Méd. d'Emulation, 1817, tome viii., p. 624.

§ De l'Inflammation des Veines, ou de la Phlébite. Journ. Complimentaire du Dict. des Sciences Méd., 1819, tome ii., p. 325, tome iii., p. 317.

‖ Treatise on Gunshot Wounds, edit. 3, 1827, p. 299.

¶ Archives Générales de Médecine, 1839, iii. et Nouvelle Ser., t. iv. p. 88 *et seq.*

Mr. Lee appeals to the results of experiments on blood out of the body. In the preface to his Jacksonian Prize Essay,* he urges that "the simple experiment of mixing some pus with healthy, recently drawn blood, will at once show that such a combination cannot circulate in the living body. It will be found that the blood *coagulates* round the globules of pus, and forms a solid mass which will adhere to the first surface with which it comes in contact; and it will be evident that it is not till the coagulum thus formed is broken up or dissolved that its elements can circulate with the blood." From this and similar experiments, Mr. Lee is led to believe that "the contradictory statements which have been made by those who have injected pus into the veins may thus be reconciled by taking into account the power exercised by the blood in the experiments which have been made. There can be little doubt that, while in some instances a portion of the pus has been *forced* into the general circulation, in the great majority of cases it has been detained in the vein into which it was first introduced, and has never become part of the circulating fluid." "We accordingly find some experimenters recording the secondary diseases which they observed, while in other hands these appearances were not produced."

Mr. Lee concludes "that the introduction of pus into the system through an injured or inflamed vein can rarely be the first step towards purulent infection of the system. Some change must previously have passed in the blood, by which its coagulating power is impaired, or some unusual mechanical means must have been employed before the pus can find its way in the course of the circulation."†

Now, I quite agree with this author that the results of experiments in this department of Pathology are contradictory; but I cannot accept his explanation—namely, "by taking into account

---

* On the Origin of Inflammation of the Veins, and on the Causes, Consequences, and Treatment of Purulent Deposits, 1850.

† Op. cit., p. 45.

the *power* exercised by the blood" in the experiments referred to. Mr. Lee's inference from his "simple experiment" *assumes* that that which is true of blood mixed with pus *out* of the body, is also true of blood *circulating* with pus *in* the *living* body. The inference drawn breaks down, because the circumstances of the two cases are so essentially different. More than this—*comparative* Pathology cannot supply the place of *direct* observation in human pathology; comparative and approximate circumstances may appear to be a short cut to the results of otherwise much laborious clinical investigation, and may appear to save the necessity of watching our opportunity for studying the nature of diseases, themselves of rare occurrence; but each species of living being is too *distinct* to allow of its pathology being *predicated*, any more than its healthy structure and functions can be predicated by anatomical and physiological knowledge of other species apparently allied. Deductions concerning one species, from observation of another, are but the links of a broken chain; and by this fallacious mode of reasoning it would be easy to apply the pathology of a monad to the therapeutics of man.

If, then, comparative pathology itself misleads in the practice of medicine and surgery, how much more fallacious must be the guidance of experiments made altogether apart from the influence of the *living* organism. This, as Hunter said, is "putting living and dead animal matter upon the same footing, which is a contradiction in itself." The inference which Mr. Lee has drawn from his experiments is therefore without appropriate foundation.

The fair inference from all the facts—pathological and clinical—which I have adduced, is, that pus *enters* the general circulation during the course of suppurative phlebitis. By analogy, pyæmia may be expected to arise also from suppurative inflammation of the *lymphatics*. Similar symptoms to those of pyæmia arise occasionally after slight pricks and wounds received in dissection, and similar consequences ensue. How far this constitutional disorder, and these consequences, are due to the animal matter

inoculated, is uncertain. The very worst constitutional symptoms may follow the slightest prick where the quantity of animal matter introduced must have been minute; and that equally bad symptoms may follow a bleeding wound where the animal matter has been washed away: yet the *most* fatal cases are generally those of wounds received in the post-mortem examination of uterine phlebitis with suppuration, and pus-forming peritonitis; and lastly, the constitutional symptoms are in the *ratio* of the local inflammation and suppuration.

For these reasons I am inclined to regard the constitutional disorder arising from dissection-wounds, when accompanied with inflammation of the lymphatics, as pyæmia, from pus having thus entered the general circulation. And as I have said, the symptoms and the consequences are similar. Severe *rigors* announce the transmission of pus; this primary symptom occurring, not at the time of the prick or cut, but after the lymphatics have become inflamed. Prostration, extreme, with a countenance expressing dread apprehension, show that the poison continues in operation. Partial reaction may succeed, but eventually the pulse becomes very weak and very rapid. A dusky yellow tinge sometimes overshadows the skin, and the tongue is coated with a brown fur. Fetid perspiration and alvine evacuations may apparently eliminate the poison and restore health, or oppressive wandering delirium betoken approaching death. Such are the chief general symptoms. Local and significant ones have also been noticed, and accurately described, by Macartney,* Colles, and others. As the disease progresses, inflammatory tumours arise successively in parts of the body remote from the original wound, and of a character which augments the constitutional sympathy, for they are not bounded by the effusion of lymph. According to Macartney, pus is rarely formed, and when these tumours have been cut into on the supposition of their being abscesses, they have been usually found to contain only a

* On Inflammation, 1838, p. 105.

bloody serum. Mr. S. Cooper states* that, in a large proportion of the cases which he attended, purulent matter was formed and discharged. A common situation for such abscess is under the great pectoral muscle.

Local signs of inflammation are frequently present. Colles† dwells on the significance of a peach-blossom redness, unlike the hue of erysipelas, and acute pain is sometimes felt in the part. Thus these secondary purulent collections contrast somewhat with those which proceed from suppurative phlebitis.

Arteritis rarely advances to suppuration, and more rarely are there any constitutional symptoms of pyæmia. But now and then utter prostration, with an incredibly rapid pulse, suggest this inference, and the oppressive delirium of pyæmia speedily closes the scene. Post-mortem examination is wanting to complete our knowledge of these cases.

Suppurative phlebitis, suppurative inflammation of the lymphatics, and possibly, suppurative arteritis, are not the only sources of pyæmia, the immediate cause being the *direct* transmission of pus from the vessels in which it is formed into the general circulation. Precisely the same constitutional disorder frequently follows the *absorption* of pus from suppurating surfaces, as wounds and sores. Velpeau, Maréchal, Rochoux, and others have attributed pyæmia solely to absorption, and in such cases.

What are the particular circumstances which allow the absorption of pus from a suppurating surface, so as to induce pyæmia? It is well known that pus may be absorbed from an abscess without pyæmia ensuing; and long since Cruveilhier‡ pointed out the important distinction between the absorption of pus when *prepared* for absorption, and pus in the condition of pus, at once entering the circulation. In the former case, no constitutional disturbance follows; in the latter, pyæmia.

---

* First Lines of the Theory and Practice of Surgery, 7th Edit., 1840, p. 188.

† Dublin Hosp. Reports, vols. iii. and iv.

‡ Anat. Path. Inflam. des Sinus de la Dure Mère, livraison vii., and Phlébite Utérine, liv. iv.

To allow of the absorption of pus in an *unaltered* condition, the *absorbing vessels* must have been laid *open;* the veins, lymphatics, or both, must have undergone 'solution of continuity;' and this, either by a wound without subsequent repair, or by rapid sloughing, without time for closure of the vessels by the adhesive process. They therefore remain open during suppuration, and their mouths—so to speak—being in immediate contact with pus, allow of its admission, and transmission.

This mode of purulent infection accords with Mr. Liston's observation,* that, "when from any cause the extremity of a large vein in a wound is not closed—when it is not plugged up by plastic matter, pus seems to enter it readily, and by mixing with the circulating fluid, causes dreadful mischief. Great constitutional disturbance accompanies the purulent deposits which follow in the solid viscera and in the joints." In other cases these results are preceded by rapid sloughing. A wound suddenly loses all power of healing by adhesion, and any adhesions that may have formed give way, the granulations become pale and flabby, and true pus is no longer discharged; the surface acquires a mottled brown colour, and by this time symptoms of pyæmia have commenced. Burns, attended with extensive suppuration, are not unfrequently followed by pyæmia; and certain specific diseases, as glanders, in like manner threaten purulent affection by absorption.

Occasionally, suppurative phlebitis advances so far as to allow of absorption through a vein having burst, and opened into an abscess. In one case, the femoral, popliteal, posterior tibial, and peroneal veins, communicated with abscesses (Cruveilhier). In another case, the internal jugular vein opened into a neighbouring abscess (Travers).

More frequently, inflammation of the lymphatics is accompanied with extensively diffused suppuration, and induces pyæmia, partly by the absorption of pus.

---

* Elements of Surgery, 1840, p. 154.

Injuries and surgical operations attended with much suppuration, and which are well known to threaten pyæmia, do so *partly*, perhaps, by absorption of pus, and *partly*, by suppurative inflammation of the disorganized veins. Compound fractures may thus occasion purulent infection; and injuries of the head probably operate chiefly by suppurative inflammation of the veins of the diploe. Dance first suggested this explanation; Cruveilhier gave the anatomical proof by dissection. Indeed, the latter observer affirms that phlebitis of bones is one of the most frequent causes of visceral abscesses, from wounds and surgical operations implicating the bones. The mere removal of a piece of necrosed bone is sometimes followed by pyæmia; but whenever destruction of the osseous texture, accompanied with suppuration, is followed by pyæmia, absorption of pus probably has been partly the cause, its absorption being determined by the anatomical condition of the veins, which are kept open by the bony channels through which they pass. Amputations of the limbs, of the breast, lithotomy, and the operation for fistula in ano, likewise threaten pyæmia, when suppuration is abundant. Absorption and suppurative phlebitis may co-operate in these cases to infect the blood with pus.

Lastly, sometimes suppurative phlebitis assuredly co-operates with absorption, as in pyæmia following venesection. The vein is punctured, and the aperture absorbs pus, subsequently diffused around the vein; while the vein itself, having suppurated, transmits pus into the general circulation. Probably suppurative inflammation of the lymphatics contributes to pyæmia, in some cases where and when absorption of pus may have taken place. A faint blush in the course of the lymphatics frequently supervenes on the sudden cessation of a purulent discharge; forthwith pyæmic symptoms commence; eventually the lymphatic vessels appear as red lines ramifying from the sore, becoming harder and more painful—more inflamed, in fact, as pyæmia advances.

Absorption and transmission of pus therefore go hand in hand towards purulent infection; and I have dwelt somewhat at length on this *rationale* of pyæmia, because it obviously suggests the

principle of *preventive* treatment. To prevent or arrest *absorption* and *transmission*, whenever purulent fluid is likely to enter the circulation, should ever be the *twofold* object held in view. To this end an exact knowledge of the earliest local appearances which threaten pyæmia is primarily imperative; while the process of either purulent absorption—from a suppurating wound or ulcer, or of the transmission of pus,—from the interior of a suppurating vein or lymphatic, and probably occasionally from an artery,—or both these modes of infection combined, suggests and explains the *principle* of prevention—namely, to cherish adhesion, and to arrest suppuration and sloughing. Does phlebitis threaten pyæmia; let the means, local and constitutional, of arresting inflammation be employed when first a red line appears—painful and hard; or when, without redness being apparent, a vein is somewhat tender, and hard under the finger. Then it is that blood begins to coagulate within the vein, and adhesion may be effected before the supervention of suppuration. Does inflammation of the lymphatics threaten purulent infection; let similar measures be employed, when first a faint blush, with perhaps here and there in relief a streak of darker colour, appears in any part; and should an artery become tender, the same preventive measures are suggested. Is a wound or ulcer the source of danger; let the exposed vessels be at once sealed up with plastic lymph, by the free application of a strong stimulant, as nitric acid, when first the aspect of such wound or sore excites suspicion.

Such are the leading preventive measures against pyæmia. Sufficient has been said to show how, in respect of purulent infection, pathology guides the prevention of fatal 'constitutional' disorder, by discovering and removing at the earliest period, the local cause in operation. It must, however, be confessed that the application of this principle to pyæmia is limited. Not unfrequently, phlebitis, or diffused suppuration, is so far removed from the surface of the body, as to preclude the opportunity of making a sufficiently early and exact diagnosis; and moreover, the approach of pyæmia may be so insidious as to mislead the most vigilant observer.

I proceed to consider what is called, *par excellence*, the 'termination' of inflammation—*mortification* and its ally, *ulceration*;—taking the latter first, as being, in many particulars, introductory.

Starting with the phenomena of ordinary ulceration,—I shall endeavour to determine its pathological nature; then the transition of this process to mortification, with its attendant 'fever;' and then the causative relation of the local disorganization to this constitutional disorder. Lastly, combining all the circumstances under which the former originates,—considering its general etiology, in conjunction with the earliest and most essential phenomena or signs thereof, we may reasonably hope to anticipate the latter, and thus to fulfil the principle of Prevention.

Observe a case of common ulceration. The shin having come into collision with some blunt obstacle, receives a tolerably severe bruise. What probably happens? The bruised portion of integument becomes inflamed; in the course of a few days a small piece separates and comes away, leaving a chasm, from which more or less discharge of some kind issues; in fact, the skin, with perhaps the subjacent cellular tissue, has *slowly undergone* 'solution of continuity,' and an *ulcer* is formed. Its formation and extension are termed *ulceration;* but the pathological nature of this process has long been an open question. All the views or explanations advanced may be reduced to two heads.

Firstly. Some pathologists maintain that ulceration signifies molecular disintegration, liquefaction, and separation of the soft parts, thereby leaving a chasm.

Secondly. Others, referring this loss of substance to absorption, regarded ulceration as an exhibition of the process of absorption; the lymphatics and veins being jointly engaged, more especially the former.

Calm consideration of the chief facts in favour of each of these rival explanations will, I think, lead to the conclusion that *possibly both* modes of removing disorganized textures co-operate in ulceration.

Hunter originated the doctrine of "ulcerative absorption"—a doctrine which eventually took firmer root than perhaps any other of Hunter's views. "When," says he, "it becomes necessary that some whole living part should be removed, it is evident that nature, in order to effect this, must not only confer a new activity on the absorbents, but must throw the part to be absorbed into such a state as to yield to this operation;" and among the circumstances which lead to absorption, Hunter enumerates "*death* of the part."*

Now, the possibility of dead tissue being removed by ulcerative absorption is demonstrated by the appearances which necrosed bone presents when a portion has been exposed, and the remaining portion for a time surrounded by living textures. For example, the shaft of the femur having become necrosed, the *sequestrum* loosens and eventually separates, or is easily taken away with the forceps. If, during the process of separation, part of the sequestrum has been exposed, that portion is seen to be smooth and polished, while the remaining portion which has been enclosed in new bone is seen to be *worm-eaten*, obviously, under these circumstances, from absorption. So also dead bone, artificially inserted into living bone, is in time partially absorbed.

New blood-vessels are developed around a sequestrum, apparently to meet the increased demands on absorption. Mr. Aston Key called attention to this fact when first he advocated the doctrine of ulceration by absorption through the veins, rather than by the lymphatics, as Hunter had alleged. "When," says Mr. Key,† "the sequestrum is removed from its case of new bone, and the interior of the latter is exposed to view, a number of flocculent bodies are seen attached to a membrane that supplies the newly-formed bone. When injected, these are shown to be highly vascular, and are seen to fill the indentations in the dead bone. If the latter be carefully taken out of its case of new bone, these

* Op. cit., p. 442.

† Med.-Chir. Trans, 1833, vol. xviii., p. 215.

vascular elongations will be found to have a slight *attachment* to those parts of the dead bone in which they are imbedded."

The absorbent function of this new vascular apparatus is in accordance with other facts of similar import. Thus, Sir A. Cooper* possessed an injected preparation of an ulcer of the leg, in which the veins were developed in a remarkable manner. They were numerous and large, and *surrounded the margin of the ulcer.* In another preparation of ulcer of the leg, the lymphatics were injected, but they appeared to be neither increased in size nor in number.

These observations prove the possibility, and even the probability, of ulceration by *absorption;* yet this view of ulceration is overruled by two series of facts.

Before stating them, I may mention a kind of compromise of the question at issue, and which I allude to chiefly on account of the general accuracy of observation which its distinguished author, Professor Goodsir, has evinced. Goodsir maintains, or did maintain,† that ulceration is a process independent of the vessels, both veins and lymphatics, they being "passive agents, mere ducts for conveying away the products of action."

"A rapidly extending ulcerated surface appears as if the textures were scooped out by a sharp instrument. The textures are separated from the external medium by a thin film. This film is cellular in its constitution, and so far it is analogous to the epidermis or epithelium."

It is alleged that this cellular layer possesses the peculiar (vital) power of disintegrating the subjacent texture, and that by virtue thereof, so far from ulceration denoting a diminution of the formative power of the part, progressive ulceration represents its undue activity. "The apparent diminution is a consequence of the extremely limited duration of existence of the cells of the absorbent layer, which die as rapidly as they are

---

* Med.-Chir. Trans., vol. xviii. p. 212.

† Anatomical and Pathological Observations by J. Goodsir and H. D. S. Goodsir, 1845, p. 14 *et seq.*

formed, disappearing after dissolution partly as a discharge from the surface, but principally through the natural channels by which the débris of parts which have already performed their allotted functions are taken up into the organism.

"In this view of ulceration there is substituted, for the hypothetical active or aggressive power of absorption ascribed to the veins and lymphatics, a power which is known to exist in the organic cell during the progress of its growth; and the ultimate removal of the matter from the scene of action is ascribed partly to the formation of discharge, partly to the yet unexplained, but at the same time undoubted, and in all probability passive, agency of the returning circulation."

I am not in a position to offer any direct evidence against ulceration by absorption in this sense; but I recur to the two series of facts to which I have alluded—the one series disproving absorption in any sense, the other demonstrating the disintegration, liquefaction, and separation of disorganized textures in the process of ulceration, and this *apparently* without the intervention of any cellular layer, as described by Goodsir.

In the first place, respecting ulceration without absorption. The veins proceeding from an ulcerated part are obviously much congested in certain cases, and therefore do not allow of that free passage of blood which venous absorption implies. A common varicose ulcer of the leg is a good illustration; and, as regards the lymphatics, ulceration may occur in a texture destitute of these vessels, or at least in which they have hitherto been searched for unsuccessfully. Ulceration of the cornea is a familiar example. Its conclusiveness is impaired by the presence of lymphatic vessels in, or immediately beneath, the conjunctiva. This anatomical fact was, I believe, first observed by Dr. Sharpey, who states* that he has distinctly seen lymphatic vessels, distended with their own lymph, on the surface of an eye which had repeatedly suffered from chronic inflammation. Again, Mr. Gaskell† has brought forward

* Elements of Anatomy, 1848, vol. i., p. 260.

† Jacksonian Prize Essay, 1837. MS. Roy. Coll. Surg. Eng.

certain facts which are inconsistent with the theory that ulceration is absorption by the lymphatics. Thus, ulceration does not appear to be most common where the absorbents are most freely distributed. They are more numerous and of greater size in the jejunum than in the ileum, yet ulceration is rarely found in the former portion of intestine. The absorbents are freely distributed on the surface of the body, yet spreading ulceration does not extend so readily on the external surface as a little below. Lastly, absorption readily takes place from the surface of serous membranes; nevertheless, their inflammation is rarely followed by ulceration.

The independence of ulceration and absorption, whether by the veins or lymphatics, is, I think, further established by another species of evidence. "If," as Key remarks,* "the formation of an ulcer be an act of absorption, the parts that are removed in the formation of a chancre are so disposed of; the absorbents, in forming a chancre, carry into the system tissues tainted by the venereal poison, and must therefore, in every instance, contaminate the whole mass of circulating fluids. A bubo, therefore, ought to be one of the *earliest* accompaniments of chancre. Whereas, during the ulcerative stage of chancre, the glands in the groin usually remain free from infection; it is when the ulcerative stage is at an end, that the gland enlarges and bubo forms. In other words, when the absorbents are most actively engaged in producing the ulcer, and in carrying the poisoned mass into the gland, the latter exhibits no sign of irritation; but when the absorbents are inactive, the gland begins to enlarge." Furthermore, as Wallace† truly alleges, if "ulceration implies absorption, contamination of the system should *always* follow a chancre," unless, indeed, we assume that the venereal poison may be received into the mass of circulating fluids, without necessarily causing constitutional disease, an admission which we are not warranted in making.

---

* Op. cit., 1835, vol. xix., p. 141.

† On the Venereal Disease, 1838, p. 51.

Duly considering the anatomical facts which I have adduced—namely, the condition of the veins in certain cases of ulceration, and the absence of the lymphatics in other cases; also, the fact that contamination of the blood is neither a constant sequent of primary venereal ulceration, nor, in its order of sequence, consistent with the hypothesis of absorption; the fair inference is, that ulceration may begin and continue without absorption.

Other facts show that ulceration is a process of *disintegration, solution,* and *discharge,* of disorganized textures. Take, again, the ordinary case of an ulcer arising from a blow on the shin. In the course of a few days, a small portion of dead tissue—a *slough* —comes away. Its discharge announces ulceration. Dead texture *can,* as we have seen, be absorbed, yet this slough is discharged. It may be urged that in such case sufficient time is not allowed for absorption; that the tissue dies too extensively and too rapidly for the process of absorption to keep pace with that of destruction. But subsequently, as ulceration continues, a similar discharge of dead tissue continues. This discharge of textures is identified when now and then they die too rapidly, to dissolve as they die, and become imperceptible. In all sloughing ulcers, whether from inflammation or fast-spreading phagedæna, the disintegrated portions of texture are visible. On the other hand, the common indolent ulcer and the varicose ulcer, also indolent, are generally free from the *débris* of the textures, which, as they have died, have been disintegrated, dissolved, and discharged. This, at least, is the fair inference; for every intermediate degree of disintegration and solution of the textures is witnessed, from the most obvious *sloughs* down to *débris* and all the varieties of *mixed discharge.*

This view of ulceration is supported by Mr. Key's observations, at the later date* referred to; by the observations

---

* Ibid. Further Remarks on the Ulcerative Process, p. 135.

of Earle,* Gaskell,† and Wallace.‡ Mr. Paget,§ also, has collated much important evidence in the same direction. Thus, in ulceration of cartilage, inflammatory exudation has no share, and the process of ejection of the disintegrated tissue may be clearly traced; but of cartilage it may be said, being extravascular, absorption cannot take place. The same process of ejection, however, is discernible in ulcerating bone (where absorption may occur), as shown by the observations of Virchow; and Bransby Cooper|| noticed, that while in pus from soft parts only traces of phosphate of lime are found, the pus from around diseased bone contains in solution nearly *two* and a *half per cent.* A similar, but less complete, observation was made by M. T. Taylor,¶ and also by E. von Bibra.**

Then, again, this view of the pathology of ordinary ulceration harmonizes with the process by which a *specific* ulcer is seen to be formed. Earle†† pointed out the sameness of the process. An incipient chancre is a vesicle which, bursting, discovers a minute slough. Its separation occasions the first loss of substance; subsequently, molecular disintegration continues the process of ulceration; for, as Wallace‡‡ observed, if a venereal ulcer be examined with accuracy, a red margin is seen, and frequently on the inner side of this margin a white line; the red margin in advance denoting the inflammation which precedes ulceration, and the white line within, the texture which was red, but is now white, having been converted into a slough *before* liquefaction and consequent separation. Minute sloughing, occasionally recurring on the surface of a venereal sore during the progress of ulceration, again suggests the true explanation of this process:—molecular disinte-

---

* On the Nature of Inflammation. Med. Gaz., 1835, vol. xvi., p. 254.
† Jacksonian Prize Essay, 1837. MS. Coll. Surg. Eng.
‡ Venereal Disease, 1838, p. 48 *et seq.*
§ Lectures on Surgical Pathology, 1845, vol. i., pp. 420, 453.
|| Med. Gazette, 1845, and Lectures on Surgery, 1851, p. 127.
¶ Diseases of the Bones. Stanley, 1849, p. 89.
** Chemische Untersuchungen verschiedener Eiterarten, 1842, p. 85.
†† Op. cit., p. 254. ‡‡ Op. cit., p. 49.

gration of the textures, being now and then exaggerated to visible sloughing of portions of them, declares the sameness of the process.

Textures undergo certain changes of structure preparatory to their ulceration; for what says *microscopic* observation? Ulceration itself is essentially molecular disintegration of the textures; but, in so far as inflammation has preceded this event, *degeneration* also has prepared the way for disintegration. A degraded condition of textural structure by degeneration ends in a disintegrated condition; and this previous degeneration is usually the *fatty* transformation. The testimony of many well-known microscopists here concurs. Mr. Paget* puts in evidence this case:—A man thrust a needle through the left ventricle of his heart. In four days he died, and the *post-mortem* examination showed that pericarditis had taken place. The muscular tissue of the heart close by the wound was inflamed; and this portion, and in a less degree all other parts of the heart, had undergone fatty degeneration, such as Mr. Paget could not have distinguished from that which occurs in the corresponding atrophous degeneration. Here, however, all parts of the heart, besides the inflamed portion, having undergone fatty degeneration, it was impossible to determine how far such degeneration might not have been prior to, and therefore independent of, inflammation; but Mr. Paget's inference is corroborated by Virchow's† observations of inflamed muscles generally. The bones also undergo fatty degeneration, with inflammation, as shown by Virchow; and he discovered a similar degeneration of the liver and kidneys, when these organs are respectively inflamed. Fatty degeneration of the cartilages, with inflammation, was noticed by Redfern,‡ and of the cornea, when inflamed, by Strube.§

---

* Surg. Path., 1853, vol. i., p. 413.

† Archiv, b. iv., h. 1.

‡ Abnormal Nutrition in Articular Cartilages, 1850; also, The Healing of Wounds in Articular Cartilages. Monthly Journ. of Med. Science, Sept. 1851.

§ Der normale Bau der Cornea und die Pathologischen Abweichungen in demselben, 1851.

Calcareous instead of fatty degeneration is the textural change occasionally witnessed. Calcareous degeneration of the articular cartilages accompanies chronic rheumatic arthritis; a similar degeneration of the laryngeal cartilages when inflamed; and the formation of imperfect dentine, with inflammation of the tooth-pulp, is another illustration.*

The proper discharge from an ulcer—or the product of ulceration, not of a granulating sore—is *ichor*, but its precise nature is not well understood. Usually, ichor is a thin sanious fluid, colourless or slightly yellow; structureless, but mixed with exudation—pus and blood corpuscles, and also with the *débris* of the ulcerating textures. Chemical examination of this discharge, embodying what is essential, and simplifying what seems differential, is still wanting. Ichor corrodes the living textures; and although its corrosive property varies considerably in degree, this discharge is thus distinguished from true pus; an important point, for pus and ichor are often nearly alike in appearance. Rokitansky† attaches so much significance to the corrosiveness of ichor, that he regards ulceration and ichorous destruction as synonymous. Ulceration is described as a wasting of the textures from the corrosive quality of the exudate. Herein (the quotation continues) ulcerous consumption of the textures differs from the loss of substance which inflamed textures undergo, within the best-conditioned exudates, through necrosis and absorption.

Without adopting this extreme view, and which does not explain the commencement of ulceration, *before* the discharge of ichor begins, daily observation of the ulcerative process shows the corrosive operation of this fluid in *maintaining* and *extending* ulceration. Summing up the pathology of ulceration, Mr. Paget properly estimates the influence of ichor by regarding its corrosive property as a contributory cause of ulceration.‡ Its begin-

---

* Surg. Path. Paget, vol. i., p. 417.

† Pathological Anatomy. By Sydenham Society, 1854, vol. i., p. 158.

‡ Op. cit., vol. i., p. 425.

ning is described as usually the detachment of a slough, or portion of dead tissue, by the removal of the layer of living tissue adjoining; the spreading of an ulcer, independent of this visible sloughing, is effected by the inflamed tissues around becoming degenerated, and detached in minute particles (molecular matter), or decomposed and dissolved in the fluid discharge or ichor; extension being probably accelerated by the influence of the discharge itself, which may inflame the healthy tissues that it rests on, and may exercise a decomposing "catalytic" action on those that are inflamed already.

Having considered the pathology of the ulcerative *process*, it is unnecessary for my purpose to describe the various kinds of ulcers. Their characteristic appearances are produced sometimes by external circumstances acting locally on the part—as friction, filth,* and various topical applications, including all sorts of 'dressings;' but more frequently these appearances arise from 'constitutional' causes, and the consideration of their operation is no part of the design of this chapter.

My purpose now leads me to trace the transition of ulceration to *mortification*.

The description already given of incipient ulceration—of an ulcer in its first stage—will have suggested the reciprocal relation of ulceration and mortification. They differ in degree, but are one in kind. The former may be exaggerated into the latter, and this may subside into that. Ulceration and mortification, therefore, are convertible by gradations of the same process. These abstract statements will be fully realized by recurring to the different results of the ulcerative process under different circumstances.

Take first the surface of an ordinary ulcer, such as I have supposed, on the shin, by way of example. Ever and anon during the progress of ulceration, some temporary cause may accelerate the inflammation; a larger portion of tissue undergoes degenera-

* Principles of Surgery. J. Bell, 1826, vol. i., pp. 126, 135.

tion and dies than can concurrently disappear as discharge: thus a portion, more or less, of dead tissue becomes visible as slough instead of having been removed imperceptibly, molecule after molecule, by disintegration and liquefaction, aided by the corrosive action of the ichorous secretion. It is as if the 'flow' of a tidal stream washed up more material than the returning 'ebb' can well recover—the line of coast shows the remaining débris. Even so, the surface of an ulcer may present a rim of slough, and from time to time another and another, as the margin of the sore recedes and enlarges. Of course the size of these sloughs will vary; sometimes approaching the result of molecular disintegration, sometimes so obviously portions of dead tissue as to leave no doubt of their nature, and of the transition of ulceration to mortification. Small fragments of bone may be thus detached and cast off with the fluid from an ulcerating surface, and these, observes Mr. Paget,* when they are not fragments of tissue detached by ulceration extending around them, are good examples of the transition that may be traced from ulceration to sloughing or gangrene of parts.

This, the true pathology of mortification, is now generally acknowledged; but its etiological importance will become apparent in considering the *prevention* of mortification. To prevent ulceration will be in many cases also to prevent this event; and the recognition of their pathological connexion gives a wider basis and more comprehensive views for the anticipation of that 'constitutional' disorder, represented by poisoning of the blood which supervenes.

The general appearances of the part affected are as follows:—*Incipient* mortification is characterized by certain physical alterations of texture.

The skin having become livid assumes a black colour, shading off to a reddish brown around the dying part. Its consistence also is altered, in some cases being soft, with considerable swelling,

---

* Op. cit., vol. i., p. 422.

pitting on pressure, and the cuticle separated in the form of vesicles containing a yellowish serum, or as phlyctenæ full of bloody serum. In fact, all the soft textures of the part are sodden and succulent—a condition due to infiltration of the cellular tissue with serum. It corresponds to the *humid* gangrene of French authors, the *hot* of German writers, and the *acute* of our own school. In other cases the part is hard, shrunken, and dry—an opposite condition, known as *dry, cold,* or *chronic* gangrene. The odour of a mortified part, in the condition of *sphacelus,* is to me that of burnt tissue; it becomes fetid by the evolution of gas, which inflating the cellular texture, forms considerable swelling; of a fluctuating character and simulating a collection of pus if deep tissues be thus distended, whereas if the superficial textures only be emphysematous, the part crepitates under gentle pressure, owing to a mixture of the gas and serum with which it is infiltrated. Chemical decomposition is now fairly established. I scarcely need add that the sensibility of the part is utterly lost, and that its temperature is considerably lowered.

What is the internal and anatomical condition of this dead part? The bones may have undergone but little change beyond appearing dry and bloodless, their periosteum being detached. The articular cartilages and tendons are dull and slightly softened, but all the *soft* tissues are variously discoloured and broken down, granular *disintegration* having taken place throughout their substance with one exception. The blood-vessels alone have escaped.

In various instances of phlegmonous erysipelas several inches of the femoral artery have been laid bare by sphacelus of the parts covering it, without that vessel yielding before death (Thomson).* And what is the condition of the blood itself in such cases, fluid and circulating, or coagulated and stagnant? After *death,* certainly, the blood is found coagulated in the arteries of a sphacelated part, and possibly far beyond that part. Thomson saw one case where mortification having commenced in the thigh,

---

* Op. cit., p. 523.

coagulation had extended through the external iliac up to its origin, from the aorta. During *life*, also, coagulation may occur in the arteries. Petit first noticed this fact respecting the not unfrequent absence of hemorrhage when mortified limbs are amputated — an observation corroborated by Quesnai and O'Halloran, and since confirmed by the experience of most surgeons. Sir A. Cooper met with a case* in which during amputation the femoral artery gave no hemorrhage when the tourniquet was loosened. On closer examination he found the inner side of this vessel completely plugged up, and sealed with coagulum, which extended at least *six* inches above the seat of gangrene.

But there is good reason to doubt whether coagulation in the arteries of a sphacelated part is a *constant* occurrence *during life*. In the cases of phlegmonous erysipelas which I have alluded to, the femoral artery was exposed by sloughing to the extent of several inches, yet it pulsated freely, so that the current of blood must have been uninterrupted. Cases of sloughing bubo, with exposure of the femoral artery and vein, exhibit a current of blood through both vessels, which nevertheless had been enclosed, and are still *imbedded* in sphacelated tissues. Assuredly, therefore, under these circumstances large blood-vessels may remain pervious and continue to transmit blood.

This fact bears directly on the question of *absorption* during mortification. Disintegrated and decomposed tissues being in contact with veins *free* to absorb, their absorption by the veins is *possible*, at least in *some* cases of spreading sphacelus.

Following in order upon this condition of local disorganization, the 'constitutional disease' at once commences. More insidious, its symptoms are not unlike those of pyæmia, although differing in degree. A wild apprehensive look, with great restlessness, are conspicuous; the features and manner at length become somewhat composed, and the face assumes a very pallid hue. In some

---

* Lectures, 1839, p. 107.

cases the skin over the whole body and the tunicæ conjunctivæ acquire a peculiar yellow colour (Brodie). Utter prostration of mind and muscular power gradually supervene, and a quivering subsultus tendinum steals over the patient. The pulse now beats very feebly, rapidly, and irregularly, feeling like a fine rough wire drawn under the finger. Sometimes the tendons contiguous to the wrist vibrate with subsultus so thrilling that this flickering pulse cannot be distinguished. The secretions are soon perverted. The skin, at first hot and dry, is then bathed with a cold, clammy sweat. The urine, fetid and scanty, may be suppressed. A brown, rough, dry tongue is accompanied with nausea and a putrid diarrhœa. As the powers of organic and of animal life fail, involuntary excito motions predominate. Spasms and convulsions shake the moribund body, while coma ends in death.

Or, in other cases, mortification ceases to spread. The reddish-brown tint of the skin around the dead part, and which has hitherto spread in advance of mortification, now becomes brighter and more circumscribed. Shortly a white raised line is seen in the living skin immediately adjoining the dead portion, and corresponding in extent to that of the subjacent sphacelated textures. Soon this line melts away into a groove by ulceration; this groove extends deeper and deeper, forming a fissure, and successively passing through tissue after tissue, at length converges, and completely detaches the whole of the dead part. I shall hereafter describe this process more minutely; it is sufficient for my present purpose to add that while the living organism is detaching itself from the dead part, *adhesive* inflammation precedes the line, or fissure, of ulceration, and corresponding to it in length and depth seals the blood-vessels, effectually excluding any further communication with the dead tissues, and preventing their absorption for the time to come. The 'typhoid fever' immediately subsides, and eventually passes away altogether. In exchange, some degree of 'inflammatory fever' accompanies the process of separation.

Such is a descriptive outline of the origin, course, and termi-

nation—fatal or favourable, as the case may be—of that constitutional disorder which proceeds from mortification. Arising from spreading sphacelus, it ceases when the dead part is detached from the living body.

This immediate dependence of the constitutional disease on the local disorganization is well known to every practical surgeon; but *how* this fever arises from the local cause in operation, and *why* it should subside when the dead part is merely separated by a natural process, although still remaining in contact with the living organism, are topics for further inquiry. Yet the pathology involved in this inquiry bears alike upon the prevention and curative treatment of an oft-fatal constitutional disease.

Now, in tracing the history of this disease I alluded to two facts of an anatomical character, which taken together show that *absorption* of *dead tissues* is an immediate cause of that fever which accompanies spreading sphacelus.

Firstly, this fever arises, and invariably, when mortification has advanced to sphacelus—*i.e.*, when dead tissues are present; and moreover, when the veins, in some cases at least, are free to absorb.

Secondly, it begins to subside, and invariably, when sphacelus has ceased to spread—*i.e.*, when the living organism is separating itself from the dead part, and when adhesive inflammation of the blood-vessels, corresponding to the line or fissure of separation, has intercepted any further communication through these vessels, with the dead tissues.

This immediate and invariable supervention of the fever, when absorption of dead tissues is assuredly possible, in some cases at least, through the veins; with its immediate and invariable subsidence and eventual cessation, when such absorption is impossible; are facts which, taken conjointly, establish the relation of cause and effect between it and the fever, and, therefore, one, if not the only, source of this constitutional disorder.

And this explanation would be complete if it could be demonstrated that the fever arose *only* under circumstances compatible

with absorption; but the state of the lymphatics has not hitherto been ascertained, and this omission impairs the self-sufficiency of the absorption theory. In those very cases of spreading sphacelus, where the free current of blood necessary for absorption is arrested by coagulation in the arteries, the *lymphatics* also *may not* be *free* to absorb; and yet the fever invariably arises. Or again, when a sphacelated part is about to be separated, and adhesive inflammation secures the blood-vessels against any further communication with the dead tissues, thus precluding absorption by their agency, the *lymphatics* may *not* be *closed;* and yet the fever invariably subsides. If, then, this fever should prove to be absent when absorption of the dead tissues is possible, and present when such absorption is impossible, some other condition unknown must be its immediate cause in such cases. Absorption could not be the *only* cause of this constitutional disorder.

Pending the further advance of pathological anatomy in respect of sphacelus on behalf of this question, I shall now endeavour to determine what light clinical observation supplies, by watching and tracing the operation of different causes of mortification.

Firstly, I select a case of remarkable significance, from the Lectures* by Sir B. Brodie. A surgeon, about sixty-three years of age, had a carbuncle on his back, attended with considerable pain. Poultices having been applied for two or three days, Sir B. Brodie made a crucial incision through the swelling. Much relief followed this operation, when there appeared another carbuncle, but on a smaller scale than the first. It was not a pimple in the skin, but the subcutaneous form of the disease, and not attendant with much pain. Incisions were postponed. It went on increasing; the overlaying skin became purple, and the whole was assuming the ordinary appearance of carbuncle. Meantime the patient continued well in health, and appeared indeed to have scarcely any ailment except the local complaint. The report pro-

* On Pathology and Surgery, 1846, p. 393.

ceeds—"But a day or two afterwards, on calling on him, and believing that it would now be right to incise the tumour, I found him in bed. On inquiring the cause, he said, in a faint voice, 'Oh! my dear friend, I am dying!' His skin was cold and clammy, and the pulse scarcely perceptible. I asked him how long he had been in that state. His answer was, 'During the night all the pain subsided, and at the same time I became ill. I believe the carbuncle itself has disappeared.' And so it had. When I examined the back I could find scarcely a vestige of it. He died in less than twenty-four hours after this change had taken place."

The carbuncle in this case having been ripe for incision, its subsidence plainly shows that dead tissues were absorbed; and the typhoid fever immediately began. Obviously, therefore, in this case, absorption was the immediate cause of the fever.

In conformity therewith, other cases as plainly show that, *ceteris paribus*, the fever is proportionate to the *quantity* of dead tissue taken into the circulation. The greater or less *rapidity* with which this process goes on is also another measure of the degree of fever. Rapid absorption of dead tissue, as in the case just quoted, whereby in the course of only one night apparently scarcely a vestige of the carbuncle remained, is equivalent to the reception, more slowly, of a much larger quantity; and the case soon terminated fatally. A large quantity of dead tissue undergoing absorption is scarcely less influential, as shown by the more typhoid character of the fever in cases of extensive sloughing.

Granting that absorption of dead tissue induces this constitutional disorder, is it *the* only cause?

What further says clinical observation? That if absorption be precluded the usual concomitant fever is absent.

The following case, recorded by Mr. Guthrie,* for another purpose, appears to me to bear this construction. After the battle of Waterloo, among the British wounded was a man who stated

---

* Gunshot Wounds, 1827, p. 129.

that he had received a blow on the back part of his leg, he believed from a cannon-shot, which brought him to the ground, and stunned him considerably. On endeavouring to move, he found himself incapable of stirring, and that the sensibility as well as the power of moving the limb were lost. It gradually became black and cold; mortification extended nearly as high as the knee, without any appearance of a line of separation, and the signs of inflammation were so slight that amputation was performed above the knee. On dissecting the limb a considerable extravasation of bloody fluid was found below the calf of the leg, and in the cavity thus formed some ineffectual attempt at suppuration had been made. The periosteum was separated from the tibia and fibula; the popliteal artery was closed in the lower part of the ham by coagulable lymph proceeding from a rupture of its internal coat. Two inches below, the posterior tibial and fibular arteries were completely torn across, and in all probability gave rise to the extravasation.

With this obstruction to the circulation, and so far to absorption, although the quantity of dead tissue—the spreading sphacelus—was extensive; it is added, that when the operation was performed the constitution was certainly affected, but only in a slight degree. The case went on favourably, but the man died subsequently from dysentery.

Up to this point, then, Clinical observation ratifies the suggestions of Pathological Anatomy; that absorption is the only cause of the typhoid fever which arises from and accompanies spreading sphacelus; but the exclusiveness of this conclusion is apparently irreconcilable with cases, the significance of which is illustrated by a remarkable one that occurred in the practice of Sir B. Brodie.* He says:—"I was called to see a gentleman who appeared to be actually on the point of death. His extremities were cold, his pulse barely perceptible. It was doubtful whether he was sensible or not. He made, on being roused, several imperfect attempts

* Op. cit., p. 298.

to speak, but could say nothing intelligible. Below the right hypochondrium there was a considerable tumour, the skin being of a dark red colour, on the verge of mortification. On examination with the fingers, I perceived a sort of emphysematous crackling, and an imperfect fluctuation. Having made a free incision, I discovered, underneath the discoloured skin, what might be called a quagmire of slough. A small quantity of putrid matter escaped; but there escaped also such a quantity of noisome and offensive gas, apparently sulphuretted hydrogen, that I could scarcely bear to remain in the room. The stench pervaded the whole house, and even could be perceived in the garden round it. Within two minutes after the performance of this operation, so trifling in appearance, so important in reality, the patient looked up, and said quite distinctly, 'What is that you have done which has made so great a difference in my feelings?' At the same time the pulse returned at the wrist; and from that moment he recovered, without any further unfavourable symptoms. After a few days, sloughs came away, probably of muscle, cellular membrane, and peritonæum, in a confused mass; and with them a gall-stone of moderate size, explaining, to a certain extent at least, the origin of the disease."

In this case "a quagmire of slough" remained for a few days after incision, yet without constitutional disturbance; whereas, the almost instantaneous relief afforded by the escape of fetid gas is scarcely to be explained by supposing that absorption of dead tissue was as suddenly arrested; rather, this *sudden* relief suggests that an overwhelming poison was removed by the discharge of gas, resulting from decomposition of dead tissues, a poison which had previously operated chiefly through the *nervous system*. But this gas is only present in advanced sphacelus, and cannot therefore be the immediate cause in operation in the first instance. The fair conclusion is, that absorption of dead matter primarily, nervous sympathy secondarily, and ultimately both, induce the typhoid fever of sphacelus.

The leading principle of prevention indicated by the foregoing

pathological inquiry is this :—to anticipate absorption, and to give free vent to any gas which may be generated, during the course of sphacelus.

Beyond this twofold principle, is another obvious one:—to save as much of the part affected as possible, by soliciting a line of demarcation between the living and dead tissues, at the earliest period possible under all the circumstances, and thus arrest the blood's contamination. But the arrest of spreading sphacelus implies a sufficiently early and exact diagnosis of the internal cause or causes of mortification which are in operation; a further application of pathology.

Pathology therefore makes three preventive suggestions in respect to sphacelus, and its spreading:—strive to prevent absorption from, and nervous sympathy with, the dead part; strive to arrest the spreading of sphacelus; above all, strive to remove or intercept, the internal causes of mortification, at the earliest period possible, and thus, the origin of sphacelus.

These causes I shall now consider, in conjunction with the most constant premonitory signs of sphacelus,—those of *gangrene*, as arising from *each* such cause.

The internal causes of mortification are analogous to those which induce inflammation, effusion, and suppuration. Mortification signifies the death of some part of the body; consequently those internal conditions which disturb and pervert nutrition by inflammation, &c., if operating with sufficient *intensity*, and for a sufficient period of *time*, end in mortification. Indeed, the very fact of pus collecting in any texture or organ, by *progressive* suppuration, necessarily implies the previous death and removal of the occupied portion by mortification and absorption. Hence, mortification is sometimes described as a 'termination' of inflammation. The same causes may have been in operation, only more intensely, or persistently, suppressing *resolution* and restoration of the part affected. But the etiology of mortification has a far wider signification. *Whatever* disturbs, or tends to disturb, the nutrition of any part of the body, endangers the vitality of that part, and

becomes a cause—*predisposing* or *immediate*—of mortification. All the conditions, therefore, on which the healthy *status* of nutrition depends, are reflected in the etiology of mortification.

Firstly. 'Blood of a certain quality,' suitable to the part to be nourished, and this blood 'flowing in a certain quantity, and with a certain speed through it,' in both these respects likewise, being adapted to its nourishment; secondly, 'an appropriate physical and structural condition of the part itself;' and thirdly, some degree of 'nervous influence;'—these are the *internal* conditions, which, when perverted, become (internal) causes of mortification. Moreover, certain *external* agents; physical—as mechanical violence and injury, heat, cold, electricity, chemical decomposing agents; and vital, as animal poisons introduced into any living texture: either of these (external causes) may *directly* and immediately kill the part affected by it; or kill, if the tissue be vascular, by *inflammation,* terminating, perhaps speedily, in mortification.

By some variation in the *degree* of its operation, any cause of mortification may subside into a cause of ulceration. I am not aware of any one cause of the former which may not thus become also a cause of the latter, and conversely.

But whether mortification or ulceration ensue, two or more causes usually *co-operate;* and it is difficult to discriminate between, and apportion the share due to each internal cause, more especially in conjunction with the influence of external conditions, such as moisture, temperature, &c.

I shall now take in order the different causes of mortification and ulceration, beginning with those which are, properly speaking, *internal.*

Some such causes are 'constitutional,' and their operation does not come within the design of this chapter. I therefore only here allude to various conditions of the blood or 'blood-diseases' whose pathological nature is at present unknown, while their local effects are well known. For example, the blood-*crases* which clinical observation has accorded respectively to 'carbuncle' and 'boil' (to 'the carbuncle of plague'); to 'erysipelas,' to 'small-pox,' to

'malignant scarlatina,' to 'glanders,' to 'ergotism,' to 'scurvy,' and to 'syphilis;' the two last more especially inducing ulceration.

The *quantity* of blood supplied to a part, and its rate of motion through that part, are alike due to and regulated by the heart's mechanism and action; the state of the vessels—arteries, veins, and capillaries, and the physical condition of the blood itself, chiefly in respect of its spissitude and adhesiveness. These conditions are, each or all, concerned in producing that mortification which ensues from defective quantity rather than quality of blood.

Passing on to the state of the 'vessels,' that of the *arteries* first merits attention.

The elastic property and contractile power of these vessels are conditions not, indeed, directly engaged in propelling the blood, yet they affect its distribution by directing its current and regulating its supply. An elastic, but weakened contractile state of the arteries—say of the femoral artery—will cause that vessel to yield inordinately to each pulse-wave, thereby inadequately transmitting the current of blood to the leg. Its nutrition failing, mortification threatens. On the other hand, an artery which has lost its elasticity has lost its capability of allowing an increased quantity of blood to the part supplied—a provision for the demands of more active nutrition as occasion, ever-varying, requires. This want of accommodation on behalf of any extra supply of blood which may be required by the part, will further endanger its vitality.

These reflections explain the *modus operandi* of certain conditions of arteries and veins as well-known causes of mortification.

Ossification, so called, of the arteries—*i.e.* their calcareous degeneration, is one such cause long since acknowledged by pathologists. Thomson* discovered very complete ossification of the arteries of the leg in connexion with mortification of the feet and toes. Mr. Hodgson† found the three principal arteries of the leg nearly

---

* Op. cit., p. 537.

† Diseases of the Arteries and Veins, 1815, p. 41.

obliterated by calcareous matter in two fatal cases of *senile gangrene;* and from post-mortem examination in very many cases, Sir B. Brodie* came to these conclusions respecting this disease :—That, in some instances, the arteries are ossified, and at the same time either contracted or obliterated; in others, they are ossified without being obliterated, even retaining their natural diameter; and that, in others, they are obliterated without being ossified. In one such instance there was no ossification of the arteries anywhere, but the femoral artery was converted into a gristly cord, so as to be quite impervious from the origin of the *profunda* to the point at which it perforates the tendon of the great head of the *triceps adductor* muscle. Similar cases to this last are described by Carswell.† While in every instance of *gangrena senilis,* which he examined after death, the arteries of the limb were obliterated to such an extent as to interrupt the circulation of the blood; in five or six cases this obstruction was due to a fibrous tissue formed either in the walls or cavities of these vessels, and which had converted them into nearly solid cords of *ligamentous* consistence. This state was traced from the toes more than half way up the leg, but always accompanied with ossification of the large branches and trunks of the thigh and other parts of the body. In other two cases the obstruction depended on extensive ossification of the principal arteries of the limb, and in several others it was occasioned by solid fibrin formed around spicula of bone projecting from the internal surface of the arteries.

To sum up all these facts; senile gangrene is induced by ossification, calcareous degeneration of the larger arteries, with fibrous thickening of the smaller ones, leading to the part affected; and these changes are accompanied, possibly, with more or less contraction of the vessels, and in some cases the coagulation of fibrin within them; conditions which, together, produce a *partial* or *complete obliteration* of the channels through which arterial blood should be supplied to that part.

---

* Op. cit., pp. 357, 358.

† Elementary Forms of Disease, 1838, Mortification.

Rigidity of the arteries, whether owing to calcareous or fibrous transformation of their parietes, predisposes to mortification, as already explained; and, in such cases, it begins in parts most distant from the heart. In the toes senile gangrene most frequently commences. This event may be aided by the defective action of a weak heart. In one of Sir B. Brodie's cases, with mortification of the right foot, the muscular structure of the heart was soft, thin, flaccid, and easily torn, and one coronary artery was impervious; in fact, those conditions were associated which, in all probability, denoted fatty degeneration of the heart. But this subject requires further investigation in relation to senile gangrene.*

The *symptoms* of ossification of the arteries, and therefore the earliest or premonitory symptoms of senile gangrene, affecting either or both legs, are, their numbness, coldness, and weakness. Clearly arising as these symptoms do from a defective supply of blood, they come and go. They are sometimes present, sometimes absent; present, when circumstances demand a more active circulation, which the unyielding arteries cannot allow; absent, when a sluggish circulation is sufficient. Rest, posture, and uniform warmth may maintain this sufficiency, and thus prevent gangrene, which is ever impending; while active exercise, implying more active circulation (such as the rigid arteries cannot allow), is soon attended with a sense of weakness in the limb, followed by coldness, numbness, and possibly gangrene, of one or more of the toes. Any posture or pressure that further impedes the sluggish circulation, retarded perhaps by exposure to cold, will further evoke these premonitory symptoms, and probably induce gangrene.

By connecting these—the premonitory and more constant, although fitful, symptoms of senile gangrene—with the circumstances under which they arise, the practitioner is led to adopt the most appropriate *preventive measures* against its supervention. Flying pains in the limb, or fixed pain in the spot where gangrene is about to commence, is an equivocal symptom, being present in

some cases, and most severely so; absent, or very slight, in others. Moreover, its significance is not understood. This symptom, therefore, affords no suggestion for the prevention of senile gangrene.

Generally a slight attack of inflammation, arising perhaps from some slight abrasion, scratch, or wound, is the immediate forerunner or immediate cause of this gangrene. Sir B. Brodie offers a reasonable explanation. During inflammation an increased supply of arterial blood is required, and the arterial trunks leading to the inflamed part become dilated so as to permit this extra quantity to enter; but when these vessels are ossified they lose their dilatability, the greater supply of blood demanded (by inflammation) is withheld, and the part perishes.

The *immediate* cause of senile gangrene being usually 'traumatic,' conveys an obvious yet most important lesson:—to avoid even the slightest operation on a part, the arteries leading to which may be suspected, from the symptoms, present or past, to have undergone calcareous degeneration. Sir B. Brodie candidly relates a case in point that occurred in his early practice. Having incautiously laid open a sinus on the instep of a man who had previously suffered gangrene of both feet with the loss of some toes, inflammation followed this slight wound and spread over the whole foot. Extensive mortification ensued the next day, and in two days more he died.

Beyond this general precaution, suggested by considering the etiology of senile gangrene, the peril of *sphacelus* cannot be prevented. Its *internal* causes are ever in operation, and ready to co-operate with some slight external cause, or some irregularity by the individual in his or her habits of daily life. The rigid or partly occluded arteries cannot adapt themselves to any demand for an increased supply of blood to the part endangered, and gangrene, ever impending, now begins. After a walk, or some additional impediment to the local circulation by pressure or an unfavourable posture, or after exposure to cold—any such circumstance, aided perhaps by a slight accident exciting inflammation

in the part, induces gangrene. So far as these unfavourable circumstances can be avoided by suitable precautions, they should be avoided when premonitory symptoms such as I have described have occurred, probably again and again, with increasing significance; and thus far senile gangrene is preventible; but its internal causes being persistent, the peril of sphacelus is imminent.

Supposing the process of destruction to have actually *commenced*, the probability or improbability of arresting its progress, of preventing *extensive* sphacelus, and of soliciting a line of demarcation between the living and the dead (thereby saving as much as possible of the part endangered), are considerations of a preventive character respecting *spreading* sphacelus, in relation to its consequent constitutional disorder; and when pertaining to senile gangrene, they can be at once determined by reference to its internal causes.

Observe, then, the first appearance of this gangrene, and watch its progress, guided by these causes.

The arteries are permanently rigid or obstructed for a considerable, but *unknown*, extent of their course leading to the part. Generally commencing on the pulpy portion of one or more of the toes, a port-wine red colour (emblem, generally, of past indulgence) or a black discoloration appears; in some cases preceded, as we have seen, by inflammation, in others by a stinging sensation, or by numbness and coldness, but frequently without any immediately local premonitory symptom. Soon a vesicle arises over this discoloured spot. More than one such vesicular spot may form. The vesicle or vesicles burst, and then the true skin, disclosed, is seen to be dead. It is encircled with a *dusky* red hue, shading off beyond. This death-warrant, advancing at first perhaps slowly, extends over the toes and back of the foot, uniformly and symmetrically, faithfully followed up by the black shade of sphacelus. Spreading equably, it reaches the rise of the instep, presenting an appearance as if the foot were thrust into a black slipper trodden down towards the heel; and, when the sphacelus has advanced a little further, presenting a singular black-hoofed appearance. At

length, gaining the ankle, life usually succumbs to the typhoid fever of spreading sphacelus; in some cases only, mortification extends upwards to the knee, or even to the thigh, always being preceded by the advancing dusky shade of death. Where will it cease to spread? At what part will the living organism have the power to detach itself by ulcerative disintegration from the dead part, excising a black, shrunken, mummified mass, or if both feet are mortified, a pair of hoofs, the very type of the residue of *dry* gangrene? Wheresoever the arteries convey sufficient blood for healthy nutrition, *there,* in that situation, this disjunction and severation of the living from the dead will be inaugurated. And ever and anon, when the smaller arteries conveying blood from *collateral* branches are less obstructed than usual, the part nourished from this tributary source appears less withered and sphacelated, and rather turgid, soft, plump, and purple-coloured, as if capable of being restored to life. In most cases the obstruction is more complete, and of considerable but unknown extent. To what extent, therefore, senile gangrene may spread, and where its progress will be arrested, cannot be determined *during life;* and is only known by the issue of the case. Nature alone decides *how much* of the limb will be spared, although a due knowledge of the causes in operation suggests appropriate therapeutic assistance.

Senile gangrene is not necessarily a result of *old* age. Those who, being young in years, have nevertheless undergone the degeneration of advanced life prematurely, are equally predisposed thereto. It was formerly held by Dupuytren, Cruveilhier, and other pathologists, that inflammation of the main artery leading to the part affected induced senile gangrene; and arteritis is certainly not peculiar to old age.

Arteritis is an occasional (internal) cause of spontaneous gangrene, resembling in appearance that which arises from calcareous degeneration of the arteries. That is to say, the gangrene induced by arteritis is equally *dry,* or more so, and horny.

Certain premonitory symptoms afford sufficient opportunity

for the prevention of gangrene arising from this cause. Pain, or at least tenderness along the course of the artery, and for a considerable extent, accompanied with firmness of the vessel—these are the symptoms which, if the artery be situated favourably for examination, and if heeded in time, may enable us to anticipate irreparable mischief. If neglected, there follows a very characteristic sign. By coagulation of blood within the artery, it feels like a hard cord, rolling under pressure with the finger. The presence or not of this cord will depend on the extent to which the vessel is occluded. Usually, as I have said, this is considerable; and other infallible signs speedily ensue, of the same character in all cases, but varying in degree with the size of the artery. If a main artery, as the femoral, then, the obstruction being extensive, shuts off all supply of blood by collateral vessels from the parent trunk; and the circulation is arrested throughout the whole limb, below the *commencement* of the coagulum. Pulsation ceases, and coldness, numbness, not without pain, for a time; weight in the limb, and loss of power, are like smouldering embers of vitality, soon extinguished in death. In such cases also the obstruction to any supply of blood is complete, and has operated suddenly; no effusion will have taken place, and the limb is shrivelled and horny—the very type of *dry* sphacelus. Discoloration, of a brownish black tint, spreads up the limb as high as the obstruction, and there terminates abruptly. Up to this point only is 'the cause in operation,' and here therefore it is that the living organism bids adieu to the dead part. The parting boundary line is here drawn by Nature, and not until thus determined is the progress of sphacelus consequent on arteritis arrested.

In further illustration of gangrene arising from a defective supply of arterial blood, occasioned by obstruction—there are cases, now and then, of a *fibrinous coagulum*, not formed in the artery in which it is found, but washed from the left ventricle of the heart, and carried along by the current of blood to that vessel, in which it becomes arrested. Such cases, contrasting in respect of their

pathology, contrast also in their premonitory symptoms. A very remarkable instance of this kind is recorded by Mr. W. H. Flower:*

"A married woman, æt. thirty-nine, was admitted into the Middlesex Hospital under the care of Mr. De Morgan, on the 4th of March, 1856. She was tolerably stout, robust-looking, and two months advanced in pregnancy. About three weeks ago she began to feel shooting pains, with a sensation of numbness and coldness, in both legs and feet. After stooping down to pick something from the ground (six days previous to her admission), these symptoms became increased in the left, but passed away completely from the right, leg. On admission, there was no pulsation in the left femoral artery, which could be felt, hard and cord-like. The foot and leg, nearly as high as the knee, swollen and crepitating under pressure, was of a purplish green colour; the toes nearly black, covered with bullæ, containing a dark fluid, cold, without sensation, and emitting a gangrenous odour. The right leg was natural. Great anxiety of countenance; pulse small and irregular; the general surface cold and moist. The gangrene continued to extend, reaching ultimately the upper part of the thigh; the general symptoms increased, and she died two days after her admission.

"On *post-mortem* examination, thirty hours after death, a firm, yellowish white, elongated, fibrinous coagulum, about the size of an almond, but rather thicker, was found lying in the entrance of the left common iliac artery, which it completely obstructed, its upper end projecting into the aorta: it was *not adherent to the artery*, and the *lining membrane* of the latter presented *no discoloration* or *sign* of *inflammation* at this part. Below, the common iliac, external and internal iliac, and femoral arteries, with all their branches, were filled with dark-coloured and tolerably firm coagulated blood.

"The pericardium contained about an ounce of straw-coloured serum, and a little recent lymph was deposited on the surface of the heart, near its base. This organ was flabby, with much fat about its base, and weighed eleven ounces and a half.

* Trans. Path. Soc. London, 1855–6, vol. vii., p. 175.

"The right and left cavities both contained a considerable quantity of coagulated blood; embedded therein, and not adherent to the walls of the heart, were three or four firm, whitish, fibrinous coagula, about the size of hazel-nuts, exactly resembling that in the iliac artery, but firmer, more opaque, and of altogether different appearance from the ordinary colourless coagula usually found in the heart after death. Owing to an accidental circumstance in the examination of the heart, it was not ascertained in which of the cavities these were found, but it is believed that they existed in both the right and left ventricles. The mitral valve was much thickened, the orifice contracted, the aortic valves somewhat thickened and shrunk."

I have italicised the condition of the left common iliac artery at its origin, where the fibrinous coagulum was arrested, because the absence after death of any trace of inflammation (at this part of that vessel), and the absence also, during life, of any such premonitory symptom as pain or tenderness, for a considerable extent of the artery before it becomes plugged up, are negative circumstances which together distinguish the operation of this cause of gangrene. The absence of pain or tenderness is, of course, alone *available* in diagnosis.

As to the rationale of these cases, the above report rightly concludes: "There can be little doubt that the coagula found in the heart had formed some time previous to death, being the result of some peculiar condition of the blood; and the symptoms would indicate that one of these, having escaped from the heart, and found its way into the general circulation, had at first been arrested at the bifurcation of the aorta, where it gave rise to partial obstruction of the circulation of both extremities; afterwards, perhaps in the act of stooping down, it became tilted into the left iliac artery, completely obstructing that vessel, while the current of blood was thus allowed to pass freely again through the right leg."

This explanation applies, *mutatis mutandis,* to *all* other cases, and their pathology suggests certain practical deductions

concerning the probability or improbability of preventing the impending gangrene, and of arresting its progress when once established.

Firstly, it being uncertain in what artery the fibrinous coagulum will eventually be impacted, the symptoms shift about from one part to another, misleading observation. In the case referred to, when the coagulum hitched upon the bifurcation of the abdominal aorta, both legs shared the symptoms of gangrene; ultimately the left only was affected. Again, there being no premonitory symptoms—as pain or tenderness along the course of the artery about to be visited with coagulum, precludes any opportunity for preventing gangrene, which immediately commences on occlusion of the vessel. Lastly, the progress of the gangrene thence arising cannot be determined otherwise than by the source of obstruction, and accordingly our preventive measures fail to arrest and circumscribe the spreading of sphacelus.

Coagulation is induced within a main artery by *mechanical violence* acting in a peculiar way. Blows may crack the internal and middle coats of a large artery, whereby they fold inwards across the stream of blood, of which lesion there are two specimens in the Museum of St. Bartholomew's (Paget). This being the immediate cause of coagulation in such cases, I should presume that sphacelus cannot be prevented, nor its progress arrested, until advanced as high as the seat of injury.

Aneurism is another lesion tending to sphacelus, and by a twofold mode of operation. Partly, by interrupting the free flow of (arterial) blood through the artery itself, and partly by pressure on adjacent veins, obstructing the return of venous blood. Popliteal aneurism has thus induced gangrene, soon passing into sphacelus. A case in point is related by Sir A. Cooper.* Gangrene commenced at the ankle, and the process of separation detached the dead tissues down to the bone. Fortunately, such cases must be, now-a-days, very rare. Sometimes, however, an internal aneurism,

---

* Lectures, 1839, p. 107.

situated altogether beyond relief, induces gangrene, it may be, of some distant part. In one case,* reported by Dr. Cockle, aneurism of the ascending and transverse aorta terminated fatally in about three years from asthenia, without rupture of the sac; and gangrene of the nose began three days before death.

Should gangrene not arise during the progressive enlargement of aneurism of a main artery, it will assuredly supervene. When the vessel bursts, gangrene immediately commences and speedily terminates in sphacelus. A case, apparently of this kind, is related by Mr. Wood.† Ruptured aneurism of the popliteal artery was immediately followed by all the signs of false aneurism; namely, sudden and considerable swelling, ecchymosis, numbness down to the toes, and pain in the limb, cessation of pulsation in both arteries, and gangrene passing at once into sphacelus, involving the whole limb, *at least* as high as the seat of injury, and probably extending beyond, under the influence of another aneurism, nearer the heart.

Now, in all cases the earliest and most exact diagnosis will ensure the opportunity for preventing gangrene arising either during the progressive enlargement of aneurism, or when it becomes converted into a false aneurism. And if the cause in operation thus foreseen be situated so as to allow of surgical interference, gangrene can be prevented. In fact, aneurism is now more a matter of pathological than of practical interest in connexion with mortification. But, having commenced, the progress of sphacelus up to where the artery is ruptured cannot be arrested.

A main artery may be *wounded* so as to cause mortification. Punctured wounds in some instances,—*e. g.*, a stab in the thigh, penetrating the femoral artery, has this issue; of which there are cases on record. Or, by gunshot wound, a main artery may be *bruised*, without being opened at the time; but after some days

---

* Trans. Path. Soc. Lond., 1858, vol. ix., p. 97.

† Ibid., p. 122.

have elapsed, sloughing of the vessel will be attended with considerable hemorrhage and false aneurism, followed by mortification. Or a main artery may be *lacerated* by extensive contusion, without even abrasion of the skin; but accompanied with considerable hemorrhage and false aneurism, followed by mortification. Such was partly the cause in the case related at pp. 528-9.

A simple fracture has been known to lacerate a main artery and cause mortification. Sir A. Cooper* once saw this kind of injury. A gentleman was thrown out of a gig while going down hill, and the wheel of the carriage passing over his thigh, produced a simple fracture of the femur. But some peculiarities were immediately observed at the time of the accident. The lower part of the leg was quite insensible, considerably swollen, and hard. After lying in bed for a week, the patient became very restless, and desired to be moved. This was done in the gentlest manner possible. He did not, however, experience the relief he expected from a change of position, and the swelling in some degree increased. Sir A. Cooper was then consulted. Pulsation could be felt opposite the fracture. Gangrene had already commenced at the knee. The limb was emphysematous, and gas had extended in the cellular tissue up the thigh to the abdomen. Amputation would have been useless. On examination after death, the femoral artery was found divided.†

In considering these and similar occasions of gangrene, from a preventive point of view, I would disregard the imperceptible differences commonly urged, of *true* and *false* aneurism; and observe only the practical distinctions of 'circumscribed' and diffused,'‡ as implying very different degrees of tendency to gangrene. Thence the great importance of the earliest and most exact diagnosis,—first, of the fact of aneurism having formed; and

---

* Op. cit., p. 109.

† For cases of fracture, simple and compound, complicated by false aneurism, see Principles of Surgery, John Bell, ed. by Charles Bell, 1826, vol. iv., pp. 390, 403, 407, 411.

‡ See chapter iii.

secondly, whether it be diffused and diffusing. Take the last-mentioned case. A week at least elapsed before the nature of the injury, beyond that of simple fracture, seems to have been clearly understood; and *then* gangrene had already commenced. Such diagnosis, therefore, is imperative; and this being combined with the cause of aneurism, as *traumatic,* or *idiopathic* and necessarily associated with a diseased condition of the aneurismal artery, suggests the propriety of applying a ligature, either immediately above and below the aperture in the vessel, or at some convenient distance from the seat of aneurism. The probability of gangrene supervening will depend on the efficiency of the collateral circulation. Thus much for prevention. But gangrene, once established by aneurism of a main artery, speedily issues in sphacelus, and its progress cannot be arrested, and the limb saved, beyond the situation of aneurism.

Associated with aneurism are the effects of a ligature applied to a main artery. I shall recur to this subject presently.

Among the causes of mortification arising from the want of a free circulation, many operate by obstructing the return of venous blood through the chief vein or veins of the part affected.

Phlebitis I place foremost, not as a frequent cause of this kind, but as exhibiting the most characteristic and premonitory appearances of gangrene from venous obstruction. *Phlegmasia dolens* for example. Dr. D. Davis* was perhaps the first who demonstrated the pathology of this disease. His dissections showed that the iliac and femoral veins are inflamed, a condition which fully explains all the symptoms observed, and the order in which they occur. Pain, or tenderness on pressure, is experienced along the course of the femoral vein to a considerable extent, which may yet be supple. But the blood within the vein soon becomes coagulated and mixed with pus; in fact, *suppurative* phlebitis is established. The vein now feels, if it can be felt at all, like a hard cord, and still very painful. No blood passes

* Med.-Chir. Trans., 1823, vol. xii.

through it, and this soon tells on all the tributary veins throughout the limb. Its cellular tissue becomes gorged with serum, giving to the whole limb a stuffed appearance, and when handled, shows the impression made by the fingers. Shortly, this *œdematous* swelling becomes hard and polished, of a pearly white colour, or mottled like marble, cold and insensible,—a marble limb, and much larger than its fellow. And this condition borders on gangrene. Preventive measures—both local and constitutional, as in arteritis—will avail most, when first, the earliest symptom, tenderness is felt along the course of the vessel.

Tense anasarca arises, once in a way, from *fibrous* obliteration of the companion vein to a main artery. Such a case is reported by Dr. J. W. Ogle.* Among other morbid conditions, there was —"total obliteration of the inferior vena cava of old standing." The right supra-renal and renal veins were natural. The entrance of the left renal vein into the inferior cava was open and free; but below, the entire vena cava was filled with firm, fibrinous, indurated material, and was shrunken and reduced to the state of a dense thick cord. This condition appeared to be of long duration, as the contained material was with great difficulty separable. A considerable quantity of soft recent fibrin blocked up the left external iliac vein.

Phlebolithes, or vein-stones, are not unfrequently found after death, within various veins, more particularly in the iliac, and their branches. These bodies are either free or attached to the vessels, from the inner surface of which they crop out and gather fibrin around themselves, as the eddying blood slowly passes by. And if one such body—situated in a principal vein, as either common iliac—becomes too large to be easily accommodated, it will seriously obstruct the return of blood from the whole corresponding lower extremity. An ossified calcareous deposit, of unusual size, that occupied the common iliac vein, is described by Dr. Ogle† as

---

* Trans. Path. Soc. London, 1855–6, vol. vii., p. 177.

† Ibid., p. 133.

having measured about two inches in length, and tapering to its extremities from the middle part, which was between one quarter and one half of an inch in thickness. The condition of the corresponding lower limb is not stated, but such a phlebolithe, and so placed, should be remembered in considering the *possible* causes of extensive venous obstruction. Otherwise, a large phlebolithe, and thus situated, being irremovable by surgical aid, has little practical interest. And this remark applies also to fibrous obliteration of the companion vein of a main artery. Nevertheless we gain something by knowing that the cause in operation cannot be overcome. Fortunately, however, in most instances, when venous obstruction has been *gradually* established, an adequate compensation is provided, by the contemporaneous enlargement of other veins, through which the blood flows without impediment. The sufficiency of this provision to prevent anasarca is shown in cases of old standing, yet complete, obliteration of the inferior vena cava. Such cases are described by Matthew Baillie* and Mr. James Wilson.†

In relation to impending gangrene from venous obstruction, there is a species of aneurism, first described, and admirably so, by Dr. W. Hunter,‡ and subsequently known by the name of *aneurismal varix*. This kind of aneurism arises whenever and wherever a suitable opening is established between an artery and a vein of tolerable size, which lie in contact with each other. Therefore, with respect to the thigh,§ the ham,‖ the neck,¶ the arm,** and other situations,†† there are cases recorded. At the bend of

---

* Trans. of a Society for the Improvement of Med. and Chir. Knowledge, 1793, vol. i., p. 127.

† Ibid., 1812, vol. iii., p. 65.

‡ Med. Obs. and Inq., vol. i., p. 340; vol. ii.

§ Mém. Chir. sur Différentes Espèces d'Anevrismes, par G. Breschet, 1834.

‖ Mém. de Chir. Clin., Larrey, t. iii. ; and Mém. de Chir. Mil., t. iv., p. 340. Méd. Opér., Lassus, 1794, t. ii., p. 442.

¶ Op. cit., Breschet.—Primum op. cit., Larrey, t. iii.

** Secundum op. cit., Larrey, t. iv., p. 341.—Op. cit., Breschet.

†† Primum op. cit., Larrey, t. iii.—Lond. Journ. Med., 1850, case by W. Cadge.

the elbow* aneurismal varix was first observed. Here it is chiefly known as the result of unskilful venesection.

In some cases this kind of aneurism is not 'traumatic;' but 'spontaneous;' it arises from an ordinary aneurism bursting into a neighbouring vein. For example, the abdominal aorta and vena cava have thus communicated just above their bifurcation;† and in the same way an opening has been established between the femoral artery and vein.‡ Under these circumstances the vessels usually communicate by only *one* opening. And if aneurismal varix have a traumatic origin, there *may* be only one opening. Thus, by gunshot wound, the ball has passed between a large artery and vein, and opened both into one.§ In one instance,‖ the aperture was occasioned by a heated iron rod having been thrust between the femoral artery and vein. Traumatic aneurismal varix is generally produced by some sharp instrument having transfixed the vein and penetrated the artery, making therefore *two* openings in the former, and one in the latter. In a case recorded by Lassus, the popliteal vein and artery were thus wounded, and made to communicate, by a sword-thrust; and similarly the median basilic vein and brachial artery have been wounded at the bend of the elbow by unskilful venesection. In such case the lancet transfixes the vein and punctures the artery beneath, having passed through the intervening tendinous expansion which stretches from the biceps to the aponeurosis of the arm. The superficial opening in the vein and skin may soon unite; while the deeper aperture in the vein and that in the artery remain, under the constant pressure of the current of arterial blood; and then they become blended into one direct communication of the two vessels. This is soon followed by engorgement, and a varicose condition of the vein—an aneurismal *varix*—is formed. Sometimes, however,

---

* Op. cit., W. Hunter, vol. i., p. 340.

† Edin. Med. and Surg. Journ., vol. xxxvi., J. Syme.—Lond. Med. Gazette, vol. xiv., p. 462, Robinson.

‡ Med.-Chir. Trans., vol. xx., Perry.

§ Diseases of the Arteries, Hodgson. ‖ Ibid.

when these openings are not in apposition,—owing either to the arterial puncture having been made obliquely from the vein, or to the vessels not lying in contact,—the jet of arterial blood passing into the vein is delayed in its passage, and burrowing (for itself) a cavity in the intervening cellular texture, forms a circumscribed "false" aneurism. This condition, by virtue of the varicose condition of the vein with which it communicates, is named *varicose* aneurism.

Whether an intervening false aneurism be formed or not—whether the communication of artery and vein be indirect or direct, the same *symptoms* follow, and they are very significant. The current of arterial blood being partly diverted from its course, is less forcible below the seat of injury; consequently pulsation below at the wrist is feeble; and as the jetting stream passes through the median basilic into the veins of the fore-arm and arm, they become distended and tortuous—varicose. Therefore the median vein more especially assumes an eel-like appearance down the fore-arm, while the median basilic and cephalic, the radial and ulnar veins, with the basilic and cephalic veins at the bend of the elbow, also exhibit in various degrees the force of the arterial current; they all become enlarged and varicose. Aneurismal varix simply, or *plus* a false aneurism, is established; the former term being then exchanged for varicose aneurism. In either case, the *varicose* condition of the neighbouring veins therewith connected is the *earliest* and *most characteristic* appearance. The aneurismal swelling is wholly or partly formed, as the case may be, of enlarged and tortuous veins. And (this swelling) these veins pulsate in unison with the (arterial) pulse, their pulsation being visible, perceptible also as a tremulous thrill, and audible as a soft, burring sound. Moreover, the turgid veins can be emptied by pressure upwards in their course; or they subside and regain their former dimensions, when pressure is made on the aperture in the artery. An elevated position of the limb will have the same effect. If allowed to progressively enlarge, the skin shares the tint of venous obstruction, assuming a livid hue;

while the whole limb downwards from the seat of injury becomes swollen, œdematous, feeble, numb and cold, even threatening gangrene. True, there are cases* on record of aneurismal varix having remained without further inconvenience for many years, to fourteen, twenty, and thirty-five years. Generally speaking, this stationary *quietus* is not granted.

Yet the tendency to gangrene is amenable to preventive measures in most instances. Bandaging the limb from below upwards, with a pad placed over the aperture in the artery, will generally prove sufficient on behalf of aneurismal varix alone, and if applied at an early period of its course. When, however, from unusual swelling over the wound in the artery, and swelling evidently not that of a varicose vein, there is reason to suspect the (additional) complication of a false aneurism, then the only alternative is to ligature the artery above and below its opening. The greater tendency to gangrene from *varicose* aneurism, may thus be anticipated in most cases. Should either this condition or simple aneurismal varix have existed for some time, the operation alluded to will be less practicable. The artery above its aperture will have become so much dilated as scarcely to allow of being successfully closed by ligature. A remarkable preparation belonging to Professor Pirrie is figured in his work.† The portion of artery above the aneurismal sac had acquired the dimensions of (human) small intestine. Hence the value of a sufficiently early and correct diagnosis, such as I have endeavoured to gather from pathology for the effectual employment of *preventive* measures.

All the foregoing occasions of venous obstruction are, in relation to gangrene, interesting, chiefly as affording a *favourable* comparison with diseases and injuries of arteries of similar character. Gangrene, however, unquestionably may arise from venous obstruction.

---

* See works by W. Hunter, Guattani, Scarpa, Pott, and B. Bell.

† Principles and Practice of Surgery, 1860.

Prolonged *pressure* on a large vein, otherwise in a healthy condition, has this effect. Tumours, therefore, in the course of their growth, are liable to induce gangrene. In a fatal case, recorded by Fabricius Hildanus, of mortification of both feet and legs, a scirrhous tumour was found, after death, compressing the inferior vena cava and aorta, near their bifurcation. Here an inadequate supply of arterial blood co-operated with the retarded return of venous blood. A ligature bound around any part of the body is another mode of pressure which may induce gangrene; and in most instances of this kind also, the arterial and venous currents of blood are both intercepted, although perhaps not equally. Tight bandaging thus operates. After venesection in one instance, a bandage was applied so tightly around the bend of the elbow, and rendered tighter by flexing the limb, that gangrene of the hand ensued, a wax model of which is preserved in the Museum of University College, London. Incautious bandaging in the management of fractures has occasioned a similar result. Phymosis is sometimes converted into strangulated paraphymosis. A man, drunken and reckless, withdraws the contracted orifice of the foreskin behind his glans penis. This stricture soon tightens with swelling, and mortification follows. Several such cases have come under my observation among hospital out-patients. Strangulated hernia is another familiar example of gangrene, perchance sphacelus, consequent on a tight ligature. Sometimes this result is sought as the object of surgical treatment. Piles are tied, and being strangled slough off in a few days. To accomplish this object satisfactorily the ligature must be drawn quite tight and allowed to remain. If either precaution be neglected, the part will probably regain its vitality.*

Now these two considerations, so important in the practice of surgery when sphacelus is sought, suggest the *kind* and *amount* of our preventive measures, when, as in most instances, an evil

---

* See an instructive case, Lectures by Sir B. Brodie, p. 307.

result is to be averted. Remove the occasion of pressure, whether a tumour or a ligature, and the precursory gangrene will itself probably be prevented. Should gangrene have already commenced, it may be stopped short of sphacelus, provided the cause in operation be not suffered to continue and the local circulation completely intercepted. Strangulated hernia, taken in an early stage, illustrates both these redeeming qualifications. A knuckle of gangrenous intestine will recover itself when the 'stricture' is divided, and the chance of recovery is regulated by the more or less early period at which the stricture is divided. The mortality of strangulated hernia increases in a high ratio, hour by hour, as the operation, when necessary, is postponed.

Sometimes the occasion of pressure cannot be removed. Internal tumours offer this difficulty. Yet the chances of gangrene supervening, even in such cases, will depend on the *degree* of embarrassment to the circulation in the part threatened—say, either lower extremity by reason of a pelvic tumour; and this embarrassment will also be more or less, according to the *rapidity* or slowness with which the occasion of pressure has come into operation. Slow-growing tumours allow time for a complementary circulation to become established in many cases, when the main trunks fail to transmit blood. Here, therefore, we are able to aid this compensation by the advantages of a favourable position and artificial warmth. The efficacy of these preventive measures is witnessed after the operation for aneurism. A large artery is ligatured, and thus a known cause of gangrene purposely brought into operation, and allowed to continue for the sake of a great good eventually. But in promoting coagulation within the aneurismal sac, and obliteration of the artery for a considerable extent of its course, we rely on the gradual establishment of a complementary supply of blood through collateral branches, and we solicit this result by a favourable position of the limb, and loosely swathing it in carded wool.

All the foregoing diseases and injuries of arteries and veins, including pressure acting in various ways on either or both kinds

of vessels, induce gangrene by reducing the *quantity* of blood requisite for the proper nourishment and maintenance of the part affected. Venous obstruction, retarding the onward current of arterial blood, *virtually* reduces the quantity supplied. In such cases, therefore, the *principle* of *prevention* respecting gangrene is to *restore* an *adequate* supply of *arterial blood;* this of course implying the removal, when practicable, of any impediment—arterial or venous. Otherwise the defective local circulation is equivalent to the loss of so much blood to the part deprived of its proper share. The actual loss of a large quantity of blood has, indeed, been known to cause gangrene in parts most distant from the heart. A drunken man was bled, and lost not less than three pints of blood. On the morrow both feet were mortified from the extremities of the toes to the instep. With wine and nourishment the sloughs and dead bones separated, and the stumps of the feet healed.*

In a case such as this, mortification is a sign of defective *systemic* circulation, and the extent to which sphacelus may spread is unlimited; but in all cases of mortification arising from the *local* causes hitherto considered, the faulty condition of a main artery, vein, or both, determines the boundary line of sphacelus. Hence the situation and character of various diseases and injuries of the larger blood-vessels are primarily important in relation to the question of arresting spreading sphacelus, and *à fortiori* to the prevention of gangrene. A sufficiently early and an exact diagnosis, through pathology, of the cause in operation, suggests appropriate preventive measures; and from this point of view I shall now pass on to another occasion of defective supply of blood.

Inflammation, terminating in mortification, involves *compression* of the *capillary* vessels. It will be remembered that undue effusion of lymph and serum, often to a considerable amount, is an essential element of inflammation, and that it reaches its height

---

* Lectures, cit., p. 350.

just before suppuration begins in the substance of any part, thereby *occluding* the capillary vessels at this stage of the inflammatory process. Consequently, pending suppuration, and while pus continues to form, the textures of the part are dying, disintegrating, and being absorbed, to make room for this new product. Mortification and absorption usually advance together at an equal pace, in relation to pus-production. Inflammation, terminating in suppuration and in mortification, are *collateral* events. The premonitory symptoms of suppuration are those also of gangrenous inflammation. When the pain becomes more acute and burning, with *throbbing*, when the skin acquires a deeper shade of redness, and when the swelling enlarges considerably, we may know that, in the subjacent textures, gangrene, no less than suppuration, is at hand.

Sometimes the textures die faster than absorption can at once dispose of them, and then mortification—as *sloughing*—is plainly visible.

The pathological condition of the part affected supplies, as usual, a satisfactory explanation of the *peculiar* symptoms, which are now those of gangrenous inflammation as distinguished from suppuration. The capillary vessels of the textures doomed to death, being compressed by interstitial effusion, the nerves included suffer pressure, while all the textures imprisoned and deprived of blood are degenerating,* dying, disintegrating, and softening. *Acute burning pain* is experienced rather than that throbbing sensation which the pulse-waves of blood excited when beating against the surface of a more solid and impervious effusion. Yet the capillary vessels do not transmit blood; circulation ceases in the part under the pressure of interstitial effusion; consequently the redness deepens into purple, and thence to a livid hue. These shades of colour are well seen when the skin is undergoing *gangrenous* inflammation. Beyond the limits of tense effusion and pressure, the vessels are still conveying blood with some

* Lectures, Paget.

force towards the part—are still exhibiting determination of blood; and there the leaden lividity and purple redness shade off to the more crimson hue of ordinary inflammation. In this situation interstitial effusion is still progressing and the swelling still enlarging; particularly when gangrene, at the more central part of the swelling, is about to commence. Lastly, the character of the swelling in its central gangrenous portion is peculiar. Here the dying textures are disintegrating and softening, macerated also by the effused fluid in which they are soaked. Hence the flabby softness which generally characterizes gangrenous inflammation.

No better illustration of these symptoms can be offered than *phlegmonous* erysipelas. The acute burning pain, sometimes at an early stage accompanied with throbbing; the purple hue and livid tint, presenting a mottled appearance, and the swelling enlarging occasionally to a prodigious size, so that an arm or leg shall be twice that of its fellow; this swelling, hard circumferentially, *boggy*, pitting easily, and flabby within, as gangrene of the subcellular texture and skin, rapidly supervene. The same general description holds good of bubo, about to slough extensively rather than suppurate—a condition frequently noticed among the more unfortunate, under-fed, and gin-drinking females who come from Woolwich to the Royal Free Hospital. To these illustrations may be added the usual characters of carbuncle, especially when this form of gangrenous inflammation occurs in a person of advanced age and intemperate habits.

The symptoms of gangrenous inflammation vary somewhat with circumstances. They are never altogether absent in gangrene resulting from inflammation; nor are they together present in any other disease. *Collectively*, therefore, these symptoms cannot mislead. Modifications, however, are met with.

Age, sex, temperament, daily occupation, and habits of life, predispose, not only to inflammation, but also to this or that issue, and modify its characters. All these conditions and circumstances of the individual centre their influence, either by affecting

the composition and vital endowments of the blood, or the intensity of the inflammation; in either way modifying the character and amount of the effusion.

Certain inflammations, decidedly gangrenous,—such as carbuncle and boil, plegmonous erysipelas, sloughing bubo, and certain secondary syphilitic ulcerations; hospital gangrene, malignant pustule, malignant scarlatina, and small-pox,—severally indicate the operation of 'blood-poisons,' or at least morbid conditions of the blood; the intrinsic nature of which, although at present unknown, assuredly modifies the characters of these several forms of gangrenous inflammation. The premonitory symptoms may be generally described by the term *asthenic*.

'Intensity of inflammation' induces gangrene, by the more forcible determination of blood, yielding a more solid and extensive effusion. The accompanying swelling is more tense, the pain more acute, and the overshadowing redness sooner becomes purple. Such premonitory symptoms are *sthenic*.

The kind of 'texture' affected with inflammation predisposes to this or that issue, and when gangrene ensues, modifies its symptoms. A striking illustration is phlegmonous erysipelas in various parts of the body. Ordinarily it extends below the skin, and through the subcellular texture, down to the aponeuroses. These textures being ill-supplied with vessels, readily become gangrenous, under pressure of the abundant effusion into the cellular texture. The symptoms are those of ordinary phlegmonous erysipelas, as seen in either of the extremities. Sometimes, however, this kind of inflammation occurs beneath the tendinous expansion of the occipito-frontalis muscle. Effusion taking place to a considerable extent, and chiefly *under* this unyielding texture, is soon followed by gangrene and sloughing of the tendon, scalp, and pericranium. A large surface of the cranium may be exposed. The premonitory symptoms are peculiar:—considerable œdematous swelling and pain, without any redness of the skin, which is rather paler than usual; a waxy, pitting, painful swelling, spreading over the whole scalp to the forehead, and thence over the face—by effusion into

the subcutaneous cellular tissue, the skin there acquiring a shade of purple colour.

But the symptoms of gangrenous inflammation are always sufficiently characteristic, although modified by circumstances. They have a general resemblance to those of gangrene from venous obstruction. In both, the gangrene is *humid*, with considerable swelling and purple or livid discoloration, contrasting in these respects with that arising from a directly defective supply of arterial blood, in which case the gangrene is *dry*, with shrivelling, and brown or black discoloration.

The humid character of gangrenous inflammation is due to effusion of lymph and serum, under the hydrostatic pressure of which mortification quickly supervenes. This antecedent pathological condition suggests the appropriate preventive measure. Ere the swelling becomes tense, punctures, or free incisions if necessary, into the cellular texture infiltrated, will instantly remove or relieve pressure, and anticipate the impending destruction of texture. The efficacy of this measure, employed at a sufficiently early period, is witnessed in phlegmonous erysipelas, including the variety to which I have alluded. Respecting that peculiar œdematous swelling, nothing but incisions promptly made here and there, down to the bone, and repeated as the swelling extends, can save the occipito-frontalis muscle and scalp from sloughing.

If mortification has already begun, and is spreading, the same measure is still absolutely necessary to *arrest* its progress. For example, in the course of carbuncle, if an incision be made within the limits of this swelling, sloughing of the subcellular tissue will spread. After such an incision the brawny swelling continues to extend, and with it the slough beneath; whereas a crucial incision down to the slough, and terminating either way in healthy texture, beyond this tense swelling, will probably limit the sloughing. The dead textures loosen and come away.

To effectually prevent gangrene supervening on inflammation, it is obviously necessary in all cases to remove any 'external' cause which may still be exciting inflammation. In many instances,

these causes are only *momentarily* applied, as by a severe blow, squeeze, or wrench. In other cases, however, the exciting cause *continues*. Pressure, as when a man has lain for days or weeks on a mattrass, will occasion gangrenous inflammation of the skin over prominent parts; *e. g.*, the sacrum and great trochanter. A sensation of pricking, as if crumbs of dry bread were between the parts pressed and the mattrass, and a purplish discoloration of the skin, foretell the approach of sloughing, and that bedsores are about to form. In anticipation, therefore, of these symptoms, and of gangrene when they are present, the portion of skin in peril should be fortified by occasionally washing it with a stimulating lotion of spirit and water; while pressure is relieved by shifting the patient's position from time to time. Or if the recumbent position be absolutely necessary, as in fracture of the spine, pressure must be relieved in some other way. An air-cushion having a hole in its centre should be laid under any suspicious part, bulging of the skin being prevented by a layer of soap-plaster. These appliances are superseded by a water-bed, which equalizes the pressure over the whole back.

Among the circumstances that predispose inflammation to become gangrenous, I have mentioned the kind of *texture* in which it may chance to occur; and now in resuming the 'general' etiology of mortification, it is necessary to take into consideration the tendencies of different anatomical conditions, as represented by the physical properties and structure of different textures.

Respecting physical properties, many textures are "soft" as compared with others,—bone and cartilage, which are "hard;" but, of soft textures, some are tough and *unyielding* investments. Such are fasciæ and aponeuroses. They predispose subjacent textures to gangrene, by subjecting them to pressure, whenever an effusion of any kind takes place therein. Hemorrhage, for example, under a strong fascia or aponeurosis is apt to occasion gangrene. The bursting of a femoral aneurism is followed by mortification of the limb, chiefly because the main artery ceases to convey blood

below the rupture, partly because the extravasated blood confined under the unyielding fascia lata compresses any collateral vessels by which a due supply of blood might become established.* That this source of pressure may be alone sufficient, is shown by gangrene arising when a *subordinate* artery is wounded under cover of a strong aponeurosis. Division of the posterior tibial artery causes considerable swelling and tendency to gangrene; of which a case occurred to Mr. S. Cooper.† But the point I am urging is most clearly demonstrated, when a smaller artery, under an unyielding aponeurosis, is wounded, and gangrene ensues; although the chief arterial trunks still remain free to maintain an adequate supply of blood. They are incompetent under pressure of the blood extravasated. Thus, hemorrhage from the peroneal artery places the tibial arteries in this predicament, and has been known to jeopardize the foot and leg.

Then again, with regard to inflammatory effusions; I have known a small hard (inflammatory) swelling in the vastus externus muscle pass rapidly into gangrene, involving the whole muscle, which became a pulpy grumous mass; sudden gangrene resulting, I should say, from pressure, under the *unyielding* fascia lata that covers the external vastus. I put this construction on a case recorded by Mr. Guthrie, and which I have more than once referred to. After a gunshot wound in the thigh, a fortnight elapsed without an unfavourable symptom, and then the man stated he felt quite well. Inflammation came on in the night, internally, deeply, and scarcely affecting the skin with redness. Early in the morning the man died. Dissection showed that the whole thigh was disorganized; and Mr. Guthrie could only compare its appearance to that of a part just falling into gangrene. A very remarkable case, illustrative of gangrene being speedily induced by inflammation, when under pressure of an unyielding aponeurosis, occurred in the Royal Free Hospital. A man in a

---

* See two cases of this kind, Lectures on Path. and Surg., Brodie, p. 315.

† Dict. of Prac. Surgery, 1838, p. 630.

dying state was admitted under Dr. O'Connor. He had considerable swelling of the abdomen, chiefly coextensive with the rectus muscle. This swelling was soft, obscurely fluctuating, and exquisitely tender. The skin over it presented a uniform blush of purple redness. Very shortly, the man became comatose and died. I assisted at the post-mortem examination; and we found within the sheath of the rectus muscle,—instead of muscular texture,—a greenish brown quagmire of slough and pus. Near the umbilicus, there was a small irregular-shaped aperture in the posterior aspect of the rectal sheath, which thus communicated with the peritoneal cavity. In contact with this aperture lay the vermiform appendix (cœcum coli), and within it a bean was found impacted, and pointing towards the opening in the rectal sheath. This appeared to be the root of all the mischief, yet a mighty effort of Nature to dislodge and discharge a foreign body through the abdominal parietes. It failed in consequence of extensive sloughing, apparently induced by the unyielding resistance of the rectal sheath.

Looseness of texture also predisposes to mortification, by allowing an interstitial effusion of blood, lymph, or serum, which accumulating, compresses the nutrient vessels. Severe ecchymosis, not occasioned by direct violence to the part affected, is a familiar example of interstitial extravasation of blood, tending to gangrene. Traumatic gangrene is partly of this kind, but to it I shall recur. Phlegmonous erysipelas—the very type of *gangrenous* inflammation—shows the effect of looseness of texture in connexion with inflammatory effusion. Lymph and serum both extensively infiltrate the cellular texture, which rapidly sloughs.

Besides those predisposing *physical* circumstances, the *structure*, in one sense, of any part predisposes to its mortification. The proportion of blood-vessels it possesses is the structural condition alluded to. And here also both extremes meet in relation to gangrene. A scanty supply of blood-vessels, and these scarcely destined for the tissue through which they pass, but mostly to be distributed in adjoining textures, is a comparatively *avascular* con-

dition, having a gangrenous tendency; as, for example, the liability of cellular tissue to slough. So, again, an abundant supply of blood-vessels—a *highly vascular* condition—predisposes to mortification, by favouring the intensity of inflammation and effusion. The skin, therefore, as compared with fibrous textures, is more liable to gangrenous and sloughing inflammation. After an extensive burn of the hand or foot, the tendons endure to the last; while, after intense inflammation of the skin, for a few days only, sloughing supervenes. Thus, the lower lip has speedily assumed a gangrenous appearance, and sloughed, from the bite of an insect.*

Predisposition to gangrene, as connected with textural conditions, is most obvious when two at least of them *co-operate*. Cellular tissue, being comparatively avascular, as well as liable, by its looseness, to suffer from interstitial effusion, most readily sloughs.

All other textures are liable to undergo gangrene; and their respective liabilities, by virtue of their respective conditions of texture—physical and vascular—are suggested by the foregoing explanation. Artery is most capable of resisting mortification. Sloughing bubo may lay bare several inches of the femoral artery without opening it. On the contrary, in this and other instances, where a large artery is exposed by sloughing, the vessel becomes more vascular, covered with imperfect granulations, and thicker. Sometimes, however, even a large artery yields to the ravages of sloughing—an event occasionally witnessed in sloughing bubo. Hennen† relates a remarkable case in point, of which the chief features were: ulceration opening the femoral artery from sloughing bubo in the groin; ligature of the external iliac artery; mortification of the leg, with a line of demarcation in the thigh; amputation close to the trochanter, and recovery.

The liabilities of other textures to mortification, as connected

---

* Elementary Forms of Disease. Carswell.

† Milit. Surgery, 1829, p. 187.

with their conditions of texture, range between the two extremes of cellular tissue and artery. But it would scarcely answer any useful purpose to follow up these intermediate liabilities. They are all represented by different degrees only of the same conditions, physical and vascular.

Nor are the various 'organs' exempt. For example, the testicle may mortify. Some years since, I was requested by Mr. Powell (Guilford-street, Russell-square) to see a patient of his who had severe orchitis. The testicle was much enlarged, as usual, the lower half of the scrotum quite black, and its lowest portion had sloughed, leaving an aperture through which the tunica vaginalis bulged, with a few greenish shreds of sloughing cellular tissue on its surface. This shining, greenish membrane appeared not unlike a gangrenous knuckle of intestine, as if a scrotal hernia had been neglected and was protruding. We concurred in opinion that such was not the case, and on the following day the tunica vaginalis had given way, exposing the testis, black as the scrotum. I excised the organ, and having thereby relieved the tension of the scrotum, its mortification ceased spreading; but the man, who had led a hard life in Canada, soon sank comatose and died.

The bladder may mortify. I once saw this in a case of neglected retention of urine of many days' duration. After death the bladder was found enormously distended, reaching nearly to the umbilicus; its texture softened and about to rupture posteriorly, while the mucous membrane had a blackish slate colour.

Strangulated hernia of the bowels, omentum, or both, illustrates gangrene and even sphacelus of these organs; and other abdominal and pelvic viscera, with scarcely an exception, are subject to hernia, strangulation, and mortification. This event is more frequently the result of protrusion through punctured wounds in the abdominal parietes. Swelling around an aperture thus made soon forms a stricture as tight as that of any strangulated hernia, and girts the protruded portion of any viscus, even causing

mortification. In like manner a portion of lung may protrude and mortify. Tulpius has recorded an uncommon case, the particulars of which I have already related. The mortified portion of lung was ligatured and cut off with scissors. It weighed three ounces, yet the patient recovered and enjoyed good health.

Protrusion of the brain—hernia cerebri—through fracture of the skull, is liable to cause mortification of the projecting portion. In one of Mr. Stanley's cases,* the protruded brain gradually lost its natural colour and acquired a light yellow appearance, split into several portions, and a very fetid odour exhaled. Its substance daily became softer, ultimately acquiring almost a semi-fluid state, and in this condition the whole mass gradually wasted. I may add, that fresh granulations arose to fill up the vacancy, and they were manifestly produced from the exposed substance of the brain. The upper part of the tumour had been pared off in the first instance, and its reproduction checked by firm pressure with graduated compressers and a bandage, which being continued when the remaining portion had sloughed away, allowed the opening to close up.

Now this general review of mortification, as it arises in different textures and organs, suggests certain appropriate preventive measures of general application.

Remove any occasion of *pressure;* whether stricture around a protrusion or hernia; tension, by fluids collected, as urine within the bladder, or effused, as blood, lymph, serum, under an unyielding fascia or aponeurosis; tension, by the same fluids effused interstitially within a loose texture; tension, from any textural condition whatever. Thus far physical impediments to a free circulation in the part will be removed. *Vascular* conditions of texture tending to mortification are to be counteracted, by speedily repressing inflammation in the more vascular tissues, and cherishing the circulation in the more avascular.

It is easy to descend from mortification to ulceration. The

* Med.-Chir. Trans., vol. viii., p. 21.

latter is the former by small instalments. Therefore similar conditions of texture predispose to ulceration. Similar *vascular* conditions predispose thereto. Thus, skin and mucous membrane, being vascular textures, are liable to inflammation and ulceration; whereas fibrous textures, being much less vascular, are much less liable. These tissues do not so readily inflame and mortify; they do not so readily inflame and ulcerate. They are not so liable to die in masses, nor to die in molecules. And, apart from inflammation, the comparatively *avascular* textures are prone to ulcerate as well as mortify; by virtue of which condition cellular tissue joins issue with the vascular skin in ulceration. Both ulcerate together in most cases of phagedæna. New or false tissues, as *cicatrix* of the skin, are mostly less vascular than those tissues which they substitute, and are proportionately prone to ulcerate and slough.

The comparative liability of different textures to ulceration and mortification *respectively* is well shown in that process by which a dead limb is gradually separated from the living tissues adjoining; or, more correctly speaking, by which they detach themselves from the dead. When, in the course of sphacelus of the foot, for example, a "line of demarcation" has been formed between the living and dead portions, say above the ankle-joint, how are the different tissues detached down to the bone inclusive? All, excepting the cellular texture and tendons, are separated by ulceration forming a fissure, which progressively extends down to and through the bone. The dead skin, vessels, nerves, muscular tissue, and bone, are evenly detached by ulceration; but the cellular texture and tendons are dead for some distance within the stump, and are irregularly detached by sloughing. Now, the cellular and fibrous tissues are less vascular, and it would appear that this condition disposes them to mortify rather than ulcerate. Therefore, while a comparatively avascular condition disposes tissues to undergo ulceration and mortification, it disposes to mortification *rather* than to ulceration. At least, this conclusion is true of these processes as respectively proceeding from inflammation.

A comparatively avascular condition is virtually established in parts remote from the heart, or by that which has the same effect—venous congestion. The skin of the lower extremities is most liable to ulceration, aided perhaps by a varicose condition of the veins of the leg. Varicose ulcer, near the inner malleolus, is an example of these concurring contingencies.

Preventive principles, similar to those on behalf of mortification, are suggested by the foregoing considerations of textural condition.

The more *vascular* textures being liable to ulceration, inflammation of these textures should be speedily repressed, when, as in most cases, this would be an evil result. It is apt to supervene on inflammation of the skin; and inflammation of the skin around an ulcer will cause ulceration to spread even more rapidly than phagedæna. Textures which are *less vascular,* or *virtually* so by contingent circumstances, should be stimulated to a more free circulation of blood through them if threatened with ulceration. Bed-sores, resulting as they do from constant pressure on portions of the skin, the parts of this texture subjected thereto are deprived of their due supply of blood, and reduced, virtually, to an avascular condition; a free circulation is restored and ulceration prevented by washing the suspicious parts with spirit and water. Chilblains occurring on prominent and exposed parts of the fingers and toes chiefly,—in parts most remote from the heart, in persons also of feeble circulation, and during cold weather—have a congestive character, equivalent to an avascular condition of texture; this may be altered, and ulceration prevented, by friction with a homely lotion—brandy and salt. The common varicose ulcer, immediately dependent on a varicose condition of the internal saphenous vein, has a congestive character equivalent to an avascular condition of texture; but ulceration is far less liable to occur when the leg is bandaged or an elastic stocking worn. Substitute tissues are *absolutely* less vascular than those they replace, and consequently prone to ulcerate. The cicatrix of a former ulcer has this tendency; yet I cannot doubt that I have often succeeded in preventing

ulceration recurring in the brown cicatrix of a varicose ulcer by washing it from time to time with a spirit lotion.

Besides conditions of texture predisposing to mortification and ulceration, I would now advert to another kind of internal cause. The 'nervous system' plays a no less important part in relation to these processes.

Injury of the spine has been followed within twenty-four hours by mortification of the ankle (Brodie).

In other cases, ulceration rather than mortification of a part supervenes on injury of the nerves leading thereto. A preparation in the Museum of St. Bartholomew's Hospital (Ser. 9, No. 9) is thus referred to by Mr. Paget:—A central penetrating ulcer of the cornea formed in consequence of destruction of the trunk of the trigeminal nerve by the pressure of a tumour near the pons varolii. The whole nutrition of the corresponding side of the face became impaired, the patient had repeated attacks of erysipelatous inflammation, bleeding from the nose, and at length destructive inflammation of the coats of the eye, and this ulceration of the cornea. An instance of ulceration and reparation, alternating with the operation and removal of the occasion of pressure on a large nerve, came under Mr. Hilton's observation; and I adduce it as a clenching proof that the death of tissues may arise from defective nervous influence. A man was admitted into Guy's Hospital with fracture at the lower end of the radius; it reunited with an excessive quantity of new bone, and the median nerve suffered compression. Ulceration of the thumb, fore and middle fingers, ensued, which resisted various treatment, and was cured only by so bending the wrist that the muscles on the palmar aspect being relaxed, the pressure on the nerve ceased. *Then* the ulcers healed and remained well; but as soon as the man began to use his hand, pressure on the nerve was renewed, and ulceration of the parts supplied by them returned.

Such cases indicate the *principle* of prevention when pressure on a nerve is the cause in operation, threatening ulceration or

mortification. A sufficiently early detection and removal of the occasion of *pressure* will anticipate these results. If a tumour, its excision ; if an impinging callus, suitable position, and so forth. Circumstances, however, may render any suggestion impracticable in certain cases—as the situation of a tumour, or perchance the difficulty of discovering, during life, the source of pressure. These difficulties were present in the second case I have mentioned. Still the preventive principle derived from pathology remains unaffected by any difficulty, either of diagnosis or of application. How far nervous influence might be restored by electricity when any long-continued pressure on a large nerve has been removed, is a subject worthy of further investigation. It bears on the prevention of local death.

In applying the general etiology of mortification, we should remember the operation of its 'external' causes. They are either physical, chemical, or organic, as animal poisonous matter; and all of them operate either by directly killing the part subjected to their influence, or by exciting gangrenous inflammation therein.

Among causes of a *physical* character—mechanical injury and contusion are apt to occasion *traumatic* gangrene, and in various ways.

Pressure, *severely*, although momentarily, applied to any part, as by a squeeze, *directly* kills it. A finger caught in the hinge of a door may thus be squeezed to death. This mode of death, by sudden and severe contusion, differs from that occasioned by *continued* but less severe pressure, which excites gangrenous inflammation—a matter already considered. In either case, however, mortification is limited to the part injured. *Indirect* contusion or concussion—as by a fall—may occasion mortification more extensively, yet co-extensive only with the part injured. Of this kind, a case is recorded by Sir B. Brodie,* the particulars of which are very instructive. A poor boy, in jumping over a ditch, fell with considerable force upon his feet, causing a compound fracture of the

* Op. cit., p. 315.

leg above the ankle. Although the external wound was trifling, the foot and leg evidently sustained a great shock. Four days after, the limb had undergone mortification as high as the knee, and it seemed to be extending to the thigh. Amputation was performed near the great trochanter. The limb was very carefully dissected. Arteries of the largest size, and their companion veins, were quite pervious. In fact, no injury whatever of these vessels could be discovered; but the cellular membrane, the muscles, and, in short, all the structures, seemed to be more or less disorganized. There were spots of ecchymosis in the large nerves; the periosteum was universally detached from the fibula, and very nearly so from the tibia.

In this case—the contusion having been severe, albeit momentary—mortification, co-extensive with the part injured, was almost immediate.

But, in so far as contusion is *less* severe, and still of short duration, there is proportionately less probability of its exciting gangrenous inflammation, and when it does, it kills the part more *slowly*. Prevention, therefore, under these circumstances, is possible, and also the arrest of gangrene, if it have already commenced. The early relief of tension by puncture or incision, as may be necessary, will effectually accomplish both purposes. Of course, the kind of texture will much affect the result of this preventive measure. For example, a man received a severe blow on his instep; the next day, inflammation had supervened; the day following, a good deal of swelling; and on the third day, sloughing of the skin had commenced. Sir B. Brodie divided it with a lancet, and discovered a large slough of the cellular membrane. The blow having pressed the skin and cellular membrane against the bones of the instep, killed the cellular membrane, but not the skin. In this case, the slough of cellular membrane would have immediately been followed by extensive sloughing of the skin, if it had not been divided freely.

Whether *contusion* be direct or indirect, it chiefly injures the capillary vessels and smaller arteries; traumatic gangrene more

frequently arises from injury to *larger*-sized blood-vessels. Considerable hemorrhage ensues, inducing gangrene, partly by pressure of the blood extravasated among the tissues, partly or principally by cessation of the supply of blood requisite for nutrition. Gangrene is not necessarily *limited* to the immediate seat of injury; it may extend, *e. g.* up a whole limb.

Respecting the prevention of impending (traumatic) gangrene, two principles are indicated.

In so far as tension threatens, it can be removed or relieved by punctures or incisions.

If the primary cause in operation be hemorrhage, persistent or recurring, thereby suspending the local circulation, and depriving the dependent part of a due supply of blood, it is imperatively necessary to secure any bleeding vessel by a ligature above and below the aperture, which also prevents increasing tension by further hemorrhage.

When large-sized arteries are wounded, it frequently happens that large-sized nerves are also injured; and as their lesion alone is sufficient to cause mortification, some influence must be attributed to this cause in many cases of traumatic gangrene.

This analysis of gangrene, denominated *traumatic*, shows that it is not peculiar either in kind or causation; it merely associates together under one title, convenient in a practical point of view, as expressing the mode of origin of various conditions, themselves the 'internal' and immediate (proximate) causes of mortification, and which have been already so considered.

Cold is another external cause, of a physical character, and operates by inducing gangrenous inflammation. It does not appear that a part frostbitten or frozen is irrecoverably dead. A limb may be perfectly insensible and cold as ice, white and transparent like marble, and incapable of being bent without breaking, and yet not absolutely dead. The combs of cocks and ears of rabbits were frozen by Hunter, and afterwards recovered. Leeches and frogs have been placed in the same state, and then restored to life. Such experiments point to a general law, that cold-blooded animals,

and parts of warm-blooded animals, when frozen, are not dead, but asleep as it were, and can be aroused, even as a seed is ready to sprout, awaiting circumstances favourable to life. How long a frozen part or animal may retain its susceptibility of life is uncertain; but this is certain, that unless reaction be *gradual,* the returning flow of blood is apt to become excessive,—to pass into inflammation, speedily ending in gangrene. While, therefore, gradual and moderate elevation of temperature induces salutary reaction and restoration, sudden or immoderate accession of warmth will inevitably excite gangrenous inflammation.

Both these unfavourable circumstances concurred after the battle of Eylau; and their disastrous effect is clearly shown by the record of Baron Larrey. During the three or four exceedingly cold days which preceded this battle (the mercury having then fallen to ten and even fifteen degrees below the zero of Réaumur), and until the second day after the battle, not a soldier complained of any symptom of frost-bite. Nevertheless, they had passed these days, and a great portion of the nights from the 5th of February to the 9th inclusive, in the snow and the most severe frost. The Imperial Guard more particularly had remained upon watch in the snow, hardly moving at all for more than twenty-four hours; yet not one of these men complained of frost-bitten feet. In the night of the 10th of February the temperature suddenly rose, and as high as five degrees above zero. From that moment many soldiers, both of the guard and line, complained of acute pain in the feet, of weight and numbness, with a pricking sensation in the extremities. The parts were scarcely swollen, and of a dull red colour. In some cases, a slight redness was observable around the roots of the toes and on the back of the foot; in others, the toes were black and shrivelled. All the men affirmed that they had not experienced any painful sensation during the intense cold to which they had been exposed by their night-watches on the 5th to the 9th, and that it was not until the night of the 10th, when the temperature suddenly rose eighteen or twenty degrees, that they first felt the effects of the severe frost.

Those who had warmed themselves in the town, or at the fires of the night-watches, suffered the *most* disastrous results.

There is, then, a period, although of uncertain duration, in which a part frostbitten and apparently dead is recoverable. During this period the prevention of gangrenous inflammation is practicable, and the preventive principle suggested by pathology is to solicit reaction in the part, short of inflammation, by the gradual and moderate application of warmth. Friction with something of nearly the same temperature as the frozen part fulfils this indication; and rubbing the part with snow is a practice both surgical and popular.

Heat kills, either at once by charring, or more usually by directly exciting inflammation with sloughing. Unlike cold, therefore, heat may kill immediately and irrecoverably beyond the possibility of prevention; and if not, at least without allowing any opportunity for the effectual employment of preventive measures. Sloughing begins as soon as a sufficient degree of heat has been applied to excite gangrenous inflammation. Any lesser degree falls short of sloughing, and the prevention of this issue need not be considered; while a burn, involving the skin and subcellular texture, is sure to slough. Burns extending deeper are proportionately more gangrenous and more sloughing, until at length a charring degree of heat, killing the part immediately, precludes any intervention.

Besides these physical agents, certain *chemical* compounds have the power of killing immediately, apparently by decomposing the living tissues with which they come in contact. Such are caustics or escharotics. I need not here enumerate the various caustics commonly used in the practice of surgery. Their escharotic action can be circumscribed by appropriate preventive precautions; each escharotic has its antidote. Acids are neutralized by alkalies, caustic potash is rendered inert by the diacetate of lead, and chloride of zinc is decomposed by the bicarbonate of potash. These illustrations will suffice.

Lastly, *animal poisonous matter* introduced into the body may

kill the part by gangrenous inflammation speedily supervening with sloughing, and extensively in some cases if the individual survive long enough. The constitutional disturbance is unlike that arising from ordinary mortification.

Bites of venomous serpents are of this kind. That of the common viper produces the following local and constitutional symptoms:—Immediately after the bite of a viper, an acute burning pain is felt in the part, which very soon twangs to a considerable distance beyond. The cellular texture is speedily engorged with serum, and a tense swelling spreads over the whole limb, as speedily acquiring a mottled livid colour. Blebs of serum arise here and there, and as the general tension is somewhat relieved, the swelling becomes œdematous and much less painful. This doughy swelling is cold, and beset with livid gangrenous spots; the cellular texture sloughs, perhaps extensively, and abscesses form unless anticipated by death.

Pending these local changes—those of acute gangrene—the constitutional symptoms are those of general depression of the nervous system and circulation. Immediately after the bite the heart's action fails, the pulse is small and feeble, and there is great prostration, with cold sweats. A relaxed and vacant expression of countenance marks the unhappy victim. He stumbles about as if in a state of muddling intoxication; or, at any rate, the nervous system shows its sympathy by vertigo, an oppressed respiration, piercing pain about the navel, and vomiting. The liver seems particularly disordered, pure bile being vomited, and in some cases discharged in large quantity from the bowels, while an orange-red colour overspreads the skin. After a time partial reaction is exhibited by a quick, irregular pulse, and the patient may recover; more frequently he sinks without a sign of hope.

The only chance of recovery after a poisoned wound, when through the wound, though itself trivial, a deadly poisonous matter has been introduced, is by withdrawing that matter, or decomposing and neutralizing it, or by preventing its admission into the general circulation. In fact, prevention, rather than an

attempt to cure, is the only sure ground of safety. Excision of the part, or the application of a cupping-glass at an early period, may save by extracting the poison. Escharotics may save by alike destroying the part and the poison in it. A ligature drawn around a limb above the part bitten may save, obviously by preventing the poison passing into the general circulation. Now, of these preventive measures, that of *withdrawing* the poison is at once the most rational, preventive, and practicable. The wound is, perchance, so situated—in the trunk for example—as to preclude the effectual application of a ligature; and if in a limb, its effect is only temporary, lasting only while the ligature is applied, and is also apt to aid gangrene. As a precautionary measure, until other measures more preventive can be applied, the ligature is useful. Caustics likewise are useful adjuncts. Employed alone, however, they involve the destruction of surrounding textures, and to some extent beyond the poison. This dilemma limits the advantage of excision; but extracting the poison *per se* is at once the most rational, advantageously preventive, and withal practicable, measure. Hence, *cupping* a poisoned wound—*e. g.*, the bite of a viper—is eminently preventive of gangrene, and, moreover, of the hazardous constitutional disturbance it induces. Cupping is preventive, partly by withdrawing the poison, and partly by depriving the vessels of their absorbent power—a disability which, if the cupping-glass be applied for half an hour, continues for an hour or two after the removal of the glass.* A ligature above the wound will aid this result, and, by limiting the effusion of serum and swelling, will also tend to *arrest* the progress of gangrene, which spreads chiefly through the cellular texture.

With this I conclude the history of mortification, with its constitutional disturbance, and regarded from a preventive point of view. I have entered into detail, somewhat at length, on this

---

* See Experimental Researches on the Influence of Atmospheric Pressure on the Blood in the Veins, &c., and on the Prevention and Cure of the Symptoms caused by the Bites of Rabid or Venomous Animals, 1826. Sir D. Barry.

subject, because of its great importance—the preservation of life probably, and certainly of a member or texture which would otherwise perish, and which, being lost, is never restored, or replaced only by (substitute) texture of inferior quality and imperfect use in the animal economy.

Various Blood-diseases arise from other perversions of Nutrition, besides that of Inflammation; also from those of the Digestive Process, Excretion, and Respiration, respectively.

The Prevention of these Diseases.

On reviewing the pathology of inflammation, we perceive that it is essentially a perversion of the process of nutrition, affecting some part of the body, and that such perversion engenders a 'blood-disease,' as the constitutional morbid condition consequent on this local one. The blood undergoes certain alterations in respect of its chemical composition and properties.

This blood-disease is not the only one consequent on diseases of nutrition. Other perversions of this process engender each its own peculiar blood-disease; and *these* constitutional morbid conditions are also of grave importance.

They severally arise, by the *addition* of something noxious to the blood, through absorption from the textures severally concerned in their production. Associated with this mode of origin, it should be observed that the same blood-conditions may be induced by various disorders of the process of digestion, in which case also something noxious is added to the blood.

Herein blood-pathology and physiology are analogous. In health, the blood's composition is ever changing by the addition of new matter, received through the process of digestion—the 'primary assimilation' of food, and also, as effete matter, the waste of the textures through their destruction in the course of 'secondary assimilation' or nutrition. So, likewise, in respect of various diseases, the blood's composition is ever changing, by 'primary *mal*-assimilation,' or by 'secondary *mal*-assimilation,' or

by co-operation of *both* these perversions—the former representing that of Digestion, the latter that of Nutrition. The products accruing from either source appear in the *urine,* and can be selected for examination by observing a very simple precaution with regard to the sample of urine, the importance of which I have elsewhere urged.*

Urine secreted at from three to six hours after a meal presents the products of primary assimilation, while that secreted several hours subsequently, when the urine from this source has run off, presents the products of secondary assimilation, or the *débris* of the textures. The latter may be denominated *urine of the blood,* and if examined in the morning before breakfast, and after an interval of fasting from overnight, will be found to contain, unlike the *urine* of *digestion,* the waste of the textures. To make this observation complete, the bladder should be emptied overnight, to preclude any admixture of the urine then in the bladder with that which is secreted during the night. By this simple precaution, the products from these two sources of urine can be detected and distinguished in most cases with certainty.

This twofold source of blood-disease was first investigated by Prout,† and afterwards, physiologically, by Liebig,‡ who designated the destructive stage of secondary assimilation 'destructive metamorphosis' of tissues. Prout was inclined to believe that in all cases of secondary mal-assimilation, the *formative* and *destructive* stages of this process are both perverted in a greater or less degree.

The various kinds of textures, as chemically distinguished,—the albuminous, the gelatinous, the oleaginous, &c.,—may severally generate morbid conditions of the blood by their *destructive* metamorphoses in secondary *mal*-assimilation.

Albuminous tissues pass into *lithic acid,* rather than lithate of

---

* The Irritable Bladder: its Causes and Curative Treatment.

† Nature and Treatment of Stomach and Renal Diseases.

‡ Animal Chemistry. Trans. by Gregory, 1842.

ammonia; and the blood, being thence surcharged with this acid, represents a gouty diathesis.

Textures abounding in *phosphoric acid,—i.e.,* the brain and nervous system,—by their destructive metamorphosis, in an over-active degree, induce the phosphatic diathesis. The blood is surcharged with *phosphoric acid,* in combination with alkaline and earthy bases, forming respectively phosphates of soda, and those of lime and magnesia.

One general law appears to govern the evolution of phosphoric acid and the formation of phosphates. They always follow *nervous exhaustion,* particularly that resulting from over brain-work. Of this mode of origin two excellent examples are given by Golding Bird.* Other pathological conditions have their influence, and the observations of Dr. Bence Jones† respecting abnormal quantities of phosphates in the *urine* are here to the point :—

"That neither the earthy nor the alkaline phosphates are permanently increased in spinal diseases.

"In fevers and acute inflammations of fibrous, muscular, or cartilaginous tissues, the total amount of earthy and alkaline phosphates is not increased.

"In chronic diseases, where the nervous tissue is unaffected, no deduction can be drawn.

"Chronic cases of mania, melancholia, and general paralysis of the insane, give no marked results.

"In chronic diseases of the brain, and in chronic and even acute disease of the membranes, there is no increase in the total amount of earthy and alkaline phosphates.

"In fractures of the skull, when any inflammation of the brain supervenes, there is an increase of the total amount of phosphates. When no head symptoms are present, no increase of the phosphates is observed, even when other acute inflammations supervene.

---

* Urinary Deposits, ed. 4, 1853; also, The Irritable Bladder, by the Author.

† Animal Chemistry, in its applications to Stomach and Renal Diseases, 1850, p. 87.

"In acute inflammation of the brain there is an excessive amount of phosphates in the urine. When the inflammation becomes chronic no excess of phosphates can be shown by the method of analysis that was employed.

"In some functional diseases of the brain an excessive amount of phosphates is observable: it ceases with delirium. Delirium tremens shows a remarkable deficiency in the amount of phosphates excreted, provided no food is taken. If food be taken this diminution is not apparent.

"The variations of the *earthy* phosphates are so dependent on the earthy matter—lime and magnesia—present in the urine, that no deduction from them as to the nature or seat of the disease is possible."*

The gelatinous textures may possibly, according to Prout, by their destructive metamorphosis, be converted into either *oxalic acid* or *sugar;* and these products, entering the blood, would constitute respectively the oxalic-acid diathesis, and the source of diabetes (mellitus).

'Primary *mal*-assimilations'—perversions of the digestive process—generate morbid conditions of the blood, similar to those which emanate from perversions in the course of nutrition.

Perversions of the digestive process are referred by Prout to the stomach, the duodenum, and the chyliferous system.

Lithic acid may be a product of the imperfect digestion of albuminous food in the stomach. Mal-assimilation in the duodenum generally results from that in the stomach, and that which takes place in the chyliferous system of vessels may form the pseudo-albuminous matter of struma.

From this triple source the blood becomes contaminated, unless the product of mal-assimilation in the stomach and duodenum is evacuated as fæcal, ill-digested food.

An excess of *phosphates* in the blood will result from an undue proportion of food containing them. A vegetable diet has

* For cases illustrating the above observations, see Lancet, 1847.

this effect, of which the experimental observations of Dr. Bence Jones* afford ample proof. Alkalies, taken continuously, favour the production of phosphates, by supplying the *base* with which phosphoric acid may combine.

Oxalic acid is occasionally introduced by particular kinds of food—*e.g.*, common rhubarb, sorrel, &c.; and then, if *hard* water, which contains lime, be drank, oxalate of lime is formed in the blood. This acid is more frequently generated by mal-assimilation of saccharine matters during the digestive process, as was first suggested by Prout.

He also first suggested the possibility of *sugar* being a product of the primary *mal*-assimilation of farinaceous matters, and thence the origin of diabetes mellitus. It is well known that starch and gum are converted into sugar by the action of acids, and equally indisputable that the stomach is prone to acidity in diabetes,—a clinical fact which accords with the view advanced by Prout. But the actual production of sugar during digestion in diabetes has been established by M'Gregor.†

Claude Bernard has since apparently demonstrated the constant production of sugar, alike from azotised and unazotised matters, by the liver (in health), and therefore the probability that this organ is at least one source of sugar in diabetes, by over-activity of its function, in respect of sugar-production. I shall have occasion to revert to this theory, in connexion with the agency of respiration.

Certain it is that sugar abounds in the blood of diabetic patients, as shown by the analyses of Maitland and Ambrosiani.

Oleaginous matters are probably generated freely by primary mal-assimilation in persons who have a marked tendency to obesity, and thence the blood becomes surcharged with fat. This excess not being relieved by secondary assimilation, it accumulates in the body, encompassing and invading the textures interstitially; until at length, usurping and occupying the place of their proper

---

* Animal Chemistry, &c. † Medical Gazette, 1837.

structural elements, it constitutes 'degeneration' by the substitution of fat; yet the blood remains surcharged from its original source of continued production.

Now, all such morbid conditions of the blood, whether resulting from primary or secondary mal-assimilation, are declared by certain corresponding conditions of the *urine*, which may therefore be accepted as 'signs' of the blood-diseases respectively from whence they proceed. But these morbid states of the urine are trustworthy signs only when rightly interpreted. Lithic-acid and phosphoric-acid urine, more especially, are indications of correspondingly different conditions of the blood, subject to certain provisions and restrictions requiring explanation.

Lithic acid is eliminated from the blood by the secretion of urine in combination with some base—soda, or more commonly ammonia—forming lithates of soda and ammonia. These salts, readily soluble in urine of the temperature of the body, are precipitated only when present in excess, proportionately to the aqueous portion of the urine eliminated, and as the supersaturated solution cools. They then appear as yellowish or red brick-dust deposits. Lithic acid may be liberated by decomposition of these salts, but it occurs only when *some other acid* is present in *excess* to replace the lithic acid in combination. Free lithic acid, being insoluble, is then precipitated, and appears as a *deposit* of 'red sand' in the urine, which may be further identified by microscopic examination. This deposit consists of minute crystals, having various shapes, of which rhomboidal crystals are the most common (p. 68, fig. 22).

Respecting the kind of acid by which uric acid is thus deposited, Parkes observes:* "It does not follow that the acidity should be owing to any single acid; it is owing either to an augmentation of all the usual acids of the urine—the sulphuric, phosphoric, and perhaps the hippuric, the lactic, and the carbonic—so that the bases are insufficient to neutralize them,—or to the

* Composition of the Urine, in Health and Disease, and under the action of Remedies, 1860, p. 218.

formation of acids after emission of the urine—viz., probably the lactic, acetic, butyric, or oxalic.

"It is of course possible that the deposit of uric acid may be owing, not to excess of other acids, but to absolute deficiency of alkali: no facts have yet been discovered on this point.

"The two causes of increased acidity of the urine—excess of normal acids, and formation of acid after emission—are sometimes in simultaneous action. The urine depositing lithic acid without lithates is not usually the high-coloured, red, pigment-loaded urine, but yellow and transparent; the acid is deposited slowly, and without admixture with lithates. Uroxanthin (indican) is often present in large quantity; and, as Virchow suggests, it may more rapidly form acid than common pigment."

The practical issue of all these considerations is this—that to rightly estimate the value of 'lithic-acid urine,' as the sign of a corresponding 'morbid blood-condition,' it is necessary to discover the *total* amount of lithic-acid excreted from time to time; and for this purpose we cannot trust any *deposit* thereof, either combined or free. The lithates may be in excess, *short* of a supersaturated solution; and lithic acid itself becomes apparent *only* when the urine is hyperacid from other causes.

The total amount of lithic acid excreted can be discovered by a simple experiment, devised by Golding Bird. Let all the urine passed in twenty-four hours be collected, well shaken, and a given quantity—say about two ounces—be mixed in a conical glass vessel with about half a drachm of hydrochloric acid. In six or eight hours crystals of uric acid are copiously deposited on the sides of the glass. To ensure their complete separation, they should be allowed to repose for twenty-four hours, and may then be washed, dried, and weighed.* Simple multiplication shows the whole amount of uric acid secreted in the twenty-four hours, without the chance of any considerable error. In estimating the pathological importance

---

* Urinary Deposits, ed. 4, 1853. See also Animal Chemistry, &c., by Bence Jones, 1850, p. 53.

of the result thus obtained, the healthy standard of quantity should be remembered; and this ranges from six to ten grains of uric acid in twenty-four hours.

The diagnostic value of 'phosphatic urine,' as ordinarily measured by the amount of phosphates *deposited* in the urine, is also erroneous. Phosphatic urine, in this sense, is only an *appearance,* not a true measure of the whole amount of phosphates present, and of the accompanying 'blood-condition.'

The pathological significance of phosphatic deposits in the urine has been investigated more particularly by Dr. Bence Jones.

Of *all* the phosphates *present,* not necessarily deposited, in the urine, those of soda are most abundant; equalling in amount three or four parts of the whole. The earthy phosphates of lime and magnesia represent the remainder. Phosphatic urine, therefore, should rather, of the two, have reference to the former salts. But the phosphates of soda are so very soluble in water and in acid or alkaline urine, that they are never deposited. In this respect resembling the sulphates of potash or soda, any excess of these phosphates remains concealed. On the contrary, the phosphates of lime and magnesia are scarcely soluble in water, and nearly insoluble in alkalies, although very soluble in acids, even in acid phosphate of soda. Therefore, whenever the urine becomes alkaline, down go the phosphates of lime and magnesia. This precipitate, however, denotes only the quantity of lime and magnesia drawn from the blood, and now appearing in the urine. The *major* portion of *phosphoric acid,* being combined with soda, remains unobserved.

By taking more lime or magnesia in the food, or by adding these bases to the urine, we increase the amount of earthy phosphates; and by a sufficiency we precipitate *all* the phosphoric acid in combination with *them,* thus leaving no *phosphate* of soda in solution. Conversely, if we could abstract all the lime and magnesia, no precipitate would appear by adding alkalies,—in which, as well as acids, phosphate of soda is soluble,—though there remained a great excess of phosphate of soda concealed in solution.

But if lime and magnesia are present, as usual, in the urine, a portion of the phosphoric acid appears in combination with them, forming a deposit of *these* phosphatic salts whenever the urine becomes *alkaline*, in which they are insoluble. Hence the more appropriate name, *alkaline* urine, suggested by Dr. Bence Jones, rather than phosphatic urine, as ordinarily understood, which represents merely the amount of lime and magnesia present in combination with phosphoric acid. If regarded from this latter point of view, the term 'phosphoric diathesis' should be extended to denote an increase in the *total* amount of phosphates, *alkaline* and earthy; or, if limited to one phosphate, it ought to denote *alkaline* phosphate, it being proportionately four or five times more abundant than earthy phosphates in the urine: moreover, the term earthy diathesis (as indicated by the urine), if used at all, ought to signify urine which really contains an *excess* of lime and magnesia, and not the precipitation (it may be of only a small quantity) of these earthy salts, the urine having lost its healthy property of retaining them in solution on becoming alkaline.

In short, alkalescence of the urine and increase in the *total* amount of phosphates have no relation of any kind to each other. They are quite distinct, and, indeed, rather opposite states.

Supposing *alkalinity* of the urine to be due to *fixed* alkali—as potash or soda—the phosphate of lime and the phosphate of magnesia are immediately deposited, and appear as a 'white sand.' Submitted to microscopic examination, these salts are seen to consist of amorphous particles, or small round globules (p. 68, fig. 25), and, occasionally only, prismatic crystals with oblique or dihedral summits. According to Hassall's observations,* crystallized phosphate of lime is common, much more so indeed than amorphous phosphate, which he regards as unusual.

This white deposit and coexisting alkaline condition of the urine occur whenever an excess of fixed alkali, or, what is equivalent, a deficient proportion of acid, is taken in the food. The

---

* Lancet, 1850, vol. i., and Med.-Chir. Trans., vol. xxxvi.

urine is *secreted* alkaline, and deposits its earthy phosphates in greater or less abundance, according to the quantity of lime and magnesia present.

Not to be misled by an alkaline condition of the urine from *fixed* alkali, it is most important to know and remember that the acidity of *healthy* urine varies considerably during the diurnal period. According to the observations of Dr. Bence Jones, confirmed by those of Dr. W. Roberts, it is *inversely* to the acidity of the stomach. During digestion, when some acid, probably the hydrochloric, is being secreted by the stomach, an equivalent amount of soda or potash, previously in combination, must remain as free alkali in the blood, rendering it proportionately more and more alkaline. Accordingly, the urine becomes less and less acid, and perhaps eventually decidedly alkaline. When acid ceases to flow into the stomach, and any superfluous portion which had been secreted is reabsorbed, the blood regains its former *average* degree of alkalescence; the urine also is secreted less and less alkalescent, and, becoming acid, its acidity rises until the next meal, when the highest degree of acidity is attained. If no food be taken, this condition of urine remains stationary for about twelve hours; immediately after a meal, its acidity again falls, and gradually approaches an alkaline reaction.* Examined at such time, alkalinity of urine might inadvertently be regarded as a morbid condition; but examination of another and another sample excreted some time after a meal, when the process of digestion is completed, corrects this suspicion; the urine thenceforth is found more and more acid prior to the next meal, when the alkaline retrogression supervenes.

This alternation of digestion, with approaching alkalescence of urine, and restoration of acidity on completion of digestion, invalidates the result of *any one* examination of the urine. A *mixed*

---

* See adverse observations by Dr. Julius Vogel; A Guide to the Analysis of the Urine, by C. Neubauer and J. Vogel, ed. 4. Translated for New Syd. Soc., by W. A. Markham, 1863, p. 296.

sample of the whole amount of urine excreted during the twenty-four hours will give its average condition.

If *volatile* alkali—as carbonate of ammonia—be the occasion of alkaline urine, then the *deposit* consists of the ammoniaco-magnesian phosphate, together with some phosphate of lime; the former appearing in the form of transparent prismatic crystals, or of foliaceous, penniform, or stellar crystals (p. 68, fig. 25). The two latter are phosphate of lime. (Hassall.*)

This deposit occurs whenever phosphates are deposited in connexion with an inflamed state of the mucous membrane of the bladder, the ammonia being supplied by the decomposition of urea, which constituent of the urine may be regarded as carbonate of ammonia, plus two atoms of water. Whether urine is ever *secreted* ammoniacal appears doubtful; without doubt, however, it may become so, after emission, by decomposition of the urea. This source of the ammonia present in the phosphate then formed was first clearly perceived by Lecanu, and has since been explicitly pointed out by Owen Rees. The decomposition of urea is effected apparently by the mucus acting as a ferment, which is always more freely secreted by an inflamed mucous membrane, as in pyelitis and cystitis. Ammoniaco-magnesian phosphate, consisting of its characteristic crystals, is abundantly deposited; and equally so, a white tenacious substance, that can be drawn into skeins of considerable length. It is said to consist of pus-globules, they having become adherent under the action of ammoniacal urine. Thence the phosphate above mentioned, together with granules of phosphate of lime, are involved in one gelatinous mass. This mixed deposit is frequently witnessed in cases of paralysis affecting the bladder, which then assumes a condition bordering on inflammation.

Now, *all* the phosphates are dissolved by acids—unlike coagulated albumen; and remain unaffected by heat—unlike the lithates; but the following points of contrast distinguish the two kinds of alkaline urine—the ammoniacal from that caused by fixed alkali.

---

* Lancet, 1850, vol. i.

Ammoniacal urine effects no change in blue litmus paper until it dries, when the pink colour immediately appears.* Urine becoming alkaline during digestion, *i.e.* from *fixed* alkali, turns pink paper blue, which remains so when dry. *Ammoniacal* urine deposits crystals of phosphate of ammonia and magnesia, while urine otherwise alkaline, from *fixed* alkali, deposits an amorphous powder of phosphate of lime. The former deposit is associated with mucus and pus; the latter with mucus only, and rarely in great quantity. *Ammoniacal* urine is constantly alkaline; that from *fixed* alkali is only occasionally alkaline, *i.e.* at particular periods of the day. *Ammoniacal* urine is a sign of local disease—inflammation of the urinary mucous membrane; whereas alkalescence from *fixed* alkali is a sign of a more general disorder, *i.e.* indigestion.

Guided by these characters, we can detect and discriminate the *kind* of alkali present in the urine, and its *source*. Yet such diagnosis, founded on the kind, and even the amount, of phosphatic salts *deposited* by the urine, signifies nothing concerning the *total* amount of phosphates *excreted from the blood*. The non-appearance of phosphates does not imply their absence, and their appearance is no measure of the total amount present. The question, therefore, an all-important one, presses, whether an *excess* of phosphates is accumulating in the 'blood,' consequent on some perversion of nutrition or digestion, and constituting the true 'phosphoric diathesis'? Mere inspection of the urine may disclose nothing respecting this constitutional morbid condition; and should the urine be alkaline, we then discover merely the amount of earthy bases—lime and magnesia excreted in combination with phosphoric acid, and deposited. But this deposit of phosphatic salts contains only part of the whole phosphoric acid present. By far the greater portion remains concealed in the soluble phosphate of soda, which is never spontaneously precipitated under any circumstances.

To determine the whole amount of phosphoric acid eliminated,

* See Trans. of the Chemical Society, vol. ii., p. 244, communication by Bence Jones.

it is necessary to ascertain the amount of this alkaline phosphate, as well as that of the earthy phosphates. Both together represent the phosphoric diathesis.

For this purpose the following experimental process is recommended by Bence Jones:—About 1000 grains of urine are to be weighed, and the earthy phosphates precipitated by pure ammonia, free from carbonate. These should be filtered, washed with ammoniacal water, and heated to redness; adding at last a drop or two of nitric acid. The amount of earthy phosphates is determined by weighing the residue. The alkaline phosphates are estimated by taking about 500 grains of urine, adding an excess of chloride of calcium, and then pure ammonia. Thus all the phosphoric acid is precipitated as phosphate of lime. This is to be filtered, well washed, and the filter and the precipitate burnt with a drop or two of nitric acid. If the filtration has been slow, it is necessary to redissolve the residue in a platinum crucible by hydrochloric acid, and to reprecipitate by pure ammonia, when the filtration will take place very rapidly. After being burnt, the crucible is weighed, and by deducting the previously determined earthy phosphates, the difference may be taken as the amount of alkaline phosphate.*

'Oxalate of lime in the urine' is of very frequent occurrence; so much so, that its presence cannot be regarded as signifying a 'morbid blood-condition,' excepting in respect of the quantity secreted. A few minute crystals are quite compatible with health; while large crystals in large quantity, and *persisting* for a considerable period, indicate such morbid condition.

This salt can be readily detected in the urine, and recognised. It is scarcely ever deposited, and then only as minute, colourless, transparent, hempseed concretions; it nearly always remains *diffused* in the urine, yet readily discovered by the microscope, provided only sufficient time has elapsed after emission of the urine subjected to examination. Not less than twenty-four hours

---

* See Phil. Trans., 1845, p. 365.

are necessary—according to Bence Jones—to insure the formation of crystals, if oxalate of lime be present. The crystals then discovered are very characteristic. They occur in three forms: most commonly as octahedra, first observed by Golding Bird, or as hour-glass or dumb-bell shaped crystals; and occasionally in the shape of small, red blood-globules, which are probably the earliest stage of dumb-bell oxalate (p. 68, fig. 24).

The urine itself is free from deposit, although usually clouded with an abundance of epithelium; and its colour is a fine amber hue, somewhat resembling the bright golden sherry colour of lithic acid urine, contrasting with the pale whey-like urine of earthy phosphates, or the turbid orange brown of ammoniacal urine, which, moreover, exhibits an iridescent pellicle on its surface, and is ropy and fetid.

The following observations by Golding Bird, respecting the composition of oxalic acid urine, relate to the circumstances under which it occurs.

In rather more than one-third of the cases examined, uric acid or urates existed in large excess, forming the greater bulk of the existing deposit. In all there existed a greater proportion of urea than in healthy urine of the same density; and in nearly 30 per cent. of the cases, so large a quantity of urea was present, that the fluid crystallized into a solid mass by adding nitric acid. The urate of ammonia found in the deposits of oxalic-acid urine is occasionally tinted with a pink hue. An excess of phosphate frequently accompanies the oxalate. The presence of sugar in the specimens examined was exceptional.

Prout regarded the oxalic-acid diathesis as a substitute for that of lithic acid, the former being preceded and followed by the latter. Liebig demonstrated the intimate relation of lithic acid to urea and oxalate of lime; the two latter having been formed artificially from the former; and this conversion of lithic acid was shown by Wöhler's experiments to take place in the bodies of animals.

Oxalic-acid urine—properly so called from the quantity of this

acid excreted—is therefore an expression of *many* morbid conditions. Taking patients indiscriminately in an hospital, Bence Jones concludes that oxalate of lime is notably present in the urine in nearly one out of three. Diseases of many kinds, and of opposite characters, are apparently conducive to this result: indigestion, especially if attended with flatulence, and in cases also where no indigestion was ever experienced; skin diseases, and in cases where the skin was never affected; in acute rheumatism, acute gout, fever, and in diseases of women and children.

'Sugar in the urine' is the expression of another 'morbid blood-condition,' resulting either from primary or secondary mal-assimilation, or from both combined in operation, and possibly also due to the liver. Saccharine matter is occasionally present, as a mere trace, in healthy urine; but any more obvious quantity, and *persisting*, is abnormal. The constant presence of sugar in any notable quantity, rather than its absolute amount, is the diagnostic sign of consequence to the practitioner, and therefore to detect the presence of sugar is the object he has in view.

Diabetic sugar is that of the grape; it is excreted in quantities from 1 lb. to 2 lbs. or more during the twenty-four hours, so that in a few months a patient may pass more than his own weight of sugar. Of course urine, thus impregnated, has a sweet smell and taste, and a high specific gravity, averaging 1040. Owing to the diuretic property of sugar, urine is secreted in enormous quantities, as much as one hundred ounces, two hundred ounces, four hundred ounces, or more being passed in twenty-four hours. This sugar readily crystallizes from the urine, and appears as an efflorescence on whatever clothing, &c., any of it chances to dry. The urine itself is clear, of a pale straw or greenish tint, and contains usually rather more than less of the ordinary constituents of urine. But the *continued* secretion of sugar in notable quantity is the grand characteristic of diabetic urine.

To detect this morbid condition in its infancy, certain tests, more delicate even than the production of crystals, can be applied with signal success.

The *fermentation* test is easily applied. Add a small quantity of yeast to some of the suspected urine in a saucer; invert a test-tube filled with this mixture, and stand it in the saucer; then place the whole in a warm room. If sugar be present, fermentation soon begins, and bubbles of carbonic acid rising in the tube accumulate and depress the fluid. Minute fungoid growths also are developed, which can be seen with the aid of the microscope. Another fungus—*penicilium glaucum*—the mildew that overspreads decaying vegetable or animal matter, and which may appear in *non*-saccharine urine, is apt to be mistaken for this 'yeast-plant'—*torula cerevisiæ*. Their distinctive microscopic characters have been depicted by Dr. Hassall.*

Certain *chemical* tests are more conclusive. They all depend on the facility with which the composition of diabetic sugar is changed; and this can be readily effected by salts of copper, and by alkalies.† Full directions for the successful application of these tests are given in Golding Bird's work;‡ the following particulars, however, are essential to our purpose—the early and exact detection and discrimination of diabetic urine:—

Trommer's test:—Add to the suspected urine in a large test-tube just enough of a solution of sulphate of copper to communicate a faint blue tint. A slight deposit of phosphate of copper generally falls. Liquor potassæ must then be added in great excess; a precipitate of hydrated oxide of copper first falls, which redissolves in the excess of alkali, if sugar be present, forming a blue solution like ammoniuret of copper. On gently heating the mixture to ebullition, a deposit of red suboxide of copper falls if sugar be present.

Capezzuoli's test:—Add a few grains of blue hydrated oxide of copper to urine in a conical glass vessel, and render the whole alkaline by adding liquor potassæ. If sugar be present, the fluid

---

* The Urine in Health and Disease, 1863, pp. 149–151.

† See Med.-Chir. Review, January, 1853. Lionel Beale.

‡ Urinary Deposits.

assumes a reddish colour, and in a few hours the edge of the oxide deposit acquires a yellow colour, which gradually extends through the mass, owing to the reduction of the oxide to a metallic state (suboxide?)

Moore's test:—Place in a test-tube about two drachms of the suspected urine, and add nearly half its bulk of liquor potassæ. Heat the whole over a spirit-lamp, and allow active ebullition to continue for a minute or two; the previously pale urine will become of an orange-brown or even bistre tint, according to the proportion of sugar present. The subsequent addition of an acid generally causes the evolution of an odour of boiling molasses. Should the liquor potassæ contain lead, a dark colour is produced by the sulphur in the urinary excretion acting on it, which might lead one to suspect the presence of sugar when none exists—a source of error first pointed out by Owen Rees. Hence it is important to preserve the test-solution in bottles of green glass free from lead.

Diabetes *mellitus* is the expression sometimes used for that disease whereof sugar in the urine is the sign; and this attributive title seems necessary to distinguish ordinary diabetes from a variety contra-designated Diabetes *insipidus*. In the former, farinaceous matters are probably converted into dextrin, and thence at once into grape-sugar; but there is some reason to believe that this succession of metamorphoses may be interrupted, and that an *insipid* sugar is then formed intermediately between dextrin and sugar of milk (Bence Jones). This tasteless sugar resembles sugar of milk, differing from it in not giving rise to mucic acid and in undergoing fermentation. It can be converted into grape-sugar by the action of acids.

Diabetes insipidus has received other names—Polydipsia (Becquerel), Diuresis and Hydruria (Willis). The secretion thus signified, of a large quantity of *watery* urine, is probably a distinct disease. Aqueous diabetes commonly occurs in connexion with hysteria; and the term *hydruria* distinguishes it from azoturia, of which disease an excessive excretion of *urea* is the prevailing characteristic. The absolute amount of urea excreted in the

twenty-four hours may be increased in hydruria; no sample, however, of such urine contains its normal proportion to the water secreted. Prout restricts the term azoturia to the possible excretion of a great excess of urea, while the water and other urinary constituents are not increased. This is a rare disease, although the urea is increased in pyrexia.

All the foregoing conditions of urine have reference to those blood-diseases which result either from perversions of nutrition, or of the functions of the digestive organs, or from both; and which manifest themselves by these morbid states of the urinary excretion, they being in their turn signs of the existing blood-disease. The kidneys are merely the channels through which the noxious matter, whatever it be, is excreted from the blood.

But the *kidneys* themselves may be at fault; they are liable to undergo certain structural changes, whereby some one (or more) of the urinary constituents ordinarily excreted is retained in the blood, and thus adding a noxious matter thereto, gives rise to its own peculiar blood-disease. In any such case, also, the morbid state of the urinary excretion is the sign of that structural change which the kidneys are undergoing or have already undergone. These organs, and the blood-disease induced, stand in the relation of cause and effect, while the peculiar state of the urine is the sign of the particular cause in operation. I allude chiefly to congestion of the kidneys causing suppression of urine, and to their fatty and granular degeneration—Bright's disease, originating a certain blood-disease by the retention of urea and excretion of albumen, as manifested by corresponding changes in the urine secreted—viz., the absence or diminished proportion of urea, and the presence of albumen in variable quantity.

The persistence of this morbid state of the urine is the visible sign of one or other of the structural changes alluded to, and its absence is almost conclusive proof that no such structural disease has taken place. In other words, Bright's disease is, with rare exceptions, *invariably* accompanied with albuminous urine; and this condition of urine *persisting* is a sure sign of Bright's disease, and of no other.

It therefore behoves us to inquire more particularly into the composition and characters of albuminous urine, and learn how it can be early and exactly detected and discriminated.

Respecting the altered composition of such urine, two facts are of leading importance,—the constant draining away of albumen, as if it were excrementitious, whereas the proportions of urea and water are both diminished. These changes coincide with the early stage of Bright's disease. The urine is scanty, owing to the diminished proportion of water, and has a smoky-brown colour; is of low specific gravity, averaging 1014—by abstraction of the urea, and easily froths—owing to the presence of albumen. Subsequently, it becomes more abundant, even approaching to diuresis, is pale and opalescent, and contains far less albumen; but its specific gravity declines more and more, down perhaps to 1004. The solid urinary constituents, amounting in health to about 68 parts in 1000 of urine, in Bright's disease decline to 14, 12, or 6 parts in 1000.

All these changes are reflected in the blood. Analysis discovers a large accumulation of water — tending to dropsical effusions, the retention of urea, and a diminished proportion of albumen, together with a rapid decrease of the red particles of the blood. "I am acquainted," writes Dr. Christison, "with no natural disease, at least of a chronic nature, which so closely approaches hemorrhage in its power of impoverishing the red particles of the blood."

No other very marked changes occur. Subjoined are the results of Franz Simon's observations, as quoted by Owen Rees.*

BLOOD.

	Water.	Fibrin.	Corpuscles.	Solids of Serum.
Health . .	775·7	3·8	137·1	83·4
Albuminuria	808·3	3·0	133·9	54·8 (1)
	859·2	8·2	75·5	57·2 (2)
	855·5	4·5	42·7	97·3 (3)

* Nature and Treatment of Diseases of the Kidney, connected with Albuminous Urine—Morbus Brightii, 1850.

(1.) A man aged 55.—First stage of granulation, anasarca: urea in blood.

(2.) A man aged 44.—First stage, more advanced than (1) anasarca, *pneumonia:* urea in blood.

(3.) A man aged 23.—Advanced granulation, after scarlatina.

It would appear that the proportion of fibrin scarcely varies from that of health, unless acute inflammation supervenes and complicates the original disorder, as in Case 2.

The presence of albumen in the urine is easily discovered and readily distinguished, provided only certain precautions be observed in making the examination. They relate either to the chemical composition of the urine submitted to examination, or to the tests employed; chiefly, these precautions have reference to the urine itself.

Albuminous urine is not merely a solution of albumen. So far as it *alone* is concerned, by applying heat to such urine, the albuminous portion—white of egg—begins to coagulate at 160° Fahr. and gradually solidifies as the temperature rises to 212°. But this urine contains other ingredients, and their variations in quantity interfere with the coagulation of the albumen.

Thus, if the urine be alkaline, or even neutral,—whether from the presence of volatile alkali, carbonate of ammonia, or from fixed alkali, as soda,—either alkali will combine with albumen, and neither of the resulting compounds being coagulable by heat, the urine remains clear when heat is applied. The albumen is not discovered, although perhaps abundant. Or again, if an opposite condition exists—should the urine be over-acid, from the presence of a free acid, as the acetic or hydrochloric, *it* will combine with albumen, and the acetate and hydrochlorate (of albumen) being uncoagulable by heat, the urine remains clear when heated. The albumen is concealed.

Supposing, however, that, on the application of heat, a white flaky precipitate does fall, resembling albumen, it may not be albumen. Earthy phosphates are likewise precipitated by heat. To distinguish between these two deposits—phosphates and coagu-

lated albumen, as well as to evolve albumen concealed by an alkaline or over-acid state of the urine,—heat having been applied, nitric acid (strong) should then be dropped into the test-tube containing supposed albuminous urine. If the deposit be phosphates, they are redissolved; if albumen, it is more firmly coagulated.

Nitric acid unaided will precipitate albumen, but it also liberates lithic acid from the lithates, and combines with urea; when, therefore, either of these constituents is present in excess, a brown deposit of lithic acid or nitrate of urea forms, and disguises the albumen. Both precipitates, however, together with the lithates, are redissolved by heat, which, on the contrary, discloses albumen.

In short, *heat* clears off any difficulty arising from lithic acid, the lithates, and urea; *nitric acid* clears off any difficulty arising from the (earthy) phosphates, at the same time liberating and evolving albumen from any prevailing alkaline or mineral acid condition.

Nitric acid, in respect of its behaviour to albumen, disputes with heat the privilege of disclosing the presence of this abnormal constituent of urine. Nitric acid unites with albumen, forming what may be called nitrate of albumen, which is not coagulable by heat. Consequently, if only just so much acid be added to albuminous urine as shall combine with all the albumen present, and form this nitrate, none of the albumen will appear when heated. Nitrate of albumen being *insoluble* in nitric acid, appears when *more* acid is added; but is again redissolved on the addition of an *excess* of acid. The happy *medium* quantity of acid is necessary to exhibit albumen,—not just an equivalent, which combining with the whole amount present, renders it insoluble, although heated; this would be too small a proportion of acid; while an excess—above that proportion in which the nitrate of albumen is insoluble—redissolves it.

To strike the balance, and moreover obviate all other possible difficulties to which I have referred, the right method of examin-

ing supposed albuminous urine is simply this:—Pour a *small* quantity—say a fluid drachm—of the urine into a test-tube; heat it to the boiling point, and then drop in *two* or three drops only of strong nitric acid. If phosphates have been precipitated by the heat applied, they will be redissolved, and the white flakes of coagulated albumen appear more clearly. On being allowed to stand, it will subside in the tube, leaving the urine above clear; thus defining the *amount* of albumen present in any given quantity of urine examined.

In the course of Bright's disease, other matters come away in the urine besides albumen. The kidneys are undergoing *structural* changes, and fibrinous or waxy casts of the urinary tubules, epithelial cells therefrom, &c., represent the progress of textural degeneration. These objects can only be discovered by examination with the microscope. They are fully described in a recent work by Dr. Basham.* The characters of albuminous urine—microscopically and chemically considered, in connexion with the structural changes of the kidney subject to Bright's disease, and other diseases—have been viewed in relation to diagnosis by Dr. George Johnson.† For the *general* mode of examining urine, full directions are given by Bence Jones.‡ A simple and practical method of analysing blood and urine is that described by Owen Rees:§ respecting the latter only, a complete guide to its analysis has been furnished by Thudichum, under the title "Pathology of the Urine" (1858). Its examination, microscopically as well as chemically, has been fully considered by Lionel Beale.‖

Turning from this to other excretions,—considered with regard to the etiology of blood-diseases,—the *sweat* being complementary to the excretion of urine, comes next in order. While, however,

---

* Dropsy connected with Disease of the Kidney—Morbus Brightii, &c., associated with Albuminous and Purulent Urine, 1858.

† Diseases of the Kidney, &c., 1852.

‡ Animal Chemistry.

§ Analysis of the Blood and Urine, in Health and Disease, 1845.

‖ Urine, Urinary Deposits, and Calculi, 1861.

much is known respecting the structure and functions of the sudoriparous glands, chiefly by the researches of Purkinje, Breschet, and Roussel de Vauzème, little can be said specially respecting this glandular system in its causative relation to morbid conditions of the blood; thus affording another proof, if more be necessary, of the incompetency of Physiology to predetermine anything in Pathology. The latter must be founded on independent observation; and in respect of the sudoriparous glands, their pathology has not yet been separately investigated. Arrest of the function of these glands—checked perspiration—has hitherto been observed only in connexion with febrile diseases; "but," remarks Erasmus Wilson,* "it is probable that the perspiratory secretion, like that of other secerning glands, may be diminished and checked, as a consequence of inflammatory disorder of the sudoriparous glands themselves, independently of the rest of the organism. Dryness of the skin, occasionally met with, is owing to the absence of secretion by the sebiparous or sebaceous glands."

Arrest of the perspiratory secretion must obviously induce *some* morbid condition of the blood, by retention of the excrementitious matters which should be eliminated. Rheumatism is usually attributed to this cause, under the influence of exposure to wet and cold. How far these circumstances are connected with the etiology of rheumatism, was noticed in a former chapter.

The perspiratory secretion of certain parts of the body—*e.g.*, the axillæ and feet—is apparently specially excrementitious; and the sudden suppression of rank sweat therefrom has been followed by typhoid symptoms of the worst character, obviously due to retention of poisonous matter in the blood.

Certain constituents of the urine and bile—*e.g.*, lithic acid and bilin, respectively—may be detected in the perspiration occasionally, and then the blood is assuredly *poisoned*. Besides, however, being complementary to other excreting organs, it would appear that the skin, in common with all such organs, eliminates

---

* Diseases of the Skin, ed. 4, 1857.

matters which are either of a poisonous nature, or, if innocent, have accumulated in *excess;* and which, in either case, had been taken into the circulation. The following substances have been detected in the sweat:—quinine, sulphur, mercury, iodine, iodide of potassium, assafœtida, garlic, saffron, olive-oil, rhubarb, indigo, Prussian blue, and copper.* Suppressed elimination of these matters will cause morbid conditions of the blood, severally varying in importance with the particular matter retained. The perspiration has been known to undergo certain inexplicable alterations of colour, becoming blue, green, saffron, yellow, ruby, or black; and unless these changes are due to colouring matters only, they bespeak some more serious perversions of excretion, which, if checked, will inevitably be reflected in the blood.

The sweat sometimes becomes altered in another sense. Instead of being excrementitious, it may carry off some one or more of the *essential* constituents of the blood, which is thus robbed of what should be retained. For example, albumen has been found in the sweat in rheumatic fever, gastric, putrid, and hectic diseases, and on the approach of death.† Or, some essential constituent of the blood, and one that is also a normal constituent of the sweat, may be secreted in *excess,* affecting the blood's healthy composition and properties, by altering the *proportion* of its essential constituents. For example, an undue quantity of sweat is sometimes secreted in very hot weather, thereby draining off from the blood an excessive quantity of *water.* This disorder, known as idrosis, was witnessed by E. Wilson, in several instances, during the burning August of 1856; and a similar condition was a prominent feature of the 'sweating sickness' that occurred in England in the sixteenth century.

Possibly idrosis should be regarded as an excessive secretion of more than water; that *all* the constituents of sweat—which, as

---

* General Pathology, Stark, p. 1127.—Elements of Physiology and Therapeutics, Baumgärtner, p. 486.

† Op. cit., Wilson.

a whole, is properly excrementitious—are simultaneously eliminated, only in undue quantity; nevertheless, the blood's composition becomes altered, relatively to the *proportion* of *other* excrementitious matters passing into the circulation. The *balance* of *effete* matters of various kinds, ever mingling in the blood, is disturbed; and whatever relatively preponderates will represent a blood-disease, enduring until such balance is readjusted by a compensatory discharge of other excretions of an opposite character. This view of idrosis, and its relation to the blood's constitution, implies a pathological principle, well illustrated by the consequences of an excessive discharge of *other* excretions. Bilious flux, for example, as compared with a diminished proportion of urine excreted, gives rise to a constitutional disturbance, which, agreeably to the principle alluded to, is thus interpreted by Dr. C. J. B. Williams:*—Urine contains a large proportion of azote; its excessive separation from the blood, therefore, leaves a comparative predominance of hydrogen and carbon in this fluid. Bile, again, abounds in hydrocarbon, and its copious removal, therefore, leaves a superfluity of azote. Accordingly, a flux of bile is either accompanied by a highly loaded state of the urine, or by fever, not subsiding until the urine becomes very copious, or deposits an abundant sediment. The most probable interpretation of this fact is, that excessive secretion of bile disorders the composition of the blood: so long as the kidneys rectify this disorder, by separating in greater abundance the solid contents of the urine, no fever results; but if the kidneys fail in their task, fever ensues and continues until they resume it; then a free secretion from them, and copious urinary deposit, is symptomatic that the fever is declining.

Contrasting with the blood-disease consequent on *bilious flux*, an opposite state of the blood, signified by jaundice, is the offspring of a continued *deficient* elimination of bile from the system. This occurs in either of two ways.

---

* Principles of Medicine, 1856, pp. 131, 132.

By some mechanical impediment, which, precluding a free flow of bile into the duodenum, permits absorption of the bile imprisoned. The obstacle in question may be constriction or closure of the common bile-duct, the hepatic duct, or of the bile-ducts within the liver.* Without any obvious mechanical impediment to the free escape of bile, suppression of its secretion is more frequently the occasion of deficient elimination of bile. Structural disease of the liver by cirrhosis, or by fatty degeneration, operates in this way. So also various blood-poisons, such as through snake-bites, pyæmic infection of the blood, typhus, and malaria, intermittent, remittent, and yellow fever, severally give rise to jaundice. Ether and chloroform, likewise, are said to occasionally have this effect. In all these cases, the liver itself may be structurally healthy; the blood-poison alone is the cause of a suppressed secretion of bile, which, in turn, reacts upon the blood, inducing jaundice. Similarly, through the nervous system, mental emotions have, in some instances, paralysed the liver and produced this disease.

Omitting the symptoms of jaundice, there are two diagnostic signs of unqualified value—both being early, both constant and unequivocal. They are, a greenish-yellow tinge of the conjunctivæ, and a dark saffron colour of the urine, leaving a bright yellow stain on white linen.

Respecting the prevention of jaundice, this ocular proof of its first appearance is, however, surpassed by chemical examination of the urine, which reveals the presence of bile, or its colouring matter, in the blood, *prior* to its dyeing any texture in the body, and when escaping by the urine in quantity too minute to be visible, or made visible by its yellow stain.

The delicate tests supplied by chemical investigation are chiefly two; and for their particulars I avail myself of Golding Bird's work on Urinary Deposits.

Pour on a white plate, or sheet of writing-paper, a small quantity of the urine, so as to form an exceedingly thin layer,

* Clinical Treatise on Diseases of the Liver, Frerichs, trans. by Charles Murchison, M.D., 1860, vol. i.

and carefully allow a drop or two of nitric acid to fall upon it. An immediate play of colours, green and pink predominating, will, if the colouring matter of bile be present, appear around the spot where the acid falls.

Pettenkofer's test:—To a small quantity of the suspected urine in a test-tube, two-thirds of its volume of sulphuric acid are to be carefully added, taking care that the mixture, which soon becomes hot, never exceeds a temperature of 144 degrees. Three or four drops of a solution of one part of sugar to four of water are then added, and the mixture shaken. A violet-red colour is developed if bile be present. This familiar test was not regarded favourably by Golding Bird. His experience led him to doubt its accuracy, and in applying it there are numerous sources of fallacy to be guarded against; chiefly, that the action of sulphuric acid on sugar develops a red colour in the absence of bile. A mixture of albumen or oil with sugar will, even in very minute quantities, under the action of sulphuric acid, produce a purple or scarlet colour, as Raspail long ago observed.

Heller's test:—Add to the urine any albuminous fluid—serum of blood or white of egg; then pour in sufficient nitric acid to produce a considerable albuminous coagulum. Examined after a short repose, it will present a bluish or green colour if bile-pigment existed in the urine; whilst, if none were present, the deposited mass will be white or merely slightly yellow.

In concluding this summary of those 'morbid conditions of the blood' that are caused by perversions of Nutrition, otherwise than *inflammation*, and by those of Digestion, or, again, by defective Excretion through the *kidneys*, *skin*, or *liver*,—it is necessary to add a few words only on deficient Respiration, regarded as the cause of a blood-disease. I do not allude to the inhalation of air contaminated with animal poisons, inducing 'infectious' blood-diseases; they have been already noticed. *Deficient* Respiration, whether by the inhalation of air otherwise impure, or the deprivation of pure air, is productive of its own peculiar blood-disease. The phenomena of *asphyxia* speedily and fatally supervene when

the cause of dyspnœa is overwhelming, slowly and imperceptibly when more tolerable.

Besides these occasions of defective respiration, by the quality of air breathed, others are identified with the *act* of breathing; whether in respect to the mechanical action of the respiratory apparatus—the lungs, thorax, and muscles engaged in respiration—or in reference to the vital influence of that portion of the nervous system by which the respiratory muscles are brought into action, and which regulates the (mechanical) play of the whole apparatus. All such internal, and in most instances organic, causes of defective respiration are foreign to my purpose. Their prevention can scarcely be realized. So also any structural disease of the *heart*—its right half—whereby the pulmonic circulation being ineffectual, the whole mass of blood becomes insufficiently aerated, is another condition beyond the reach of preventive medicine as at present understood.

Insufficient supply of *pure* air arrests those changes which the blood should undergo during its passage through the lungs. Physiologically considered, respiration is that function or process whereby oxygen is received into the blood, and hydrocarbonaceous matter eliminated in the form of carbonic acid and water. They represent a portion of the effete matter accruing from textural decomposition, or, it may be, superfluous hydrocarbon introduced into the circulation as food; part of which, however, first forming bile, is subservient to the process of digestion prior to its elimination through the lungs, the remaining superfluity of hydrocarbon being deposited as fat. Bile, therefore, is not merely an excrement, although I have hitherto so regarded it in order to simplify our view of 'excretion' in relation to blood-disease.

Carbonic acid and water, collected from these sources, are eliminated by the act of expiration; while the venous blood, thus disburthened, is again converted into arterial blood and renovated by the absorption of oxygen. If, then, a due supply of pure air be wanting, the blood gains proportionately less by its transit through the lungs, and remains (proportionately) venous. With each returning act of inspiration, the blood is less and less reno-

vated, and hydrocarbonaceous matters accumulate, until at length *venous* blood passes to the left side of the heart, and thence through the systemic circulation. The brain—ever foremost in its demand for arterial blood—fails first; the heart next, as black blood revisits it through the coronary arteries. This successive failure of the cerebral functions, and of the heart's action, is more marked when the respiration is *suddenly* stopped,—*e. g.*, in asphyxia by hanging. In such case, also, the lungs refuse to allow black blood to pass through their capillaries, so that it accumulates in the pulmonary artery, right half of the heart, and systemic veins, which become engorged; and this pulmonic *obstacle* reacting on the already *failing* heart, soon brings the general circulation to a stand-still.

When, however, the supply of pure air is *habitually* insufficient, these phenomena are not observed. The individual so circumstanced is gradually reduced to the lethargic state of a reptile—slow in mind and body. This venous state of existence—this living death, implies the habitual retention of hydrocarbonaceous matter in the blood—matter as positively excrementitious as urine or sweat, and equally poisonous.

Associated with the pathology of slow asphyxia is the etiology of diabetes mellitus. The experiments of Bernard render it possible, if not probable, that the liver—normally elaborating sugar—may, by over-activity of its function in this respect producing an excess of sugar, become at least one cause of diabetes.* Again, normally speaking, sugar formed by the liver is burnt off, in part, if not altogether, by respiration,† and eliminated in the shape of carbonic acid and water; thus disposing of such hydrocarbonaceous matter. Let, however, the act of respiration be impaired by injury or irritation of the floor of the fourth (cerebral) ventricle, or of the medulla oblongata, then sugar accumulates in the blood—constituting the immediate internal cause of diabetes. Defective

* See Experimental Objections to this view.—Researches on the Nature and Treatment of Diabetes, F. W. Pavy, M.D., 1862, pp. 43 and 63.

† See Objections.—Physiology of Saccharine Urine, G. Harley, M.D., Med.-Chir. Review, vol. xx., July, 1857, p. 203.

respiration, therefore, no less than hyperactivity of liver-function, may be a cause of this disease; and if so by one mode of impaired respiration, then probably any other occasion of defective respiration, as by an habitually insufficient supply of fresh air, will also tend to the same effect—diabetes.

Impure air is productive of the general phenomena of asphyxia, and also of *peculiar* consequences, which should be known and distinguished in reference to *each* particular kind of gas. This in order, by timely warning, to suggest the prevention of its admixture with the air we breathe.

Sewer emanations and the air of cesspools are the most common sources of contamination. Other kinds of impurity are derived from special sources—*e.g.*, manufactories where animal (or vegetable) substances are employed, slaughter-houses, dissecting-rooms, tan-yards, catgut, soap, glue manufactories, &c.; those also where chemical materials of a volatile and poisonous nature are used—*e.g.*, white lead, emerald green, arsenic, &c.

Sewer or cesspool emanations consist chiefly of sulphuretted hydrogen gas, which is always present, together with sulphide of ammonium, carbonic acid, nitrogen, sometimes phosphuretted hydrogen, and various organic living products—in fact, the very opposite constituents of pure air. An alkaline gas, diffused through sewer air, was detected by Dr. Odling.

The physiological, rather than the chemical, aspect of this sewage question has been experimentally investigated by Dr. Barker.* Premising that certain small animals were subjected to experimental observation, the results obtained show those effects which the *compound* impure cesspool produces, and those also that arise from the specific influence of *particular* gaseous poisons, and which, singly or collectively, emanate from the cesspool and the decomposing vegetable heap to pollute filthy localities.

The general symptoms are those of intestinal derangement, followed by prostration, heat of surface, distaste for food, &c.

* The Influence of Sewer Emanations, 1858.—On Malaria, Fotherg. Prize Essay, Med. Soc. Lond., 1858.

Of sulphuretted hydrogen, vomiting and diarrhœa are the first and most prominent symptoms. The latter is painful, the vomiting difficult and exhausting; and eventually insensibility and prostration supervene. When the dose of poison is at first very large, prostration and insensibility are immediate. In the experiments alluded to, the *dose* of sulphuretted hydrogen necessary to produce these effects was small in every instance. So little as 0·428 per cent. proved absolutely and rapidly poisonous. A much smaller proportion, 0·206 per cent., proved fatal in some cases; and so minute a dose as 0·051 produced eructations, tremors, rapid and irregular respiration, extraordinary rapidity of pulse, and diarrhœa.

Sulphide of ammonium induces symptoms different from those elicited by sulphuretted hydrogen: vomiting, without purging, although occasionally accompanied with tenesmus. When the dose is very large, death speedily ensues, with quickened and laborious respiration. Small doses repeated for many hours excite the circulation and thirst, followed by rapid sinking. The surface, from being unusually hot, becomes unusually cold. The tongue is protruded, dry, dark, and cold; while constant jactitation of the limbs, and a feeble, quick pulse, subside ultimately in death, even after some hours of exposure to pure air.* The *quantity* necessary to produce this result (in animals) has not yet been determined.

Carbonic-acid gas first affects the respiration. Prostration follows, and if the inhalation be prolonged, diarrhœa. The effects of this gas also vary with the *dose*, and the preceding are those of small, but long-continued doses. In larger proportions, insensibility, coma, and asphyxia are almost immediate. When carbonic acid has been breathed in small quantities for a long time, sufficiently to produce insensibility, the effect continues for some time after placing the animal in pure air. The quantity

---

* See also an Essay on the Cause of the Coagulation of the Blood, B. W. Richardson, p. 345.

of carbonic-acid gas which can be inhaled with impunity is unknown with certainty: from 1 to 2 per cent. is sufficient, when long inhaled, to produce decided symptoms of imperfect oxidation and subsequent prostration.

Such are the chief peculiarities of those phenomena which denote blood-poisoning, by sewer or cesspool gases.

Blood-disease, arising either from the habitual deprivation of pure air, or from habitually breathing impure air, as sewer emanations, points at once to defective house-construction, or at least defective repair. Either or both of these defects are very prevalent, and thus affect large classes of society, whose circumstances in other respects are singularly dissimilar. Rich and poor share these evils, if not equally. Private dwellings, although 'well-built,' are frequently not well drained; and, being air-tight above, are exposed below to sewer emanations from insecurely 'trapped' drains. Poisonous gases thence arising and diffusing themselves through dwellings so constructed, are retained in proportion as the window-sashes fit well and comfortably. The houses are almost gasometers. The ill-ventilated and ill-drained hovels of the poor, in many districts of London and the provinces, require no comment. Buildings of a public character, where many persons habitually assemble, are open—less to pure air than to criticism. Such are shops, printing establishments, factories, prisons, workhouses, schools, hospitals, barracks, churches, theatres, the law courts, and public offices generally. Ill-ventilated for the most part, they are also in many instances ill-drained. Impure air, therefore, aided by habitual deprivation of fresh air, is at work among those who, from necessity, duty, or pleasure, are led to frequent such places; and it is against this *twofold* evil in all cases that preventive measures should be directed and can be applied with success.

The guiding Principles of Prevention in respect of the various blood-diseases due to the causes assigned in this section, are at once suggested by the causes themselves.

On behalf of defective respiration, *ventilation* and *drainage*

demand immediate consideration. The average quantity of fresh air necessary to effectually aerate the blood is at least 800 cubic feet for each individual in twenty-four hours.* To secure this *minimum* adequate supply may be no easy matter. In other words, the practical application of the physiological principle implied by sufficient ventilation, in the actual construction of various forms of buildings, may prove a difficult problem. The chief difficulty would appear to be the combination of warming with adequate ventilation.

Preventive measures, aiming at this combination, have been experimentally investigated, and specially so by a Government Commission appointed for such purpose.† In their Report the following analyses were made of investigations relating thereto :—

Analysis of different plans for the prevention of smoke, by feeding from below upwards, and rotary or inverting grates.

Analysis of plans for the prevention of smoke, by a downward current.

Analysis of plans for consuming smoke, by means of hot-air chambers above.

Analysis of plans for preventing smoke, in part by deflecting hot air downwards, and by diverting cold atmospheric air direct into and through the fire.

Improvements in the forms of registers.

Improvements for economizing and utilizing heat, by returning warm air into the apartment.

Improvements for economizing fuel and heat, by combining reflection from polished surfaces with conduction and radiation.

Fire-grate stoves.

Miscellaneous improvements, including facilities for diminish-

---

* See Principles of Human Physiology, W. B. Carpenter, 1853, p. 530.

† Warming and Ventilation of Dwelling-houses and Barracks. Report to General Board of Health, 1857.—See also General Report of the Commission appointed for Improving the Sanitary Condition of Barracks and Hospitals, 1861.

ing the dirt and waste of an open fireplace, blowers and screens, facilities for quickly extinguishing fire, and attached fireguards.

Stoves in general.

In so far as these investigations determined the regulation of temperature in connexion with a due supply of air, they were calculated to overcome the chief difficulty experienced,—that of providing appropriate ventilation. In various ways fresh air can be duly supplied, but not warm as well as pure, simultaneously. Exposure to a cold blast is the more usual method of ventilation; witness Dr. Reid's experimental failures in the Houses of Parliament.

Drainage, fortunately, is more under control, whereby sewer emanations can be prevented, or their noxious properties neutralized.

Thus, various deodorizers and disinfectants render these effluvia inert: they are thus classified by Letheby:*—Those which combine with the putrid gases, and fix them in an involatile form,—metallic salts. Those which act chemically on them and break them up,—chlorine, sulphurous acid, nitrous acid, &c. Those which cause the putrid vapours to unite with oxygen, and so expedite decay,—the manganates and permanganates, fire, water, porous solids—*e.g.*, clay and charcoal.

Of all these disinfectants, probably none is so efficacious as the last—charcoal; and especially vegetable charcoal. Its virtues were first urged by Dr. Stenhouse,† and have since been advocated by Letheby. Wood and peat are the most effective. Either can be readily applied wherever or however sewer vapours may chance to escape. "You have but to place a small box containing a few pennyworths of charcoal in the course of the draught, and purification of the air will be complete. As far as we know, the strength and endurance of this power is almost unlimited; so that

---

* Report to the Commissioners of Sewers of the City of London, 1858.

† Journ. of Soc. of Arts, Feb., 1854.—Pharm. Journ., vol. xiii., p. 454, and vol. xiv., p. 329.—Economic Applic. of Charcoal to Sanitary Purposes, Lect., March, 1855.

when once the air-filter has been set up, it will last continuously for years."

Mechanical and chemical provisions for ventilating sewers have been fully investigated by Mr. Haywood.*

Temperature is the main question to be considered in reference to the prevention of *jaundice*. Not that other etiological conditions are unimportant, but they are for the most part beyond our control.

A hot climate *predisposes* to jaundice by enfeebling the circulation and inducing congestion of the liver. It is probable also that diminished oxygenation of the blood, through habitually breathing a hot atmosphere, aids this effect on Europeans, prior to their acclimatization. Hydrocarbonaceous matter, ordinarily eliminated by the lungs, accumulates in the blood, and the liver is proportionately overtaxed—a burden increased by indulgence in stimulating liquors, especially malt beverages, which, abounding with hydrocarbonaceous matter, fall heavily on this organ, and further tax its functional power beyond endurance.

Such being the predisposing influences of high temperature, any change of temperature approaching to cold is the *immediate* or exciting cause of jaundice. In *every* case, Sir Ranald Martin affirms,† that he has seen in England amongst those who have returned from India, cold has been the immediate cause of this disease. Taking seventy-two cases of icterus typhoides, by Lebert, one-third occurred in November and December.

The preventive measures suggested by these observations are obvious. They are most important to persons about to visit any tropical climate. Although unavoidably subject to the physiological influences of heat, certain precautions are available. Prudent moderation in the use of stimulating beverages, and the careful regulation of clothing, should be rigidly observed. Besides ade-

---

* Report to the Commissioners of Sewers of the City of London, 1858.

† Influence of Tropical Climates in producing the Acute Endemic Diseases of Europeans, ed. 2, 1861.

quate protection against the vicissitudes of external temperature, the cautious indulgence of cold drinks—iced beverages—is an injunction not to be forgotten.

On returning home, after residing for some time in a tropical climate, it is advisable, if possible, to pass the ensuing winter in a more even climate than that of England. And when residing again in this country, the continued observance of the precautions already enjoined, especially as regards vicissitudes of temperature, will prove the most efficacious preventive measures.

Suppression of the *perspiration*, no less than of the bile, is often caused by cold, and particularly by any sudden exposure. Rheumatism arises from some error of assimilation generating the rheumatic poison, without which there is ample proof* that this disease would never occur. Yet it is equally certain that such mal-assimilation is elicited by many causes. Predisposition may do much to develope the poison, but various exciting causes bring it into operation. Cold predisposes by its depressing influence on the system, and is also the *chief* exciting cause of rheumatism. Cold and moisture combined are the conditions most favourable to the generation of the rheumatic poison; while by checking the cutaneous excretion—opposing the effort by which this poison is naturally expelled—they promote its accumulation in the blood, and become the immediate cause of an attack of rheumatism.

Precautions against exposure to cold are therefore the circumstances essential to the prevention of this disease.

Passing from the skin to the kidneys; suppression of *urine*, or what is almost equivalent thereto, retention of urea in the blood (uræmia), ranks among preventible diseases.

Uræmia is the most fatal result of "Bright's disease" of the kidney, as albuminuria is its most certain and alarming symptom. Nutriment, in its best form—albumen—is incessantly draining away from the blood; while effete and noxious matter—urea, representing the decay of the textures—is silently retained; thus conceding the powers of life to the dominion of death.

* Rheumatism, &c., H. W. Fuller, 1860, p. 29.

Certain structural changes in the kidney are the immediate internal causes in operation;—congestion; interstitial effusion, with desquamation and damage of the tubuli urineferi; fatty or waxy degeneration, or rather *disintegration* of the effused matter; and lastly, its absorption and contraction, constituting the so-called 'granular degeneration' and irreparable destruction of the organ itself.

Whether we regard these structural changes as a consequential series, or as so many independent forms of renal disease, they are one and all comprehended under the title of 'Bright's disease;' for with *each*, 'albuminous urine' is invariably associated.

Now, this disease and accompanying albuminuria arise from preventible causes in most instances. Scarlatina is an occasional cause—rarely, however, a productive one, unless brought into operation by cold. After scarlatina has run its course, the residue of poison remaining in the blood appears to be naturally expelled by the kidneys, imposing extra functional duty on these organs; yet they generally fulfil their appointed task, unless when thus congested—bordering on inflammatory excitement, exposure of the body to *cold* should further impose an additional and intolerable burden. Then, under the pressure of *extreme* congestion, albumen is filtered off, urea retained, and fibrile dropsy supervenes. This *additional* strain on the excretory power of the kidneys, and its results, are obviously preventible. So also the more prevalent intemperate use of spirituous liquors imposes extra work on the kidneys; but even this strain may be made for years with impunity, although a hazardous experiment. Exposure to cold, however, becomes intolerable; it provokes albuminuria.

Such is the usual etiology of this disease, as originally investigated by Bright,* and which the experience of other observers has since confirmed. That "intemperance seems its most usual

---

* Reports of Medical Cases selected with a view of illustrating the Symptoms and Cure of Diseases by a Reference to Morbid Anatomy, 1827, vol. i., p. 3.

source, and exposure to cold the most common cause of its development."* These causes suggest appropriate preventive measures.

Diseases arising from 'mal-assimilation,' whether *primary* or *secondary*, admit of prevention, in many instances by watchful regulation of the diet.

'Diabetes *mellitus*,' not amenable to any known medicinal treatment, can be controlled for an unlimited period by appropriate diet. The rule to be observed is, a scrupulous avoidance of every kind of food containing sugar, or which can be converted into sugar. Rigorous abstinence is not equally imperative in all cases, yet the principle of prevention remains the same.

Animal food, therefore—including fish of all kinds and eggs—is quite unobjectionable; while of vegetable food, the choice is restricted to greens, bran-bread or cake, and such articles as do not belong to the saccharine class of aliments. Of liquids—brandy and water, tea and coffee, are safe.

The diabetic bill of fare does not allow of much variety, and constant self-denial is required to keep within its bounds. Nevertheless certain indulgences may be enjoyed with impunity; and, so far as the personal experience of *one* diabetic patient in particular affords adequate information for the guidance of others, Mr. Camplin's observations† respecting himself are valuable and encouraging.

Genoa maccaroni proved to be one of the best substitutes for the bran-cake. Milk need not be forbidden. Cruciferous vegetables afforded many agreeable varieties;—cabbage, cauliflowers, broccoli, Brussels sprouts, &c.; sea-kale and spinach are quite harmless; onions are allowable, and in most cases turnips. Lettuces agreed when eaten sparingly with oil and vinegar. Tea is preferable to coffee, and with it milk may be taken freely,

---

* Guy's Hospital Reports, 1836, vol. i. Cases and Observations illustrative of Renal Disease, accompanied with the Secretion of Albuminous Urine, p. 339.

† Med.-Chir. Trans., vol. xxxviii.

cream only in small quantity. Cocoa is allowable if prepared from the 'nibs,' not that which is sold in cakes or powder. Pale French brandy should be taken, but only in measured quantities, say a tablespoonful with water. Wines are better excluded, excepting claret, which is a most suitable beverage.

Other hygienic means were very advantageous. Sponging with tepid water, followed by friction, proved highly beneficial; so also sponging with cold salt and water in summer, and an occasional warm bath in the winter. Warm clothing, a leather waistcoat, and gutta-percha soles to the boots in winter, appear equally important. Change of air and occupation were so favourable, that wheaten bread was substituted for the bran-cake during the period of relaxation.

A few words respecting preventive medicinal treatment will suffice. Free perspiration affords some relief to the diuresis. Besides, therefore, the sudorific appliances just alluded to, the salts of ammonia are serviceable; the sesquicarbonate is an exception, at least it was so in Mr. Camplin's case. Citrate of ammonia, combined with citrate of iron, was useful. Bitters and alkalies proved very beneficial. Opiates are valuable in some cases as a temporary means of checking the secretion of urine.

Such are the preventive measures—dietetic chiefly, hygienic and medicinal subserviently—by the early and patient employment of which diabetes may be kept in subjection, and for an unlimited period. By their instrumentality Mr. Camplin not only rescued himself from a deplorable state of health, but was preserved from the ever-threatening recurrence of this disease during a period of no less than fourteen years.

The '*oxalic-acid* diathesis' connected with mal-assimilation seems to be allied to the saccharine diathesis.

The avoidance of sugar, and sugar-forming food, is therefore obviously indicated. Every species of vegetable food is questionable; and such as contain oxalic acid—*e.g.*, rhubarb, sorrel, &c.—are unquestionably forbidden. Animal food, with brandy and

water instead of beer or wine, form a suitable diet; but the water should be *distilled*, to deprive it of lime, with which the oxalic acid, otherwise combining, would lead to the formation of a urinary calculus. This being the principle of prevention by diet, its moderation, according to circumstances, is of course allowable; for a less exclusive selection of food is tolerated in many cases.

Appropriate medicinal treatment will aid preventive regimen. The mineral acids are efficacious, particularly hydrochloric and nitromuriatic acids, in conjunction with bitters. They were recommended by Prout to be taken daily for about a month, or until lithic acid, or the lithates, reappeared in the urine. "By adopting," says he, "such a course of acids three or four times in the year, and by a carefully regulated diet, I have seen this diathesis gradually subdued, and at length removed altogether."

The '*phosphatic* diathesis' is more complicated. Guided by its etiology, the principle of prevention is this:—to arrest, if possible, that destructive metamorphosis of nervous tissue which generates phosphoric acid in abnormal quantity. Consequently temporary alienation, at least, from all anxiety and corroding care—in short, mental relaxation—is primarily imperative. But the phosphatic diathesis is encouraged by vegetable food. A more animal diet, therefore, with beer and wine, is scarcely less imperative.

Deposition of the phosphates—'phosphatic urine'—implying, as it does, an alkaline state of this fluid—suggests the use of mineral acids. Opium also will aid in restoring acidity to the urine, besides subduing nervous excitement. The *earthy* phosphates are thus held in solution, and become invisible. The phosphatic diathesis itself, however, can only be kept in subjection by directing our preventive measures to its origin. Another occasion of 'phosphatic urine,' is an inflammatory condition of the urinary mucous membrane; thence the decomposition of urea and liberation of ammonia, by which the urine becoming alkaline, the triple phosphate of ammonia and magnesia,

with phosphate of lime, is deposited. As regards the mere deposition of these phosphates, mineral acids will, in like manner, counteract this tendency.

The '*lithic-acid* diathesis' bespeaks mal-assimilation, or represents an excess of animal food over and above the wants of the system, which is accordingly expelled *in limine* from the blood by the kidneys without having contributed to the nourishment of the body. A reduced proportion of animal food is obviously the leading preventive measure, and active exercise day by day an equivalent precaution.

Lithic acid passes off by the urine in the form of lithate of ammonia, which is liable to be decomposed by the action of any free acid present in the urine; and lithic acid itself being insoluble, appears as a reddish-yellow sand, or may form a calculus. This event will be intercepted by the occasional administration of alkalies, of which bicarbonate of potash is best for oft-repeated use.

Lithate of ammonia is soluble in urine at the temperature of the body, and its solution is thus secured, provided only that the urine be not overcharged. Dilution of the urine will prevent supersaturation and deposit. The free use of aqueous drinks is calculated to fulfil this indication, and anticipate, therefore, the formation of a lithate of ammonia calculus.

Such are the *principles* which, being derived from the etiology of 'urinary deposits,' guide the employment of preventive measures, and regulate those further details that are to be found in works specially devoted to this branch of Pathology.

Various Blood-Diseases arise from Contagion, illustrated by:
- Contagious Matter derived from Animals:
  - Hydrophobia, Snake-bites, Malignant Pustule, Glanders, Vaccinia.
- Contagious Matter derived from Human Species:
  - Hospital Gangrene, Puerperal Fever, Primary Syphilis—Chancres and Buboes.

The Prevention of these Diseases, and their Propagation.

When investigating the Principle involved in the diagnosis of Wounds (Chapter I.), I alluded, in conclusion, to the fact that

poisoned wounds, so called, owe their importance and tendency, not to the pathological nature of the local lesion itself, but to the introduction of some poison into the general circulation; the wound by which such inoculation has been effected being, in most cases, itself unimportant. I therefore purposely omitted the consideration of these lesions, and now take them in further illustration of that Etiological law which has hitherto engaged our attention:—Constitutional morbid conditions proceeding from Local causes; in the present instance, 'Blood-diseases,' arising from Contagion, properly so called, as distinguished from Infection.

Restricting this latter term to the communication of blood-disease by inhalation, by Contagion I mean the communication of disease by contact. Such disease is not, indeed, necessarily constitutional, but possibly merely a local affection; for example, itch, as contrasted with Secondary or Constitutional Syphilis.

Still, under the same general heading may be associated certain remarkable species of 'blood-disease' which have severally received names suggestive either of the *particular* constitutional morbid condition, or of the local lesion from whence it proceeds. Hydrophobia, Snake-bites, Malignant Pustule, Glanders, Vaccinia, Hospital Gangrene, Puerperal Peritonitis, Primary Syphilis—chancres and buboes, are all naturally associated together as being Contagious, and the offspring of Contagion. But of these, the first five are derived from *animals*, the remaining three from the *human* species. This difference allows a corresponding division of the whole subject. And when I shall have described consecutively the local and constitutional phenomena of *each* contagious blood-disease, and traced the relation of cause and effect between the (local) inoculation of each particular poison, and the mighty internal disorder subsequently displayed, we shall then be able to duly estimate the probability of preventing this, perchance fatal, disorder, by such timely intervention as may be necessary to withdraw the poison, or, at least, to arrest it on its way to the general circulation, or otherwise destroy its potency.

Hydrophobia may be taken first as the most apposite illustration of Constitutional disorder induced by Contagion.

Formerly, the reality of a hydrophobic virus was doubted, and even now-a-days the importance of a well-grounded belief on this starting-point must be obvious in relation to the appropriate means for preventing the consequences of the bite of a rabid animal. The dog and cat, and other animals of the canine and feline species, are peculiarly prone to hydrophobia; and there is abundant evidence to show that the bite of a 'mad dog, cat, wolf,' &c., induces this disease by virtue of the *saliva* thus introduced.

The wound itself is often trivial—a mere scratch or graze from a tooth of the animal in the act of snapping; yet a very large proportion of persons bitten by a rabid animal undergo hydrophobia, sooner or later, and die; and it is incredible to imagine that so many individuals, differing in their constitutional idiosyncrasies, should alike experience the same constitutional disorder, and that a fatal one, from a wound scarcely worthy of the name. The (morbid) saliva introduced by a scratch, itself trivial, is the only *peculiarity* of such lesion, and a very significant one.

Many recorded facts, read in this light, become intelligible.

Of fifteen individuals bitten by a mad dog, and attended at Senlis by the Commissioners of the French Royal Society of Physic, ten were bitten on the naked flesh, and five through their clothes. Of the *former*, five proved fatal.

Near Rochelle, twenty-four persons were bitten by a rabid wolf, and eighteen of them perished.*

Of ten other individuals bitten by a wolf, nine died rabid.†

Three persons bitten by a rabid wolf near Autun, all died of hydrophobia.

The existence of a hydrophobic virus, in the shape of a peculiar morbid saliva, has been also demonstrated experimentally.

---

* Recherches sur la Rage, Audry, éd. 3, p. 196.

† Mém. de la Soc. Roy. de Méd., p. 147.

Several animals were inoculated with the saliva of a rabid dog, recently dead; a dog, a cat, a horse, and a cock were thus infected by Dr. Zine.

Dupuy, in like manner, induced the disease by taking a sponge, which had been bitten by a mad dog, and rubbing it on the open sore of a sheep.

Youatt drew a silk thread backwards and forwards through the mouth of a rabid animal, and having inserted it as a seton in the neck of a sound dog, this animal subsequently died of hydrophobia.

If, then, the inoculation of a peculiar virus be indisputably the cause of hydrophobia, is this disease capable of being conveyed by clothes impregnated therewith? Can it be communicated by 'fomites'? Clearly so; for a dog's tooth is itself a fomes. To this effect, also, Mr. Trevelyan, writing to Dr. Bardsley, states that, after losing one pack of hounds by rabies, he not only removed the straw, but had the benches of the kennel scalded with boiling water, and afterwards all the joints painted and filled up with hot tar; the walls were then whitewashed, and the pavement cleaned with hot water. Thus secure, as he imagined, he collected another pack; yet rabies again broke out and recurred year after year. In consequence of these continual attacks, he removed the pavement, threw the earth beneath into the river, refitted, new painted, and whitewashed the kennels; ever after which the pack continued perfectly healthy.

The *saliva* of a rabid animal being the source of hydrophobia by contagion, explains many things otherwise anomalous.

This virus is usually introduced through a bite of the animal diseased, but the mere application thereof to ever so slight a wound, scratch, or other 'solution of continuity,' in an *absorbing* surface, is sufficient. In such case, the lick of a rabid dog generally induces hydrophobia. The surety of inoculation will obviously be affected,—by the number of wounds inflicted, by lodgment of the virus or its escape by hemorrhage; and above all, by unobstructed inoculation through the naked flesh as compared with the protection offered by clothes.

Apart from these modifying circumstances, in some cases no evil consequences ensue from the bite of a rabid animal, and the immunity enjoyed must be ascribed to some (unknown) peculiarity of constitution.

Overlooking exceptional cases, hydrophobia ensues in a variable period from the date of the reception of the virus. The wound has generally healed, or it may not have healed quite soundly. In either case, slight pain of a rheumatic character shoots from the site of the bitten part to some distance; so that if the hand be the part first affected, the pain extends up the arm and shoulder, fixing itself in the trapezius muscle probably, or the proximate side of the neck. Sometimes a tingling heat, or even a sensation of cold, is experienced, rather than pain; but either sensation equally *extends*,—say, up the arm and shoulder. Generally, actual pain is felt, and eventually shooting towards the heart. Meanwhile the cicatrix swells, reopens, and discharges an ichorous matter. Occasionally no local symptoms occur; neither the customary radiating pain, nor any inflammatory condition of the cicatrix.

After the lapse of some days perhaps, other and more formidable, because constitutional, disorder begins. Much might be said, in the way of interesting detail, respecting the symptoms then exhibited; but it would be foreign to my purpose—their prevention. Nevertheless it is necessary to clearly understand whatever we propose to prevent, and therefore to realize such a picture as may serve for (differential) diagnosis—in this instance —between Hydrophobia and Tetanus, which correspond in some particulars.

In both, the nervous system, together with its ally, the muscular, are the subject of all those phenomena which characterize these affections.

The cerebro-spinal axis acquires undue susceptibility. Rapid-flowing thoughts, reviving memory, and fertile imagination, are expressed by a more animated manner and conversation; although, in some cases, a downcast pensive mien, yet withal irritable and

peevish manner, prevails. Far more frequently, however, thoughts and fancies whirl through the brain tumultuously. A dull heavy pain caps the head and oppresses the temples, light is intolerable, and the slightest noise jars the whole frame; while sleepless nights or starting dreams, an overwhelming dread, muscular twitches, and pains in the neck, back, and limbs, complete the picture of morbid excitement—bordering on delirium. This state of comparative ease lasts only four or six days, possibly only a day or two; when the most remarkable symptom of true hydrophobia supervenes,—an *indescribable dread of fluids:* any attempt to drink—even the sight or sound of water, the thought of it, or anything associated therewith, as the noise of tea-cups, or of a pump—immediately excites a convulsive paroxysm, threatening suffocation. In some cases the slightest breath of air, a fly settling on the face, or a bright light, has the same effect.

Be it observed, these convulsive attacks are *paroxysmal,* with *complete* intermissions. A viscid mucus accumulating in the mouth occasions an *incessant action of the lower jaw* to extricate it. The pains now piercing the epigastric and præcordial regions, more particularly, the general sensibility, becoming more and more acute, and the convulsive paroxysms more frequent, desperate and protracted, soon exhaust the patient's bodily power, while his mind is hurried on to *furious mania;* and thus, when fighting for breath, or utterly worn out, the poor sufferer expires. The second or third day usually brings this happy release. It may be postponed to the fifth or sixth day. Age makes some difference, for children endure not longer than twenty-four hours generally.

I have italicised those symptoms which, contrasting with the phenomena of tetanus, serve to distinguish hydrophobia. To these characteristics may be added two more;—the *early* period of *death* from rabies as compared with tetanus, and its *longer* period of *latency.* On this point, however, considerable extremes are recorded.

In one hundred and thirty-one cases, none of the patients

became ill before the eleventh day after the bite, and only three before the eighteenth day.

Of fifteen patients whose cases Troillet was acquainted with, seven were attacked between the fourteenth and thirtieth days, five between the thirtieth and fortieth, two a little beyond the latter period, and one after fourteen weeks.

Of seventeen persons bitten by a rabid wolf, near Brive, ten were afterwards attacked as follows:—one on the fifteenth day; one on the eighteenth; one on the nineteenth; one on the twenty-eighth; one on the thirtieth; one on the thirty-third; one on the thirty-fifth; one on the forty-fourth; one on the fifty-second; and the last on the sixty-eighth day.

In two hundred and twenty-two cases, the disease showed itself eleven times before the tenth day; twenty-one, between the tenth and twentieth day; twenty, from three to six weeks; eighty-nine, from seven weeks to seven months; eighty-nine, from seven months to twenty-seven months; one, after four years; one, after five and a half years.

In Hamilton's table, it occurred in seventeen cases, between eighteen and thirty days; in sixty-three, between thirty and sixty days; in thirteen, between three and six months; in seventeen, between six and twelve months; in four, from ten to twenty months.

In both these series the period of incubation extended to between the third and eighth week.

The extreme periods, as determined by Dr. J. Hunter, were thirty-one days and eighteen months.

The most extensive and authentic observations accord about *forty days* as the '*mean* period of latency' in the *human* subject.

Here, then, is a season during which preventive measures can be employed with prospect of success. What are they? Prompt removal of the hydrophobic virus by the cupping-glass, and free excision of the bitten part.

It is almost impossible to say how late, in the period of incubation, these means would prove efficacious; for, if not employed

in any given instance, some constitutional peculiarity might itself avert the supervention of hydrophobia. There is just this poor chance of safety. But, considering the very large proportion of cases in which the disease *does* inevitably arise, no time should be lost.

Excision, therefore, should be practised at the *earliest* opportunity, and not postponed, however long it may have been neglected; provided, of course, that in either case there be sufficient reason for believing or suspecting that the bite was inflicted by a rabid animal. Before, then, resorting to a measure which entails permanent mutilation, more or less, the state of the *animal's* health in question should at once be inquired into.

The dog and cat chiefly concern us in this country. Rarely is the rabid state any approach to the popular notion of a 'mad' dog. No wild excitement appears, no savage tendency to bite, and certainly no dread of water, so remarkable in the human subject. Rather will our suspicion be aroused on ascertaining that the animal evinces only some strange departure from its usual habits and manner. In very many instances this peculiarity is a disposition to pick up straw, bits of paper, rag, thread, or any small object in the way. The animal laps water greedily. A disposition to lick is noticed in some cases, and particularly to lick anything cold—cold stones, or the cold nose of another dog; great aversion, however, to strange dogs and cats, especially to the latter, is very commonly observed at an early period.

Some such *unusual* propensity is soon followed by an *irritable*, peevish manner. The animal snaps those about it, and resolutely fights if the least provoked, soon becoming furious. When thus *obviously* dangerous, it is forthwith secured. Yet a dog dangerously rabid may be perfectly quiet and natural, save in respect of some unsuspicious, because unobtrusive, symptom. Allowed to run about, patted and played with as usual, it bites in an unguarded moment. In *this* state, therefore, as well as when obviously rabid, the animal should be at once chained up. Instead of being killed, it can then be watched, to ascertain the real nature of its

indisposition; thereby *determining* the necessity for excision in the case of any person who has been bitten. If the animal be affected with rabies, it will probably die in a few days; and this operation is imperatively demanded, as the only means of preventing hydrophobia in any of the human species who, having been bitten, would otherwise perish eventually. The safe custody of the rabid animal will also effectually prevent *further* mischief being done during his short period of probation.

Supposing an animal, not itself rabid, or bordering on that condition, to have been bitten—a healthy dog, bitten by another dog decidedly rabid. The latter will, of course, be killed; but when may the former apparently healthy dog be allowed to go free? This question, bearing directly on the spread of rabies among animals, bears equally on the prevention of hydrophobia in the human species, by eradicating the source of this disease.

The question turns on 'the period of latency' among *animals*. In the dog it is considered to terminate about the end of the sixth week. At the Veterinary School, Alfort, when a dog is bitten, it is chained up for fifty days, and, if healthy at the end of that period, is restored to its master. Mr. Samuel Cooper used to mention in his lectures, at University College, an instance of a more extended period than that which regulates the preventive measure adopted at Alfort. A large Newfoundland dog, having been bitten by another dog, did not become rabid until seventy days had elapsed; information the more valuable, since Mr. Cooper himself watched the case from beginning to end.

In Lord Fitzwilliam's pack the disease appeared at various intervals, from six weeks to six months.*

We may therefore conclude, that an animal bitten by another in a rabid state should be chained up for a *longer* period than the experience at Alfort would suggest as an adequate preventive injunction—imprisonment for six weeks.

Preliminary to this question is another—the 'communicability'

---

* Morbid Poisons, R. Williams, M.D.

of hydrophobia or rabies among animals and man,—a consideration essentially relating both to its *propagation* and prevention.

Many facts tend to show that the communicability of this disease depends on the inherent capability of the animal affected to engender it spontaneously; failing which, he may bite in vain.

The dog can generate rabies, and therefore can communicate it to another animal, or to man, in the shape of hydrophobia.

Sheep, horses—the herbivora—and man cannot generate the disease, and therefore cannot communicate it. Thus, rabies in a flock of *sheep,* consequent on the bite of a rabid dog, is not communicated from one sheep to another, although the sound are often bitten by the diseased, and in parts stripped of wool (Dupuy). At the Alfort Veterinary School three sound sheep, two dogs, and a horse were inoculated with the saliva of a rabid *horse;* not one of them became affected.

In keeping with this view, *man* cannot communicate hydrophobia. By the experiments of Vaughan and Babington, animals were inoculated with the saliva of hydrophobic patients, but without any effect. In an exceptional experiment by Magendie and Breschet, one dog of two thus inoculated became affected; but this animal might have been previously diseased, especially as rabies was rife at the time. Paroisse inoculated three dogs with the saliva of a man in hydrophobia; the animals were kept and watched for nearly four months afterwards, during all which time they remained quite unaffected. Similar experiments were conducted by Gauthier, Giraud, Girard, and Bezard, with the same negative results. Lastly, there is no instance of one human being acquiring hydrophobia from another labouring under this disease, although in many instances attendants have been bitten by such persons.

In conclusion, the general inference to be drawn from these facts is this—that in estimating the danger to human life incurred by the liberty of rabid animals, those only which can *generate* rabies—*e.g.*, the dog—are dangerous. The human species affected with hydrophobia is harmless, should there be any tendency to bite.

Such considerations, coupled with a due knowledge of the duration of latency in different animals, particularly the dog, suggest adequate precautionary restraint; while, in the event of hydrophobic virus having been communicated to man through the bite of a rabid animal, the prevention of the disease then impending is fulfilled by free excision of the part bitten, as soon as possible, aided, if necessary, by cupping.

Of 'poisoned wounds,' in the ordinary sense, the bites of venomous snakes, happily of rare occurrence in this country, possess much surgical interest, chiefly with the view of *preventing* their constitutional effects, rather than the more hopeless intention of curing them.

The local effect of a poisoned wound is essentially *cellulitis;* inflammation of the (subcutaneous) cellular texture, announced by acute burning pain, accompanied with some, and perhaps, subsequently, enormous diffused swelling, not at first involving the skin. In severe cases the swelling spreads rapidly, and to an almost unlimited extent, so widely may it range.

The bites of the rattlesnake and of the cobra di capello answer to this general description.

A piercing pain is immediately felt, rapidly shooting through the limb; swelling quickly succeeds, and a mottled livid redness, indicating that the skin is now involved. The cellular texture of the whole limb, and perhaps down the proximate side of the trunk, becomes gorged with a bloody sanious fluid; and, as if to relieve this tense yet diffused swelling, phlyctenæ arise here and there. Very shortly the pain abates, the tension is exchanged for a flaccid softness, the limb is cold and benumbed; while patches of gangrenous skin announce that the work of destruction has commenced, not however disclosing the ravages already wrought beneath the skin in the subcellular texture, and still less the extent to which it may eventually be sacrificed.

Rapidly as all this mischief is accomplished, the 'constitutional' disturbance begins almost concurrently with the first introduction of the poison. Soon after the poisonous bite has been inflicted,

symptoms of muddling intoxication ensue. The victim mumbles incoherently, and staggering, as if dead drunk, is overcome with helpless prostration and oppressed breathing. Other and even peculiar symptoms are witnessed. Profuse cold sweating, bilious vomiting, and perhaps evacuations of bile; while a yellow hue overcasts the skin. Excruciating pain about the navel is sometimes experienced. The pulse quivers irregularly, the nervous system succumbs to the potent poison, and the sufferer expires.

Now, in order to prevent this fatal issue, and, moreover, the formidable antecedent symptoms, remember the poison begins to operate almost immediately, varying in this respect, however, with the dose. Still its period of latency is short.

In one case—carefully recorded by Sir E. Home*—a man was bitten by a rattlesnake at half-past two P.M., and brought to St. George's Hospital by three o'clock; during this brief period of only *half an hour* the constitutional disturbance had become overwhelming; and in the interval, when the man went of his own accord to a chemist's shop for relief, he was observed to stagger, and appeared drunk. Death ensued. In another instance,† the bite of a rattlesnake began to manifest its effects within the first half hour.

If, then, we assume the period of latency to be *under half an hour*, preventive measures should be very promptly employed. And what are they? Means whereby the poison can be removed from the bitten part, or arrested before entering the general circulation, or neutralized. Caustics—the actual cautery included—fulfils the latter purpose; a ligature above the part affected, the second object; excision, or the application of a cupping-glass, are calculated to withdraw the poison. Of these appliances, that of cupping is most efficacious and practicable, if not the only one of value.‡

---

* Phil. Trans., 1810. † New York Med. and Phys. Journal, vol. ii.

‡ Experimental Researches on the Influence of Atmosp. Pressure on the Blood in the Veins, and on the Prevention and Cure of the Symptoms caused by the Bites of Rabid or Venomous Animals. D. Barry, 1826.

Malignant pustule is also communicated by 'contagion,' the poisonous matter being caught directly from beasts, or their remains; for the disease is not propagated apparently by the human species, from one individual to another.

The local symptoms are these:—Soon after any accidental inoculation with such morbid matter, a stinging sensation is felt, and a red point appears, hardly elevated above the skin. Then, at this point, the cuticle rises into a blackish vesicle, which speedily runs into a slough, surrounded by an œdematous swelling, having a violet tinge, and spreading rapidly in all directions. Occasionally, neither vesicle nor pustule arises, only swelling. Such was the character of the disease in three cases observed by Lawrence.* With obvious swelling, however, a sensation of tension, rather than pain, is experienced. Should several pustules arise, the disease is proportionately more perilous, and especially if situated on the neck or face; for then the swelling may be so considerable as to threaten suffocation or congestion of the brain. Indeed, malignant pustule is not unlike carbuncle in appearance, but differs from it in being always the result of contagion. And this etiological consideration will always determine the diagnosis.

A variable period having elapsed, the constitutional symptoms supervene. Fever, attended with pain over the stomach and vomiting, delirium and prostration.

The causative relation between the local lesion and this constitutional disorder is shown by their invariable sequence. The latter always *follows* the former; and, moreover, it is very doubtful whether the constitutional disorder can be induced by contact with the same morbid matter, unless succeeded by the formation of malignant pustule. This local lesion, therefore, would appear to be the *only* cause—the only mode of origin, of the constitutional morbid condition.

The nature of the morbid matter itself is unknown, but it is developed in beasts affected with 'contagious carbuncle.' The poison

---

* Lancet, 1825-6, p. 127.

may be imparted by any accidental inoculation, in handling the animal when alive, or during the manufacture of hides, wool, &c. Malignant pustule is consequently most rife among butchers, tanners, shepherds, and wool-beaters. It may also be produced by eating the flesh of animals thus diseased. Instances in proof of this mode of production are cited by Wagner and Turchetti. Temperature and moisture have apparently some influence, for the disease is most prevalent in damp localities, and in wet autumnal weather.

The morbid matter—whatever it be—retains its poisonous power for a long time; but the disease is probably not propagated by the human species, from one individual to another.

Preventive measures are obviously suggested in accordance with the known etiology of this disease; although in the pursuit of certain avocations it may be difficult so to protect the hands and exposed parts of the body as to escape contagion. Even then, however, *this* preventive overture remains—the progress of malignant pustule can generally be *arrested* at its commencement.

Glanders is another disease derived from certain *animals*—the horse, ass, or mule—similarly affected.

Inoculation of the human species is apt to occur by handling either animal when glandered; and the constitutional disorder thence arising may be briefly described as fever, resulting in the production of many inflammatory tumours in different parts of the body, which have a great tendency to suppurate and fall into gangrene. Towards the close of glanders, in eleven of fifteen cases (Rayer), puriform mucus, mixed with blood, oozed from the nostrils; in ten of these cases, the discharge came from one nostril only; and in all cases the quantity was inconsiderable, sometimes scarcely appreciable. The eyelids, also, are tumified, and secrete a thick viscid mucus. This disease runs its course in a period varying from a few days to many months.

Its period of latency (in man) varies from two to eight days.

I am not aware of any means whereby the progress of glanders can be arrested when once inoculation has taken place, however

early our intended preventive measures may be applied. If in this respect unlike the bite of a rabid animal, a snake-bite, or malignant pustule, glanders has at least one advantage—the disease can always be *avoided.*

As bearing on the question of prevention, the early and exact diagnosis of glanders in animals is important. It is communicated to man by an animal in a state of disease so obviously characteristic that the danger cannot be overlooked.

Two varieties of this disease, in the horse, ass, or mule, are recognised by Williams.*

In *gangrenous* glanders, the animal immediately loses its spirits, and staggers; the nasal and conjunctival mucous membranes are beset with a number of red points, which, at the end of twenty-four to forty-eight hours, become livid; the nostrils now discharging a yellow matter, streaked with blood. This condition lasts two or three days; then the nasal membrane falls into gangrene, and large ulcers form where portions have sloughed. The discharge increases and exhales a fetid gangrenous odour; œdema of the nostrils, scrotum, and legs soon supervenes: at length, the nostrils being glued together, respiration fails, and the animal dies.

In *pustular* glanders, the same general debility and fever are observable as in gangrenous glanders. The specific inflammation of the nasal membrane is an eruption of pustules, said to resemble confluent small-pox, followed by a copious yellow viscid discharge from one or both nostrils. After two or three days, these pustules ulcerate, sometimes internally, so as to destroy the bones and cartilages of the nose. By absorption of the nasal morbid secretion, the sub-maxillary glands become swollen and tender, but only on the inflamed side of the head. Such enlargement is called the 'kernels.' Œdema of the nostrils, the sheath, and hind limbs succeeds, as in the gangrenous variety; and respiration failing, death ensues on the eighth or tenth day, at latest.

---

* Morbid Poisons.

Glanders is often accompanied with 'farcy,' and farcy often ends in glanders.

Button farcy is characterized by inflammation of the cellular texture, forming tumours in different parts of the body, the head, neck, and extremities, particularly the hind legs. In four or five days, they soften and ulcerate. It is an inflammation of the lymphatic glands and vessels, usually beginning in the hind extremities, attended with lameness, and forming an irregular swelling of the limb, which at length ulcerates and discharges a sanious fluid.

The period of latency in glanders affecting animals is generally short. Two asses—one about a year old, the other about a year and a half old—were inoculated by Turner. In the former the maxillary glands became tender on the second day, and the discharge from the nostrils was established on the following day. In the latter the maxillary glands enlarged on the third day, but the nasal discharge did not appear until the sixth day. In a horse inoculated with farcy matter, the disease did not appear until the end of three months, and then precisely at the points of puncture. Gerard states that he introduced the matter of the discharge every day, at different times, into the nostrils of certain horses by means of a brush, and that the disease appeared on the seventh day, but in two others not until the thirty-second day.

Fortunately, however, glanders is not an eminently contagious disease, either from one animal to another, or from this source to the human species; and its communicability from one human being to another is very doubtful. A case once apparently occurred in St. Bartholomew's Hospital:—a healthy nurse contracted disease from a glandered patient, and she died after a short illness, having every symptom of glanders.

But assuming that glanders can be readily detected and distinguished from all other diseases, the prevention of its *first* propagation will consist in forthwith destroying the diseased animal from which, as the centre of contagion, it might spread.

This preventive precaution is not alone sufficient protection to

man or beast. The disease is communicable by *fomites*, as well as by direct inoculation from one animal to another. In this way, some of the discharge from the nose of a glandered horse having remained about the manger, rack, or partition of a stable, may be thawed by the breath of a new horse, or introduced into the system in the act of nibbling or licking, whereby sound horses have speedily become glandered when put into a stable whence a glandered one had been taken weeks or months previously—thus reviving the disease, and with imminent peril to grooms and others in attendance. "Let, then," says Youatt, "the halters, head-gear, and bridles be burned; the clothes washed and baked; the pails newly painted; the racks and ranges thoroughly scraped, then washed well with soap and water, and afterwards with chloride of lime and water, in the proportion of a pint of the strong solution to a pail of water; let the walls be well scraped and washed with the chloride of lime and water, then well lime-washed; the floor be first thoroughly scoured, then sluiced with the chloride: and, with all these precautionary measures, every possibility of danger will be removed."

Strangely contrasting with all such scrupulous injunctions for the avoidance of contagious disease, there is *one* at least of animal origin—which is now sought throughout civilized communities, and even enjoined by law—it being the protective substitute for another contagious disease, formerly the most fatal scourge of the human race. I allude to the protection conferred by cow-pox against the ravages of small-pox.

Acting on a popular belief prevalent among the dairy-farmers of Gloucestershire, that no person who had undergone the trifling eruption of cow-pox could take small-pox, our immortal Jenner first confirmed this belief by the direct test of inoculation, and at once foresaw its mighty significance in the exemption which might thus be vouchsafed to his own and future generations by undergoing the trifling *substitute* disorder. Then, extending his experimental inquiry, he found that inoculation with lymph procured from the *vesicle* of this disorder in the human species—

'vaccinia'—was no less protective than that derived from its original source,—the teat of the cow. Thence the practice of 'vaccination.'

Of course this grand discovery received the welcome usually offered to any innovation far in advance of the age in which it is presented to the human understanding. All the "idols of the den," of "the theatre," and of "the market," had to be dethroned ere this majestic symbol of Truth could gain access through the portals of prejudice, when at length it was acknowledged as the most precious contribution to the resources of Preventive Medicine; and as such Vaccinia will be found in this work in its own proper place, associated with the prevention of small-pox.

The next group of contagious diseases—those derived from *man*, and thence propagated—comprises at least three species:—Hospital Gangrene, Puerperal Peritonitis, and Primary Syphilis—chancres and buboes. I omit other diseases of this kind—gonorrhœa and certain skin-diseases, which are *essentially* local affections, and do not originate constitutional disorder,—in the shape of consequent 'blood-disease.'

Hospital gangrene is fortunately unknown to but few surgeons in civil practice now living; and we must refer to those of the past for information. The shortcomings of personal observation, are however amply compensated by the testimony of many witnesses on record; for it will be readily believed that a disease so formidable has ever been watched with deep interest whenever it occurred. From *original* sources of information, therefore, we are enabled to identify this disease, while its etiology suggests appropriate preventive measures. Hospital gangrene has received various other names; not because of any doubt respecting its pathological nature, but as expressive of its various characters, and mode of origin. Thus, Phagedænic Gangrene, Sloughing Phagedæna, Putrid Ulcer, Pourriture, Contagious Gangrene, and Hospital Sore, are severally synonymous.

The term *Hospital* gangrene, or *Hospital* sore, is assuredly not

always applicable. Overlooking the many phases of this gangrene, and regarding only its more constant phenomena; its nature is perhaps best expressed by denominating it, *essentially,—gangrenous* inflammation; that is to say, inflammation certainly, but inflammation passing at *once* into gangrene, thereby appearing only a process of textural disintegration and disorganization. Sometimes the process of destruction resembles more that of phagedæna, alternating with rapid sloughing; so that the worm-eaten phagedænic surface suddenly becomes a large slough, and then again phagedænic. These different aspects of the disease are apt to mask its really gangrenous character. What, then, are the phenomena more constantly observed? Acute pain, sudden engorgement and bloated swelling, dusky-red discoloration around the doomed part, and conversion of its textures into a putrid glutinous or slimy slough, exhaling a peculiar fetid odour. Rapidly extending, all the soft textures are soon melted down, leaving only the bones staring, of an ebony black,—as if the rafters of a house where a fire has raged.

Observe how these general characters are clearly visible in the portraits of this disease, drawn by eye-witnesses; varied, however, by special circumstances; principally, by the kind of previous injury to the part affected, the textures engaged, and the constitution of the individual.

To identify this gangrene under all such modifying circumstances, I shall trace its *origin* and *progress,*—in a stump after recent amputation, an incised wound, granulating as an ulcer, a recent gunshot wound, a small puncture, an old sore, and lastly, a blistered surface,—where the characters of this disease appear in their most elementary form.

For the particulars of these six aspects of Hospital Gangrene, I avail myself, chiefly, of Blackadder's original and valuable treatise.*

When a *stump* is affected, and the patient has a plethoric habit, or is accustomed to live freely, intense inflammatory action

* Observations on Phagedæna Gangrænosa, 1818.

soon runs through its whole substance; swelling rapidly increases, so that in a few days the stump acquires more than twice its former size, and being much indurated, occasions the most excruciating pain. In this state the patient may become delirious, and die suddenly by effusion into some of the larger cavities. More frequently, however, gangrene seizes upon the integuments and cellular texture, large sloughs are thrown off, and some of the larger blood-vessels giving way, the patient sinks under the exhaustion of repeated hemorrhage. For it is commonly found that the usual modes of stopping hemorrhage from a stump are in such cases either inadmissible or totally inefficacious.

Sometimes the progress of the disease in a stump is more *gradual*, yet ultimately nearly as fatal,—inflammation is much less acute, there is comparatively little tumefaction, and the pain is far less severe; but the discharge is much more copious, and the cellular texture connecting the integuments and muscles is rapidly destroyed. Hemorrhage generally supervenes later than in the preceding instance; it is, however, the most common cause of death.

An instance of this latter variety of gangrenous inflammation affecting a stump occurred in a patient of mine, at the Royal Free Hospital, in March, 1862. I amputated a man's arm for a large epithelial cancer situated on its inner aspect. The operation was necessarily performed very high up,—about two inches below the head of the humerus; the flaps were quite free of the disease, were ample, muscular, and well adapted; yet in the course of three days the stump suddenly began to swell, and the sutures, threatening to cut their way through the integuments, were removed. The flaps then lay easily together, but a pulpy slough was uncovered externally, which exhaled a mawkish odour. Two small ligatures were detached spontaneously, from the midst of this slough, *without* hemorrhage; and on the ninth day, the main ligature from the axillary artery separated spontaneously, also without hemorrhage. Nevertheless, two days hence, secondary hemorrhage sprang suddenly from this artery, and so freely that

the man would probably have perished then and there, had not the house-surgeon, Mr. Hill, chanced to be in the ward at the time. The whole stump was now one pultaceous mass, the fragments of flaps retracted, exposing the bone, from which even the periosteum had melted away. The poor man, however, experienced little or no pain in this wreck of a stump. He sank and died on the fifteenth day after the operation. Then, on separating the remaining bits of flaps, their muscular portions were seen to be converted into black shreddy pulp, bathed in a quantity of grumous bloody fluid, which rolled out plentifully from underneath the pectoral muscles; the muscles themselves were transformed into the aforesaid black pulp, which extended as far as the sternum. I also noticed a coextensive pulpifaction, posteriorly —beyond the bit of lower flap.

Sometimes, a stump, almost soundly *cicatrized* to all appearance, will suddenly burst open and undergo gangrenous disintegration. Such cases are well described by John Bell.* In one day he saw three stumps burst open, each of which had so nearly cicatrized, that you could have covered the small spot remaining unhealed with the tip of the little finger.

When this gangrene supervenes after any amputation, the case may be regarded as that of an *incised wound*, healing probably by adhesion, but suddenly diverted from primary union, and undergoing the aforesaid process of destruction.

Observe the same gangrenous inflammation supervening on a healthy *granulating* wound, or ulcer.†

The wound, or ulcer, becomes painful and swollen, loses its healthy florid appearance, while the granulations, which were small and distinct, become flabby, and in some cases appear as if they were distended with air; in others, vesicles containing a watery fluid or bloody serum, have been observed, and the

---

* Principles of Surgery, ed. Charles Bell, 1826, vol. i., p. 144.

† Observations on Hospital Gangrene, with reference chiefly to the disease as it appeared in the British Army during the late war in the Peninsula. John Boggie, M.D., 1848, p. 42.

sensation in the sore has been described as resembling the stinging of a gnat. The secretion of pus is arrested, and the surface is covered with a tenacious viscid ash-coloured matter, which adheres firmly. After some time a discharge of thin ichorous matter ensues, a very peculiar cadaverous febrile smell, the pain increases, the edges of the sore are reverted, and generally assume a circular form; an erysipelatous redness encircles it, extending possibly to a great distance, even over a whole limb; the neighbouring glands, as those of the axilla or groin, swell, inflame, and perhaps suppurate. Omitting the constitutional symptoms thence arising, the (local) inflammation goes on apace, thin ichor continues to be discharged in great quantity, and a thick slough, apparently of coagulable lymph, like melted tallow, covers the whole surface of the sore, the fetor is intolerable and the pain insupportable. At last an oozing hemorrhage soaks the dressings, or a larger blood-vessel having sloughed, robs the patient of his last remaining source of strength, and the typhoid constitutional commotion soon terminates in death.

Blackadder noticed that when the disease attacks a *large* recent wound, its whole surface may be affected at once; while in other cases, the gangrene commences on, or near, the lips of the sore.

Passing from incised to *contused* wounds, "when," says this authority, "the disease attacks a recent gunshot wound, the discharge, two or three days after contagion, is found to be lessened, and to have become more sanious than purulent. The sore has a certain dry and rigid appearance, its edges are more defined, somewhat elevated and sharpened, and the patient expe-riences in it a stinging sensation, as if occasioned by a gnat. Then, or a day or two later, the integuments at the edge of the sore become inflamed, and the surface of the sore itself assumes a livid or purple colour, and appears covered with a fine pellicle, such as forms on coagulating blood."

In respect of a gunshot wound, John Bell compares the disease to erysipelatous gangrene, and denominates it Erysipelas

Gangrænosa. He extends this view to Hospital Gangrene, as seen affecting *any* narrow wound. A punctured wound ever so trivial, a mere scratch, may present a swelling one day, on the next erysipelas apparently of the arm has supervened with dreadful pain and low fever; on the third day the arm will become livid, and covered with vesicles; and in two days more fall into gangrene.* Much as such inflammation may resemble erysipelas, the *dreadful* pain and *immediate* gangrene *rapidly* extending, answer rather to the disease we are considering; and it is better to restrict the term erysipelas to a disease having its own peculiar characters.

"When," observes Blackadder, respecting hospital gangrene attacking a *puncture* or scratch, "the morbific matter is thus inserted, its first appearance resembles that of a part inoculated with *vaccine* virus." The primary inflammation begins at the end of the second, or early on the third day; it reaches its height about the sixth; but when the scab begins to form in one disease, *phagedænic* ulceration begins in the other, and if allowed to proceed soon affords sufficient proof of the non-identity of these diseases.

Should the disease attack an *old sore*, where a considerable depth of new texture has been formed; a vesicle arises, filled with a livid or brownish-black fluid, which bursts and assumes the appearance of a small dark coloured spot. Such vesicle or spot is usually situated at the edge of the sore. Phagedænic ulceration, spreading therefrom, makes comparatively very slow progress through the surface-bed of new texture, but having reached the subjacent natural texture, its progress is suddenly accelerated, acute inflammation supervenes, and a large slough forms.

Lastly, when attacking the skin from which the *cuticle* has been *removed*, as by a blister, one or more small vesicles first appear, filled with a watery fluid, or bloody serum of a livid or reddish-brown colour. The situation of this vesicle also is gene-

* Op. cit., vol. i. p. 140.

rally at the edge of the sore. Its size is not unfrequently that of a split garden-pea, and it is easily ruptured, the pellicle which covers it being very thin. If the vesicle contains a watery fluid, and has not been ruptured, its appearance resembles a greyish-white slough; if containing a dark-coloured fluid, or if ruptured, it appears a thin coagulum of blood, of a dirty-brownish black colour. During the formation of the vesicle a painful sensation in the sore is generally experienced, like that of a gnat stinging.

Thus the earliest and most elementary phase of Hospital Gangrene is *vesicular;* just as gangrene generally begins, and senile gangrene in particular. The features peculiar to, and characteristic of, this so-called Hospital Gangrene, ensue *subsequently*—its slimy slough, its rapid progress, its appalling devastation. I shall presently have to notice one character which foretells all the rest, I mean *propagation* by *contagion* (and possibly by infection also), whereby the disease runs through a whole ward—a whole hospital, if unchecked, passing from bed to bed with the rapid strides of death.

The local *results* to be anticipated are always sad, sometimes shocking. Among Blackadder's cases; in one, half of the cranium was denuded, the bones were black as charcoal, the integuments detached posteriorly to the second cervical vertebra; anteriorly, to the middle of the zygomatic process of the temporal bone; and this was originally only a superficial scalp wound. In another case, the integuments and cellular texture, on the anterior parts of the neck, were destroyed and the trachea laid open, presenting a horrid spectacle. Among John Bell's cases, in one, the skin and muscles were carried away from the shoulder down to the bellies of the supinator muscles; and eventually, when the sloughing terminated, nothing seemed to be left of the arm except the bone, covered with a velvet-like surface of shining red flesh; and this was originally only a narrow splinter wound on the middle of the outer side of the arm. Continuing our course down the body, in one case, a very slight and superficial wound of the thigh grew into a sore, at first no bigger than the palm of one's hand; in

two days as large as the crown of a hat; and in a week the whole skin of the thigh was destroyed, the muscles were stripped of skin and fascia from the hip to the knee, the trochanter was almost laid bare, the ham-string muscles were exposed to a considerable extent, and all the muscles of the thigh dissected in a manner which no drawing could express.

Another slight wound in the thigh, "had the cellular membrane so destroyed in the course of a few days, that you could put your clenched fist into the hip, and lay your hand sideways betwixt any two muscles of the thigh. You could have counted each muscle, as if dissected, from the tuber-ischii to the ham. The branches of the profunda femoris first gave way, then the sciatic vessels: for three nights the patient lost two or three pounds of blood each night: it would have been almost cruel to stop the hemorrhage had it been possible, so very desperate was his situation: on the fourth day he died."

A similar but worse case, if possible, was seen by Blackadder. The muscles, large arteries, and nerves of *both* thighs were exposed and dissected, the integuments and cellular substance having been entirely removed, excepting only a narrow strip of the former, which remained on the outer side of either thigh. Yet this also was originally a simple flesh wound.

With even these cases in view, imagination will scarcely realize the ravages of this gangrene. A tattered skeleton, still held together by ligaments? No; for the joints may be laid open extensively, and the knee, ankle, elbow, or wrist disarticulated.

Long before any such irreparable injury has been inflicted, the constitutional powers take affront. The period, however, at which the constitution begins to exhibit symptoms of irritation is extremely irregular, sometimes as early as the *third* or *fourth* day, sometimes as late as the *twentieth*.

The countenance assumes an anxious or feverish aspect; the appetite is impaired; thirst succeeds, and the tongue is covered with a white mucus. Some constipation at first, ends in diarrhœa. The pulse is rather irritated than accelerated. The general symptoms, however, have an inflammatory or typhoid character, accord-

ing as the causes of either predominate. When an inflammatory diathesis prevails, the system becomes gradually more irritated, until acute inflammation attacks the sore, an event that frequently happens about the end of the second week. At this period the pulse is frequent and sharp, and not uncommonly the patient undergoes one or more shivering fits, succeeded by great increase of heat, seldom or never terminating in a profuse perspiration. The cold fit is sometimes followed by a bilious intestinal evacuation, with mitigation of the febrile disorder. If the local mischief be not arrested, weakness increases daily to exhaustion; the fever loses its inflammatory character, and unless the patient be cut off by hemorrhage, he falls a victim to extreme debility. When the disease has a typhoid character, the pulse is small and frequent, the appetite and strength gradually fail, not unfrequently diarrhœa supervenes, and the patient at length sinks, retaining his mental faculties to the last.

Now, the question presses—whether does the constitutional disorder or the local disorganization precede? What is the order of succession; which is the cause; which the effect? Authorities are nearly equally divided on this question; but the *weight* of authority decidedly preponderates in favour of the *local* origin of hospital gangrene.

Thus, Blackadder affirms—"that in no single instance which he had an opportunity of observing, did the constitutional symptoms precede the local, unless the case be held an exception, where a stump became affected after amputation had been performed, on account of the *previous* effects of the disease.

"That the morbid action could almost always be detected in the wound or sore *previously to the occurrence of any constitutional affection.*

"That in several instances the constitution was not affected until some considerable time after the disease had manifested itself in the sore.

"That when the disease was situated on the lower extremities, the lymphatic vessels and glands in the groin were observed to be in a state of irritation, giving pain on pressure, and were some-

times enlarged before the constitution showed evident marks of derangement.

"That the constitutional affection, though sometimes irregular, was in many cases contemporary with the second, or inflammatory stage.

"That when a patient had more than one wound or sore, it frequently happened that the disease was confined to one of the sores, while the other remained perfectly healthy, and this even when they were at no great distance from each other."

To these five arguments three more may be gathered from Welbank's well-known essay,* forming so much additional support in favour of the local origin of this disease.

Thus, the fact of its earlier stages being attended with little or no constitutional disturbance, was also noticed and urged by Welbank; and, "that when such disturbance does supervene, from the increased extent of the local malady, and unites in alliance doubly subversive of healthy processes of resistance, the latter stages of the disease are progressively accelerated.

"That part of the diseased surface may clear off and granulate, while gangrenous disorganization continues progressive at the opposite edge.

"That, however advanced the sore, it not unfrequently becomes healthy, and rapidly so, on the application of appropriate local measures;" and, "that the symptoms of general disturbance also which supervene are promptly relieved by measures that control the local condition."

Partly at variance with these facts and arguments, is the experience of Guthrie:† "That the febrile symptoms do seem to follow the appearance of the local alteration, is, in many cases, indisputable; that they precede or accompany the local symptoms in many other cases, is indisputable; and that the disease in a mild state, although capable of committing much mischief, is neither preceded nor followed by febrile or constitutional symptoms, cannot be doubted."

---

* Med.-Chir. Trans., vol. xi., 1820.

† Commentaries on the Surgery of the War in Portugal, &c., 1855, p. 165.

On the other hand, there are, or have been, those who affirm that the constitutional disorder generally, at least, precedes the local disorganization.

Thus, John Bell wrote:*—The hospital sore is *usually* preceded by a degree of fever.

Thomson—writing also from his own observations—states,† that these two classes of symptoms—the local and constitutional—are not invariable in the order of their appearance; for *sometimes* the one, and sometimes the other class, seems to occur first in the order of succession; but that the constitutional symptoms *usually* precede the local. Hennen also advocated this doctrine even more strenuously.

The question of 'local' or 'constitutional' origin may be further examined by reference to the *known causes* of hospital gangrene.

All observations respecting the etiology of this disease agree in certain particulars; that it is not developed epidemically, but rather as an *endemic,* springing up in some way among those who become affected. Thus, newly-built hospitals are free, so also are generally those built on high ground; while the disease arises in crowded and ill-ventilated hospitals. The *possibility* therefore of some species of 'blood-disease,' resulting from inhalation, is obvious. Thence this constitutional source of hospital gangrene would be *one* mode of origin.

Thomson, indeed, regards the disease as possibly 'infectious.' "I have seen (says he) hospital gangrene introduced into an hospital by a single individual, and, when proper precautions were not taken, spread extensively among the other patients, but chiefly among those who lay *nearest* in the ward to the person originally affected, or among those who had had *most frequent intercourse* with him. I have also known patients attacked in succession with hospital gangrene who had used the same bedding, or who, without using the same bedding, had occupied in quick succession the same small apartment."

* Principles of Surgery, ed. cit., vol. i., pp. 142, 147.

† Inflammation, p. 458.

Fomites, therefore, can convey the infectious matter, and retain it some time.

So highly infectious is this disease, according to Boyer's observation,* that it broke out in wounded patients, who, hoping to escape, had quitted the infected hospital, and retired to elevated situations where they breathed the most salubrious air.

The 'infecting distance,' or range of the contaminated air from the focus of infection, is unknown; but the disease certainly attacks patients at *some distance* apart (Thomson). Nor is the period of latency, after infection, more exactly determined. "I think (says this author) I have repeatedly seen the disease attack patients in less than three days after they had been exposed to its influence."

The production of hospital gangrene by infection seems to be disproved by Blackadder's observations, and chiefly by the result of the following experiment. He placed three patients with clean wounds alternately between three other patients severely affected. Their beds were on the floor, and not more than two feet distant from each other; but all direct intercourse was forbidden, and they were made fully aware of the consequences of inattention to their instructions. The result was, that not one of the clean wounds assumed the morbid action peculiar to the disease, nor was the curative process in any degree impeded.

Respecting the conclusiveness of this experiment, it is barely possible that rather less than two feet was *beyond* the infecting distance of hospital gangrene, assuming the disease to be infectious. But this assumption is negatived, and the purely 'local' (as opposed to 'constitutional') origin of hospital gangrene established by a large series of observations. The local cause itself is the state of the wound or sore induced by the direct application of morbid matter from the wound of one patient to that of another, and so on, by using the same sponges, water, &c., in dressing. The disease is thus *propagated* also by 'contagion.'

---

* Traité des Mal. Chir., tom. i., p. 322.

This mode of communication can be traced as the *only* cause, in all cases since the days of La Motte, 1722, and Pouteau, in whose posthumous works, published 1783, hospital gangrene was first specially noticed.

Pouteau observed that it may be communicated to the most healthy wound or ulcer in a person of the best constitution, and breathing the purest air, by merely placing in contact with any such wound or ulcer sponges, lint, &c., contaminated by contact with a sore undergoing the disease. Thomson concurs in the accuracy of this observation, as well as advocating the infectious character of hospital gangrene. Delpech* traced its propagation in almost every instance to the *direct* application of the morbific matter to the sores. Blackadder also recognised this mode of propagation in ninety-nine cases out of every hundred. Welbank arrived at a precisely similar conclusion. He speaks of the disease as being 'highly contagious' by using the same sponges to different patients.† And Guthrie specifies this as one of the 'conclusions'‡ of his observations during the Peninsular War. Nay more, that it is contagious through the medium of the atmosphere applied to the *wound* or ulcer.

Boggie's experience§ during the same war compelled him to admit that hospital gangrene may be propagated by contagion, although he is disposed to attribute less importance thereto than to the continued operation of other local causes of an 'irritating' character, in the shape of dirt, acrid applications, motion or mechanical irritation, attributing also considerable importance to other stimulating causes affecting the constitution; as hot weather, stimulating food, and the intemperate use of wine and spirituous liquors. In short, Boggie considers it inflammatory gangrene, and recommends antiphlogistic regimen.

While, however, admitting that all such causes may *predis-*

---

* Mém. sur la Complication des Plaies et des Ulcères comme sous le nom de Pourriture d'Hôpital, 1815.

† Op. cit.

‡ Commentaries on the Surgery of the War in Portugal, &c., 1855, p. 171.

§ Op. cit.

pose to hospital gangrene, it does not arise in numberless instances where these and similar causes are conjoined in full operation. Their *essential* importance, therefore, cannot be allowed.

The etiology of hospital gangrene is so far obscure that its cause or causes, in the *first* instance of its occurrence, require further elucidation.

The disease may possibly arise from over-crowding in an ill-ventilated hospital or other institution; and is perhaps communicated by infection, from the sore itself emitting a poisonous exhalation; but it is assuredly also propagated by contagion.

Guided by these views, the nature of our preventive measures is at once suggested. When the *first* case appears in any hospital or other institution, our immediate object is to decompose the poisonous slough and discharge, and, moreover, arrest the progress of gangrene. Strong caustics, such as will fulfil these intentions, should therefore be forthwith applied. Arsenic was originally recommended by Blackadder. The liquor arsenicalis, or diluted with an equal quantity of water, or with twice that quantity, was used, according to the emergency of the case. Strong nitric acid was first employed by Welbank. Either of these caustics should be applied until a new, hard, dry slough is formed, encrusting a clean and healthy surface.*

To prevent the *propagation* of this gangrene, punctilious cleanliness as regards the hands of the dresser, lint, water, and other appliances, should be daily observed in dressing all other wounds, even the most trivial and healthy. The bedding, also, should be changed, and clean linen furnished, as often as may be necessary to prevent their impregnation and the accumulation of fomites. Free ventilation and isolation of the patient will have similar preventive efficacy, and should not be neglected, although the disease may not be assuredly infectious. The protective value of these measures has long since been fully established by their results.

---

* For further details as to dressings, the writings of the authorities above mentioned may be consulted with advantage.

Thus, in military hospitals, the disease is apt to occur. Yet my own experience in respect of hospital gangrene, as it appeared during the late war, in the hospitals in the Crimea and at Scutari, does not approach what was formerly daily observed in *civil* hospitals. In the Hôtel Dieu, for example, it raged without intermission for two hundred years—so much so that "a young surgeon," says an ancient French author, "who is bred in the Hôtel Dieu, may learn the various forms of incision, operations too, and the manner of dressing wounds; but the way of curing them he cannot learn. Every patient he takes in hand must die of gangrene." Now-a-days, such scenes have passed away like a dream. This disease has not been witnessed for many years in the hospitals of this country, excepting on two occasions,—in the Middlesex Hospital, 1835,* and in the University College Hospital, 1841.† With these exceptions, true hospital gangrene—contagious gangrene—has not reappeared; and many experienced surgeons of the present day have no practical knowledge of it whatever.

The disappearance of a disease, once so frequent, once so formidable, can only be attributed to its causes having since subsided; and they are precisely the circumstances that have been specially obviated by the hygienic arrangements of the well-appointed and well-regulated hospitals of recent years; nevertheless, we are still bound to recognise hospital gangrene and to remember its etiology, as the best security for our continued observance of those protective measures which can alone prevent the recurrence of this disease in future.

Puerperal peritonitis is another illustration of contagious disease inducing constitutional disorder—puerperal fever; always serious, often fatal.

After parturition, the internal surface of the uterus resembles a large open wound or sore, ready therefore to absorb any poisonous matter in contact therewith; and it would appear, that putrid blood or portions of placenta may undergo absorption, or inflam-

* System of Surgery, Chelius, trans. by South.

† Lancet, 1845, vol. i., Lectures, Liston.

mation supervening, that its products are liable to be thence absorbed into the general circulation. The consequent blood-disease is a most formidable constitutional disorder.

Peritonitis first ensued, commencing on the uterine reflection of the peritoneum, and exciting some degree of inflammatory fever. But the constitutional disorder induced, is not *this* fever; it is entirely superseded by absorption of the aforesaid poisonous matter, and resembles *typhoid* fever.

From the subject of puerperal peritonitis, as a centre of contagion, the poisonous matter is apt to be conveyed by the hand of the accoucheur, and thus inadvertently communicated to other lying-in patients,—rich and poor. The disease is undoubtedly contagious, although this may not be the only mode of propagation.

Absolute cleanliness therefore in performing the responsible duties of accoucheur, from patient to patient, and especially in lying-in hospitals, where many patients are together under the care of the same medical attendant, represents the cardinal principle of prevention, as regards the propagation of Puerperal fever from its original source.

For further information respecting the contagious character of this disease, the reader is referred to the observations of R. Lee,* R. Fergusson,† Armstrong,‡ Robertson,§ and Gordon;‖ and for a more extended evidence of its contagious origin, see the Registrar-General's Report, vol. v. An elaborate analysis of the pathology and etiology of Puerperal Fever has been contributed by Churchill.¶

The Local origin of Constitutional disease is further illustrated by the usual consequences of the syphilitic virus, when applied to any absorbing surface of the human body.

---

* Researches on the Pathology and Treatment of some of the most important Diseases of Women, 1833, part i.

† Essays on the most important Diseases of Women, 1839, part i.

‡ Facts and Observations relative to the Fever commonly called Puerperal, 1819.

§ Med. Gazette, No. 214.

‖ A Treatise on the Epidemic Puerperal Fever of Aberdeen, 1795, p. 57.

¶ Diseases of Women, ed. iv., 1857, p. 656 *et seq.*

Like certain other potent agents in nature, the syphilitic virus having hitherto baffled every attempt to isolate it, is known by its effects.

Chancre announces the admission of this virus into the body; *bubo*, its absorption, and passage through the lymphatic glands, thence to the blood, which becoming contaminated and in some way deteriorated, represents *Constitutional* syphilis.

Here then is a continuous course of events; but the two former, being antecedent, are denominated *primary* syphilis, and they are local; while the latter is *secondary* or constitutional; and their relation, as cause and effect, fully illustrates—by the *career* of Syphilis—the local origin of constitutional disease.

The prevention of this consummation will be determined by the promptitude and exactitude with which the local disease or cause in operation can be detected, and removed, should opportunity offer.

Now the birth of primary syphilis is open to observation and experiment. Moreover, it is generally supposed that some time elapses ere the syphilitic poison begins to work.

Hunter mentions instances of chancres not having appeared until twenty-four hours after sexual intercourse; in one case seven weeks elapsed, in another two months, before chancre appeared. Lawrence adduces an instance of five weeks' delay.

The value of such data obviously turns on the reliability of the patient's own testimony; and be it remembered—in relation to the probability of constitutional syphilis ensuing—that absorption of the poison is not postponed, and that this is accompanied with, and denoted by, the formation of chancre.

What then is a chancre? A primary syphilitic ulcer. But what are its peculiar and diagnostic characters, if any?

In all cases, beginning insidiously with a trifling itching, affecting, say the glans penis, or rather a spot in the furrow at its base, near the frænum, or it may be on the prepuce or the skin of the penis itself; there soon appears a small pimple whose summit speedily becomes a vesicle, containing at first a thin transparent

lymph, gradually becoming thicker and opaque—in fact, purulent. This papule, vesicle, and pustule is *alike* the origin of all* chancres; yet how diversified their subsequent appearances, and how like those of ordinary ulcers. The granulating healing sore alone excepted, we recognise—the simple syphilitic ulcer, the indolent and indurated, the irritable, the phagedænic sometimes indolent, the inflamed, and the sloughing phagedænic. When the pustule of an incipient chancre bursts, we observe a 'simple' ulcer, without any special characters. This, the first of all primary (syphilitic) ulcers, may soon become 'indurated' by effusion of lymph beneath its base, and around.

When "confined to the *base* of the ulcer," such induration—elastic, cartilaginous, and terminating abruptly—was regarded by Hunter† as characteristic, and *the* specific diagnostic mark of true chancre. Yet Hunter himself acknowledged, in the sentence immediately preceding this, his definition, that the indurated base is not peculiar, being common to other indolent ulcers.

A slight modification, as it appears to me, of Hunter's doctrine, has since been revived and advocated by M. Ricord,‡ who affirms that cartilaginous induration at the base, and *around*, a primary syphilitic ulcer, is absolutely *pathognomonic* of "infecting chancre," or that species which accompanies and denotes constitutional syphilis. He regards this character as the local expression, by chancre, of the syphilitic blood-disease—in short, the first secondary symptom.

But precisely the same condition of induration may be produced artificially by irritating applications—*e.g.*, kali purum (Hennen), corrosive sublimate (Acton), and better still, says Ricord, by chromate of potash, or by nitrate of silver, the nitric and sulphuric acids; "so as to deceive even the most careful and experienced practitioners." I may add, for example, that Aber-

---

* See different view—Recherches sur la Syphilis, 1861, J. Rollet; and Syphilitic Inoculation, 1863, H. Lee.

† Venereal Disease, edit. Home, 1810, p. 229.

‡ Lecture on Chancres, trans. C. F. Maunder, 1859.

nethy* was thus betrayed by an indurated sore, and recommended mercury to be rubbed in to salivation! Observing, however, that it produced no amendment, and ascertaining that the hardness had several times *subsided* and returned, an unirritating dressing was used, when the induration disappeared without the use of mercury.

Under other circumstances, pertaining rather to the state of the general health, and the condition of the digestive organs in particular, the primary ulcer becomes 'irritable,' that is to say, painful and disposed to bleed easily. This, therefore, cannot be considered a *specific* sore. Nor again is the 'phagedænic' in any way characteristic. The same irregular worm-eaten sore, with sharp, undermined edges, may either be syphilitic, or proceed from the depraved constitutional condition by which a sore becomes phagedænic. Less characteristic is an obviously 'inflamed ulcer,' and that mixed appearance known as 'sloughing phagedæna.' In short, the primary syphilitic ulcer may assume many characters, but none of them are *peculiar* to syphilis. Primary syphilis cannot thus be diagnosed with more than equivocal probability.

This *negative* result of observation is supported by the united testimony of all who during the present century have most patiently watched, in vain, to discover any characters whereby *chancre* can be surely distinguished. First:—Rose† (1817), whose experience in the hospital of the Coldstream Guards ranged over a large number of cases, admitted that although there are many symptoms common to chancres, they are not entirely peculiar to them. Hennen‡ (1829) acknowledged with regret that there are "not any invariable characteristic symptoms by which to discriminate the real nature of the primary sore;" and, having witnessed many instances of self-deception, in attempting to diagnose a sore for the cure of which mercury is indispensable, from one of a different nature, he repudiates the pretensions of those who assume to themselves the possession of a "tactus eruditus" by

* Surgical Observations, 1804, p. 135.
† Med.-Chir. Trans., vol. viii., p. 358.
‡ Milit. Surg., 1829, p. 525.

which they can distinguish this kind of chancre. Recurring to the diagnosis of chancre, rather than the kind, Mr. Bacot's experience* (1829) led him to affirm, that chancre may present every variety of appearance to which a breach of surface is subject; and Colles† (1837) stated, "as the result of long, attentive, and anxious observation," that primary venereal ulcers present an almost endless variety of character. To this effect also were the observations of Wallace‡ (1838); for, says he, neither the mode of origin, nor the form, nor the colour, nor the size, nor the number of the ulcers of primary syphilis, are pathognomonic. Acton§ (1851) avowed it was incontrovertible, that other sores, not of a specific nature, may assume all the aspect of real chancres; and Labatt‖ (1858), considering the great variety of appearances presented by primary ulcers, experienced the difficulty of classifying them, and confessed that hitherto every such attempt had ended only in disappointment.

Much less then is there *the* specific chancre.

I pass from this aspect of primary syphilis to the question—What is *syphilitic* bubo? for perchance *its* physical characters may be more uniform and peculiar, although available only at a later period than those of chancre, to guide our diagnosis.

Now it is alleged that bubo denotes absorption of the syphilitic virus from the primary sore, which, on its way to the blood, through the absorbents, irritates the absorbent glands, whereby they become swollen and hard, and perhaps suppurate. Compared with chancre, bubo can scarcely be called a *primary* symptom, for although the time of its accession after chancre is uncertain, it is always somewhat *later*. In many cases bubo *never* supervenes, the syphilitic virus apparently passing on uninterruptedly to the blood, without any demonstration during its journey, as if a high-

---

* Treatise on Syphilis, 1829, p. 149.
† Prac. Obs. on the Venereal Disease, 1837, p. 75.
‡ On the Venereal Disease, 1838, p. 84.
§ Diseases of the Urinary and Generative Organs, 1851, p. 380.
‖ Obs. on Venereal Diseases, 1858, p. 48.

way robber should overleap a toll-bar without paying the customary toll.

This uncertainty respecting the formation of bubo, and delay in its coming, seriously impairs its diagnostic value, with reference to the prevention of secondary or constitutional syphilis. Then, again, true syphilitic bubo may possibly, although not probably, occur without previous chancre. Bubo may be the only primary symptom. Such buboes therefore have been named "primary buboes," and by the French "bubons d'emblée." Delpech, Sir A. Cooper, and many others deny their existence; whereas Fallopius, Hunter, Wallace, Lagneau, Swediaur, Bertrande, E. Wilson, and other authorities affirm that they do occur.

As being possibly the *only* primary symptom present, the question presses—are there any characters whereby *syphilitic* bubo can be assuredly identified? If no peculiar characters distinguish it, then bubo, no less than chancre, alone considered, is equivocal evidence in our diagnosis of primary syphilis.

I need not dwell on the possibility of bubo arising from irritation of the lymphatics, in some part more or less remote, the irritation having apparently been propagated along the course of the absorbents leading to the swollen glands, without, however, any poisonous matter having been absorbed. The femoral glands becoming enlarged from the irritation of a chafed toe is a familiar illustration. This *sympathetic* bubo is easily distinguished by the kind of local cause in operation. But if swelling of the lymphatic glands exist alone, unaccompanied, and not preceded by any kind of sore, in any situation, then how can we pronounce *that* swelling a syphilitic bubo?

Many species of bubo are attributed to syphilis, yet they all appear to be one and the same kind in different stages of progressive inflammation. Beginning as a hard and somewhat painful swelling of the lymphatic gland usually nearest to the spot whence the poison is absorbed; the absorbents themselves proceeding to the enlarged gland, share this inflammatory induration, and sometimes feel like hard whipcord. Along the back of the penis such

cords may be felt leading to the groin, where mostly, just above Poupart's ligament, lies the swollen gland. Or, in the female, if a chancre be situated anteriorly on the vulva, this swelling is discovered at the external abdominal aperture; and if situated posteriorly, then betwixt the labium and the thigh, inflamed lymphatic vessels lead to a swollen hard gland in the groin. Occasionally bubo is situated elsewhere, when the vessels of absorption pass through other than the inguinal glands. Hunter saw a syphilitic bubo far down on the thigh; and he met with other instances on the lower part of the abdomen.

Wherever placed, as the gland enlarges it successively engages the surrounding cellular tissue and skin; the latter becoming inflamed and adherent, fixes the tumour. Suppuration *may* ensue. The skin acquires a stretched and shiny appearance, while the cuticle desquamates in receding circles, and the cutis becomes thinner. At some point fluctuation can be felt, the thin skin cracks, matter is discharged, and a cavity exposed that speedily assumes the condition of an ulcer.

Up to this point, the most critical observer could detect nothing to characterize the swelling syphilitic bubo, beyond the fact of its association with a sore, the nature of which, however, is itself doubtful; less certain, therefore, would the diagnosis be were bubo present alone. Carmichael* acknowledges his inability, before ulceration, to determine whether bubo be syphilitic or not; and if the former, what *degree* of venereal disease; but that when ulceration takes place, the bubo partakes of the same mildness, or malignity, as the primary ulcer from which it originated. Yet these signs are equivocal, for, like chancre, the ulcer of an open bubo at once acquires all the characters of some kind of *common* ulcer; it may be the indurated and indolent, the irritable, phagedænic, inflamed, or the sloughing phagedænic. The size of this ulcer, of course, varies; usually not larger than the bubo itself, occasionally, and fortunately but seldom, it becomes enormous,

---

* Clin. Lectures on Ven. Diseases, 1842, p. 109.

extending inwards over the perinæum, or downwards over the thigh, or upwards, perhaps, as high as the navel, while its depth may threaten the femoral artery.

Now all this—the origin and progress of an inguinal bubo—does not pronounce it syphilitic. Alone, its physical characters are fallacious—association with a sore elsewhere, the nature of which, as determined by its physical characters, is doubtful, renders the evidence only probable in some degree. Is there any *pathognomonic* sign or test which converts this indefinite probability into certainty? If a sufficiently early, as well as certain test of the primary (local) disease, it would provide the opportunity for preventing 'constitutional syphilis.'

Inoculation supplies the requisite test. The pus from a chancre, or bubo, being introduced beneath the skin of any part by a lancet charged therewith, produces a chancre. Thence in like manner another may be produced: first a pimple, then a pustule, then an ulcer. This power of reproduction is the conclusive test, and was known to and used by Hunter. Since his time Evans, Bell, and other observers have alike dwelt on its value, and Ricord himself, who almost accords to 'induration,' the rank of a pathognomonic sign of the 'infecting chancre,' nevertheless regarding chancre as a distinct *species* of ulcer; acknowledges that its individuality "exists neither in its form nor in its floor, nor in an absolute manner in any one of its external characters. Its nature is in the pus which it secretes. Inoculation is the *only* pathognomonic character of chancre, and which *alone* suffices to establish the diagnosis."*

Here then is an infallible test of chancre—the nature of its pus, as manifested by its inherent power to reproduce a similar ulcer—offspring and parent being alike, if under the same constitutional influence; and the offspring—chancre, inherits the power of propagation.

Now this power is *inborn*, and available therefore as the test

---

* Op. cit.

of chancre from the very first formation of pus, when the chancre is yet a pustular pimple. Inoculation is the *earliest*, as well as the most infallible, criterion of chancre. Hence its diagnostic value in relation to the prevention of Secondary Syphilis. Not so 'induration.'

I have already exposed the equivocal character of this symptom, considered as an exact sign; and Ricord unwittingly bears testimony to its coming *too late* for the *prevention* of Constitutional Syphilis by local measures—*escharotics*. Ricord's experience leads him to affirm that the "specific induration" never precedes the ulceration; that generally toward the *end* of the first week following the infecting coitus this induration manifests itself; in the second week it becomes developed, and that while its development is rarely delayed until the third week, it is *never met with earlier than the third day*. About *the end of the first week*, and never before the third day, appears the induration. But Ricord's experience of escharotics applied to chancre, is that "within the *first four days* only, you can thus ward off the general intoxication—you then kill the syphilis in its germ." Sigmund, of Vienna, also states as the result of his observation in upwards of a thousand cases, extending over eleven years, that secondary manifestations never appear when the chancre is completely destroyed within the first four days. "I will tell you in a word," continues Ricord, "the grand secret in the treatment of chancre; it is to reduce the specific ulceration to the state of a common ulcer, and to transform a wound possessing a special principle for its maintenance, into one which no longer has such a resource." This object is effected in the most marked manner by *cauterization*,—not a slight superficial cauterization, which only destroys the surface of the ulcer, but a cauterization deep, broad, and destructive. Cauterization, by means of a paste consisting of sulphuric acid and charcoal, applied over the whole sore and around, so as to include the infected peripheric zone of tissue beyond it. This local treatment alone converts the chancre into a simple healing ulcer; and "it is, moreover, in a social point,

the most powerful *prophylactic,* since by destroying surely and promptly contagious affections, it extinguishes the *nuclei* of infection."

Now this preventive treatment is possible only within the first four days, and Ricord's "specific induration" would not suggest it until the end of the first, possibly not until the second week, and never *before* the third day. In fact, Ricord regards induration as the first *secondary* symptom.

The guidance of inoculation, however, anticipates constitutional syphilis from the first moment of a pustular pimple. It is the earliest, no less than the most exact method of diagnosing chancre. Moreover, this method acquires a critical value from the fact established by Ricord and other observers, that the *intensity* of the constitutional manifestations about to ensue, cannot be measured by the number of chancres present simultaneously. The multiplicity of chancres no more increases their poisonous effects than the multiplicity of vaccine pustules insures protection against small-pox. Inoculation from a *single* chancre assuredly foretels *any* measure of constitutional perturbation; it is the brand of syphilis.

What if this method of diagnosis, and the appropriate preventive treatment of constitutional syphilis, be postponed to a *later* period? Even then the open suppurating *bubo,* if present, is a chancre, and its pus will produce another chancre by inoculation. Not the pus secreted in the cellular tissue round about the suppurating gland, but that issuing from the gland. Here, then, is an *additional* test. We overtake the specific pus on its way to the blood, and possibly in time to anticipate its poisonous effects. Mere gonorrhœal bubo, or sympathetic bubo, arising from any *irritation,* does not yield *specific* pus; and the only question affecting the value of diagnosis by inoculation from bubo, is whether it be *sufficiently* early? Ricord observed that bubo, however large, which has arisen from a chancre when about to heal, is simply a swelling of the gland, and fails to supply virulent matter. The poisonous pus hitherto secreted by the chancre was

transmitted quietly through the absorbing lymphatics, without occasioning any outbreak in the glands; and now that they do offer some resistance, as it were, the pus still transmitted from the chancre is no longer specific.

But up to this time the *chancre* itself continues available for inoculation. The period of "*statu quo specific*" extends to when the ulcer cleanses itself, begins to throw out healthy granulations, and to cicatrize from its circumference. The duration of this period is considerable—frequently weeks, sometimes months. I have now (August, 1860) under my care a female patient in the Royal Free Hospital, who has had an eruption on her skin, has still an ulcerated throat, and whose eyes present, each, around the iris, a red zone bordering on iritis; yet with this train of secondary symptoms, two *specific* pus-secreting chancres still remain—one on either labium; and these, the patient states, she acquired in December, 1859! No other chancres are present, nor are there any vestiges of others having been; and indeed, judging from her course of life since the time mentioned, when she was received back to a respectable home, it is probable that no sexual intercourse has since taken place. On this assumption these two chancres have continued specific during a period approaching *eight months*. Ricord maintains that the soft chancre, or that which he terms non-infecting, in a constitutional sense, mostly cicatrizes in the course of a few weeks; not unfrequently, however, the period of repair is prolonged *much beyond;* and that this form of chancre persists in preserving its virulent specificity almost up to the last moment of its existence; while, indeed, cicatrization is proceeding at the circumference, the pus is specific in the centre of the ulcer; on the other hand, "the infecting chancre once developed, is not slow to limit itself, rapidly attains the period of *specific statu quo*, and passes with equal rapidity to the period of cicatrization."*

The case I have adduced is apparently one exception at least to the invariability of M. Ricord's dictum, and the records of

---

* Op. cit.

other observers are, I think, opposed to it. Of course the influence of different modes of therapeutic treatment must be taken into account; and of all differences in this respect affecting the results of statistics, the *mercurial* and *non-mercurial* treatment are the most influential. Subject thereto, I must allow that *some* of Rose's* well-marked *indurated,* or infecting chancres of Ricord—which were treated without mercury—conformed to the law laid down by the latter authority; they *rapidly* lost their specificity, for they rapidly healed. But such evidence of the short duration of the specific pus-forming power of this chancre is overruled by the history of *thousands* of other cases. Guthrie narrates cases treated without mercury, where the specific period, as measured by the chancre not healing, extended over six, eight, ten, and in one case twenty-six weeks. To the same effect are the very numerous cases endorsed by Sir James M'Grigor and Sir William Franklin.† In 1940 chancres taken *indiscriminately*, and which healed without mercury, the average period was, without bubo, twenty-one days, with bubo, forty-five days; and in 2827 chancres, including a larger proportion of indurated sores treated with mercury, the average period for healing was, without bubo, thirty-three days, with bubo, fifty days; (being a difference, I may remark, adverse to the generally supposed efficacy of mercurial treatment).

During a *long* period, then, extending to weeks or months, chancre retains its specific pus-forming power; and coextensive therewith the inoculation test is available, so as to authorize our employing preventive measures against Constitutional Syphilis further than by local treatment—escharotics, which are successful only when used during the first four or five days after the birth of chancre. And the *slow-healing* power of a supposed chancre is itself corroborative proof of its nature. This character—signified by the absence of granulations — was long since observed by

---

* Med.-Chir. Trans., vol. viii., p. 558.

† Milit. Surg., Hennen, ed. ii., p. 545.

Hunter, and recognised by Colles,* as a prominent feature in his definition of true chancre.

A self-reproducing and slow, perhaps very slow, healing sore or ulcer, is, in point of fact, a chancre; and while the latter character corroborates our exact diagnosis, it also provides a sufficiently early opportunity for the employment of certain preventive measures, after the first opportunity has passed away. I allude particularly to the anticipation of Constitutional Syphilis by mercury.

It is not my present purpose to do more than advance the diagnosis of chancre, and etiology of its constitutional consequences up to this *preventive* point of view. What these consequences are, were described in Chapter VIII. The slow-healing power of the primary sore, and of an open bubo, somewhat suggest the prevailing character of Constitutional Syphilis. From the first moment of the pus-forming pimple, the whole career of syphilis is one of *disintegration* of the tissues, and an abortive effort of the reparative power. Disintegration, by desquamation or other destructive eruptions of the skin; disintegration by corroding ulceration of the tonsils, tongue, lips, palate, and perhaps the nose; disintegration by iritis, with puriform lymph; disintegration, by irreparable destruction of the texture of the testis; disintegration, by caries and necrosis. Premature baldness, fretting ulceration around the roots of the finger and toe-nails, and a wan *cachectic* pallor, bespeak the consummation of syphilitic decay. Yet the body crumbles in this way when saturated with mercury.

---

* Pract. Obs. on the Venereal Disease, 1837, p. 75.

CHAPTER XI.

LOCAL MAY PROCEED FROM LOCAL MORBID CONDITIONS; OR, THE RELATION OF DISEASES OF NUTRITION—*e.g.*, INFLAMMATION AND OF LOCAL NERVOUS AND MUSCULAR AFFECTIONS, RESPECTIVELY, TO THOSE OF PARTS (CONTINUOUS) CONTIGUOUS, AND REMOTELY SITUATED IN THE BODY.

The Pathological Principles which guide the Preventive view of some of these morbid conditions.

Pathological Anatomy applied, during life, to most early and exactly detect and discriminate the structural condition, situation, and extent of diseases (and injuries), regarded as internal causes, in order to the Prevention of their consequences,—by the extension of such local morbid conditions to textures and organs (continuous and) adjoining, and the sympathetic affections of organs remote; also the disorganizing results in the texture or part first affected. Illustrated by inflammatory diseases of the joints, thus considered.

Preventive Measures of some of these morbid conditions.

The Etiological Principle above stated is one of very wide signification. All diseases of Nutrition, by their progressive extension, are instances of local morbid conditions, inducing others in textures (continuous) contiguous, and in remote parts. The progress of inflammation, in whatever part of the body it may occur, and the course of inflammatory diseases of the joints particularly, affords a series of illustrations plainly marked and well defined, suggesting also the *general* Principle.

I shall therefore now trace the course of synovitis and scrofulous caries, respectively; and in doing so, the guidance of Pathological Anatomy during life, to the earliest and most exact detection and discrimination of 'internal causes' in operation, will be further illustrated, and consequently its immediate bearing on the Prevention of disease.

Each of the aforesaid diseases can be detected during life in

its earliest stage, and distinguished, by virtue of a certain pathologico-anatomical condition, *i. e.*, 'swelling' of a peculiar shape and size, and having other characters depending on the particular tissue affected.

These positive signs are, be it observed, quite independent of any symptoms of functional disturbance,—as peculiar pain, or inability to move the joint. Any such mere *symptoms* supervene too late in the course of these diseases, and are then too inconstant and unessential to avail much for diagnosis. The more early and exact signs presented to the surgeon by a careful manual examination and inspection of the joint affected, can alone guide him, in the earliest stage of synovitis, and at the earliest period when scrofulous caries can be detected and distinguished with certainty; and possessing *this* knowledge, he may most reasonably hope to prevent the consequences of these diseases;—ulceration of the adjoining cartilages, and suppuration with hectic fever; and, moreover, prevent the disorganizing results of synovitis and caries, by chronic thickening of the synovial membrane, or destruction of the bones forming the joint.

Observe commencing synovitis, say of the knee-joint. We discover a painful spot, situated perhaps at the inner edge of the patella. This spot gradually extends, and the pain spreads over the whole articulation. Very soon a *fluid* and *fluctuating* swelling supervenes, which being due to distension of the synovial capsule with serum and fluid lymph, is of corresponding shape and size. This swelling therefore is characteristic; and varying in form with each articulation, its shape is also somewhat modified by compression of the surrounding ligaments and tendons. This pathologico-anatomical character is found, by repeated dissection, to be connected with a certain state of the synovial membrane, as the primary seat of inflammation. If now the joint were opened, and this membrane exposed, we should discover its increased vascularity, of a more or less scarlet colour; and that possibly some blood had escaped tinging the serum and lymph effused within the capsule. At this *early* period, the synovial membrane still retains its usual

thinness and pliancy. It may however happen, that pus is subsequently poured into the joint, and concurrently with such effusion the cartilages lose their substance, as if ulcerated. Here then we distinctly trace the extension of pus-forming synovitis to disintegration of the neighbouring cartilages. The early recognition therefore of its characteristic swelling will afford *the* opportunity for removing primary synovitis; by availing ourselves of which, the secondary disease of an adjoining texture will assuredly be prevented.

This anticipation of disease through a sufficiently early detection of the internal cause in operation, is illustrated in like manner by the history of scrofulous caries; its practical application however to this disease is less satisfactory than in respect of synovitis: for the earliest structural changes in scrofulous caries being less apparent their progress is more insidious. Indeed, we do not at first observe any well-marked external sign of internal disorganization, and are only led by the equivocal evidence of functional symptoms, *e. g.*, pain and lameness, to suspect the destructive process going on within the bone. So far Pathology does more than Pathological Anatomy to aid, if not determine, our diagnosis of *incipient* caries: yet this assistance is but little. The pain in the head of the tibia, for instance, may at first be only trifling, perhaps intermittent, yet fixed; aggravated also on percussion of the joint, and increasing in severity as the disease progresses. If now the cancellated bone were examined, we should find its consistence softened, and the cancelli filled with reddish serum; but no further destructive change may as yet have ensued. Weeks, or perhaps months, may elapse before the characteristic white, *puffy*, and *elastic* swelling of soft tissues around the joint arrests our serious attention. At length, however, the globular shape, no less than the great enlargement of the whole articulation,—an appearance rendered even more conspicuous by the emaciation and semi-flexed position of the limb,—together corroborate our diagnosis; perhaps to be confirmed, only when too late, by the grating friction of the articular surfaces, now denuded of

their cartilages, and by undue mobility of the joint, when thus disorganized and loosened, or eventually the joint becomes dislocated and fixed. The consequences of scrofulous caries being so disastrous, its earliest detection and removal is the more imperative. Destruction of the articular cartilages is, in truth, more frequently consequent on caries than pus-forming synovitis; but respecting both these diseases, inflammation affecting the tissues of joints generally commences peripherally, and proceeds inwards to the deeper textures. The *primary* ulceration of articular cartilages is, in fact, a comparatively rare occurrence. Accordingly we discover some signs of previous inflammatory vascularity, either of the synovial membrane, or of the cancellated bone, adjoining the cartilage, subsequently involved.

If, then, ulceration of the articular cartilages is in most cases an event *secondary* to either caries or synovitis, obviously, by the earliest detection and removal of whichever disease be present, we may reasonably hope to prevent a consequence so fatally destructive of the articulation.

Now the earliest unequivocal sign of synovitis and caries is 'swelling,' and by the earliest recognition and interpretation of this sign we are assuredly guided to arrest the progress of inflammation. But such recognition and interpretation of swelling, implies a due knowledge of Pathological Anatomy, and of its application (during life) to diagnosis.

Thence is derived, both, the requisite diagnosis of synovitis and caries respectively, and the anticipation of (secondary) ulceration, consequent on the extension of inflammation to the *adjoining* cartilages. The value of this method of diagnosis, in relation to prevention, is further illustrated by the otherwise inevitable consequences of the same local diseases, in respect of *remote* organs and functions.

For, concurrently with ulceration from caries, and more rarely from synovitis, certain general consequences arise. The heart beats feebly, or at least irregularly, and the pulse is weak and fickle, the respiration is hurried; the organs of digestion soon

sympathize, for the appetite fails and diarrhœa ensues, while the skin pours forth abundantly the night-sweats of hectic fever, and the urine deposits its lithates as the body wastes and the strength declines. The sufficiently early detection and removal of either cause in operation prior to suppuration, would assuredly have alike anticipated, not only adjoining structural disease,—ulceration of the cartilages, but also these disturbances of remote functions.

In like manner we may anticipate destructive *results* in those tissues where the disease originated. Results should be distinguished from consequences. A consequence is that which follows after, and on account of, a previous condition which may itself have disappeared. A result is not merely an effect, but may be considered as the final issue, or last stage, of a condition itself persistent. If thus the progress of acute synovitis be not speedily arrested, this condition will almost invariably terminate in chronic *thickening* and *induration* of the capsule. Again, if scrofulous caries run its course, then the cancellated bone *softens* and *disintegrates,* the reddish serum within the cancelli is replaced by an albuminous granular cheesy substance, while neighbouring cartilages disappear, and the joint collapses. In either case the function of the *part affected* is *irreparably* sacrificed, whether that of the synovial membrane now thickened and inflexible, or that of the articulation itself from the total and permanent disorganization of chronic caries. This eventual *result,*—irreparable loss of function, might certainly have been anticipated by a sufficiently early detection of its structural cause.

The *primary* ulceration of articular cartilages is singularly insidious, a fact which imparts peculiar importance and interest to the *earliest* possible *detection* and *discrimination* of this condition. But as if to verify the guidance of Pathological Anatomy, during life, we observe the incompetency of mere Functional symptoms, such as pain, to determine our diagnosis. If sufficiently early, it cannot be exactly determined thereby.

The commencement of primary ulceration of the articular

cartilages is announced by few if any external signs of incipient disorganization.

Pathology does more for our diagnosis at *this* time than for that of caries. By 'pain,' fixed and intense, aggravated on friction (rather than on percussion) of the cartilages, and increasing as the disease progresses, an early alarm is given. If now the cartilages were seen, we should find a portion to be vascular, unlike its healthy condition, and we might discover a softened spot, but no loss of substance may as yet have ensued. Weeks, or perhaps months, pass away before any swelling supervenes, and then so trifling an effusion into the cellular tissue around only the margins of the cartilage affected, that but for the characteristic shape of this swelling—corresponding as it does with the boundary of the cartilage, and moreover, the total absence of fluctuation and elasticity peculiar to the swelling of synovitis ; we might not even then refer such trivial external appearances to the grave and irreparable injury which the joint has now undergone. Textural disintegration of the cartilage has resulted in its loss of substance, and the gliding motion of smooth cartilage is exchanged for the grating attrition of denuded bone. Our only hope is anchylosis, or perchance the formation of a substitute-membrane, or the porcellanous transformation of the articular surface of the bone itself. The swelling is more obvious when the cartilages of *both* articular surfaces have undergone ulceration.

Here, then, most emphatically, by detecting and over-ruling the earliest structural change, perhaps mere vascularity, we should be enabled to prevent, not only the impaired action of the joint coincident with ulceration, but also the irreparable loss of function contingent on anchylosis.

To fulfil this intention, the characteristic swelling, supervening only in the advanced stage of primary ulceration, is the most exact ground of diagnosis, but comes too late for the purpose of prevention ; and the pain, although more early, is not sufficiently characteristic.

The superior diagnostic value of Pathological Anatomy by

signs—such as swelling—is thus well illustrated by inflammatory diseases of the joints. It supplies the most exact and earliest *reliable* method of diagnosis: hence the immediate relation of this method to the prevention of the consequences, and results of diseases.

Turning again to inflammation as a local cause of local disease by its own extension to textures, continuous and contiguous, we observe this *Etiological* Principle further illustrated by the course of inflammation in *all* parts of the body. The joints furnish, certainly, well-marked examples, but similar extension is illustrated by the supervention of ostitis on periostitis, by cellulitis consequent on erysipelatous inflammation of the skin, and, conversely, inflammation of the skin occurring after primary cellulitis. Taking internal organs, from the head downwards; meningitis is followed by cerebritis, scrofulous and purulent ophthalmia, by inflammation and destruction of the cornea and deeper textures of the eye; laryngitis, by inflammation of the subcellular texture and œdema glottidis; gastritis and enteritis respectively, by extension of the inflammation in bad cases to the submucous cellular and muscular textures, and even to the peritoneum, giving rise to peritonitis. Cystitis and metritis may each have a similar course, the latter, more especially in puerperal cases, extending to the peritoneum—puerperal peritonitis. Lastly, by way of illustration, I may mention orchitis supervening on gonorrhœa, in which event probably the primary inflammation travels along the course of the vas-deferens.

Thus, then, any part of the body may become the seat of *secondary* inflammation. So also nervous and muscular affections may arise from causes more or less remote from the scene of their manifestations; and *these* secondary affections are liable to occur in almost any part of the body.

I shall now take a systematic view of *sympathetic* affections, nervous and muscular, as they emanate from or are exhibited by organs and in regions, from the head downwards. Most of my illustrations are collated from the valuable writings of Dr. Whytt

on this subject; others, from the works of Abernethy and Brodie; and some instances of my own observation are interspersed.

Beginning with the head: violent pains in the head that have their seat most commonly in the membranes of the brain or pericranium are frequently attended with nausea and vomiting. The "*spasmus cynicus*," locking of the jaws, and a universal tetanus followed a wound of the left side of the head, by which the temporal muscle was divided. Light and noise are offensive to the eyes and ears respectively in severe headaches. Wounds and contusions of the brain generally occasion bilious vomitings. Certain impressions made on the "*sensorium commune*" by external objects instantly impart to the eyes either a dull, a lively, or a fierce look. Grief, vexation, or fear lessens the secretion of the saliva, destroys the appetite, and is apt to occasion diarrhœa. The great consent between the brain and heart appears from the sudden and remarkable effects of the passions on the latter organ.

The Eyes.—When one eye is affected with inflammation, a cataract, or the *gutta serena*, the other is soon after attacked with the same disease. Contraction of the pupil is not owing to light acting as a stimulus on the iris, but solely to the sympathy between it and the retina. Sympathy between the pupils is so intimate that even in gutta serena the pupil of the morbid eye follows the motions of the sound one. We shut both eyelids involuntarily whenever anything threatens to hurt either eye. A bright light coming suddenly on the eyes sometimes excites sneezing. The sight of savoury food occasions a flow of saliva in a hungry person. Yawning and vomiting are often catching.

The Ears.—The noise of a file and other harsh sounds affect the teeth with an uneasy sensation. The whetting of a knife has caused the gums to bleed. Great and unexpected sounds, such as the explosion of a cannon or musket, make us instantly close our eyelids. As the ear is frequently pained when the fauces are inflamed, so irritation of the *meatus auditorius* will often excite coughing and sometimes vomiting. A constant pain of one side

of the head, with numbness of the left arm and leg, suppression of the menses, and epileptic fits have all been occasioned by a glass ball, not larger than a pea, sticking in the ear.

The Nose.—The effluvium of 'Hungary-water,' or spirit of wine, drawn strongly into the nostrils, increases the flow of the saliva into the mouth, and sometimes stops a tickling cough. The smell of savoury food to one who is hungry immediately induces a flow of saliva. Sternutatories not only increase the secretion from the nose, but also from the lachrymal vessels. After smelling volatile salts, or eating too much strong mustard, pain is often felt above the eyebrows; and again, after taking a large draught of cold water in winter, that part of the forehead just above the nose is affected with a painful sensation. Acrid substances applied to the olfactory nerves affect the diaphragm, intercostal and abdominal muscles, with convulsive motions.

Boyle* mentions several instances of persons being purged by smelling a cathartic medicine, and that in some this effect failed to ensue when from coryza the olfactory nerves had lost their power of distinguishing smells.

The Teeth.—A carious tooth will sometimes occasion violent pain in a sound one, though at a distance from it; and the pain ceases as soon as the bad tooth is drawn or its nerve destroyed. Pain in the teeth often affects the cheek-bone, one side of the head, the throat, and the corresponding ear. Children, from irritation of the gums in teething, are liable to vomiting, purging, cough, fever, and convulsions.

The Heart.—Fatty degeneration of this organ, associated with calcareous deposit in the walls of the coronary arteries, may, from time to time, induce anginal pain, radiating to the left shoulder and arm.

The Trachea.—Irritation of the windpipe, or any of its small branches, excites coughing, or convulsive motion of the muscles employed in expiration; nausea, vomiting, and convulsions are

* Usefulness of Experimental Philosophy, part ii., p. 242.

sometimes the consequence of violent or long-continued irritation of these parts.

The Lungs.—The sympathy of the lungs with the diaphragm and intercostal muscles is manifested by their motion, even in ordinary respiration; still more so by the laborious breathing which always accompanies insufficient transmission of the blood through the pulmonary vessels.

The Diaphragm.—When the diaphragm is inflamed, the stomach, brain, and muscles of the face are affected sympathetically, as manifested by the delirium, vomiting, and *risus sardonicus* attending this disease.

The Stomach and Intestines.—A disordered state of the stomach and intestines, with wind or vitiated secretions lodged in them, will sometimes so affect the brain as to deprive the individual of reason; at other times producing vertigo, "*cephalæa*," hemicrania, *clavus hystericus*, palpitations, intermissions of the pulse, difficult breathing, sudden flushings of heat, and sweating, &c. By hard drinking, or a large dose of opium, the eyes lose their lustre. The headache following a debauch proceeds chiefly from the stomach, as appears by the removal of the pain upon drinking a few glasses of strong wine. Disorder of the stomach will sometimes occasion dimness of sight. Acidity of the stomach is apt to occasion painful sensations in distant parts—*e. g.*, frontal headache. Eating ice may have the same effect, or induce pain in some other remote part. The late Dr. Woollaston ate some ice-cream after dinner. He soon became lame from a violent pain in one ankle. Suddenly he turned sick, and vomited the ice-cream, when the pain in the foot and lameness instantly ceased.

Convulsive action of the stomach and intestines often spreads to the throat, occasioning difficulty of breathing and a sense of suffocation. On the other hand, irritation of the fauces or pharynx excites vomiting. Nausea or a disagreeable sensation in the stomach makes the pulse quicker and smaller, brings on a sweat, and sometimes greatly increases the secretion of saliva or urine. When the stomach is empty, and affected with hunger,

the saliva flows much more copiously into the mouth than after a full meal, or when the natural appetite for food is wanting. Inflammation of the stomach and intestines is attended, at its commencement, with shivering of the whole body, and great coldness of the hands and feet. Long-continued vomiting and purging occasion violent cramps of the muscles of the legs and thighs. A tremor of the hands is often lessened or removed for a while by a dram, and this effect is owing solely to its action on the stomach prior to the spirit entering the blood, which does not happen so soon. The particular sympathy of the stomach with the diaphragm and abdominal muscles appears from their convulsive actions in vomiting and hiccup. Dyspepsia is sometimes accompanied with pain in the left shoulder-blade. Gastrodynia is often attended with pain over the whole chest. A violent spasmodic pain in the stomach often renders the pulse much slower than in health.

Inflammation of the intestines is frequently attended with vomiting and suppression of urine. Opisthotonos, or tetanus, is often occasioned in hot climates by retention of the meconium in the bowels of infants. Itching of the nose is a common sign of worms. Irritation of the intestinal canal, as in colic and cholera, is apt to cause pain in the calves of the legs. Worms in the rectum, or other irritation there, excite itching of the anus and scrotum, or of the pudenda in females.

The Liver.—Biliary calculi irritating the ducts frequently occasion nausea and vomiting. Inflammation or congestion of the liver is generally accompanied with vomiting and hiccup, and often with pain in the right shoulder-blade. From suppuration of the liver two patients experienced numbness and debility of the right arm, thigh, and leg.

Abernethy's observations respecting what *he* regarded as the 'Constitutional origin of Local diseases,' had reference to local orders arising from derangement of the *digestive organs*, and especially the *liver*. Dyspepsia and defective or imperfect secretion of bile were designated Constitutional causes, from whence

proceed numerous affections of organs and parts, perhaps remotely situated in the body. These causes are, I think, more correctly speaking, *local* (causes), and the said affections of other organs so many illustrations of local disorders emanating from them. Thus, in accordance with the Etiological Principle advanced in this chapter, I would interpret Abernethy's cases.* They are as follow :—

Nervous and muscular disorders may arise from derangement of the digestive organs. Injuries of the head are liable to be maintained and aggravated by derangement of the digestive organs. Certain unhealthy indurations, abscesses, and sores, bespeak "a combination of nervous irritability and weakness;" but they are seldom, if ever, unattended with disorder of the digestive organs. Carbuncle and scrofula denote a similar condition of the system in general, but their exciting cause is some derangement of these organs. Diseases of various glands—*e.g.*, the testicle and breast—may have a similar origin. Disorders of parts which have a continuity of surface with the alimentary canal are apt to arise in like manner.

Having traced various local affections to derangements of the digestive organs, Abernethy takes a converse view of this law, and shows the causative relation of local diseases to *his* (so-called) constitutional morbid conditions. Thus, sudden and violent local irritation will sometimes produce an equally sudden and vehement disorder of the digestive organs; and a slighter degree of continued local irritation will produce a less violent disorder of these organs.

Next in order to hepatic manifestations, Whytt notices those sympathetic affections which are induced by diseases of the kidneys and ureters. Nausea, vomiting, costiveness, and inflation of the bowels are often due to inflammation of the kidneys, or to calculi in the ureters. A stone in the pelvis of the kidney, or in the ureter, sometimes occasions a frequent inclination to pass water,

* Surgical Observations on the Constitutional Origin and Treatment of Local Diseases, 1824.

and heat in the extremity of the urethra. When one of the kidneys is inflamed, little urine is secreted by the other. When a stone is passing through the ureter, the testicle of the same side is sometimes drawn upwards and swells, and an erect posture is then painful.

The Bladder and Rectum.—Irritation of the neck of the bladder, or extremity of the rectum, causes constant contraction of the diaphragm and abdominal muscles. Strangury and tenesmus are reciprocally related as cause and effect. The pain of hæmorrhoids is sometimes accompanied with sickness and faintness. A stone or ulcer in the bladder is attended with a sharp pain near the end of the urethra, especially after making water. "I had," says Whytt, "a patient with ulcer of the bladder, who, when he passed urine, not only experienced violent pain in the point of the penis, but down the thighs and legs, even to the soles of his feet, as if he were standing barefooted on burning coals."

Irritability of the urinary bladder arises from many *local* causes, which (no less than the Constitutional) I have elsewhere examined in detail.* It may proceed from habitual constipation, and from various diseases of the rectum—hæmorrhoids, fissure in ano, inflammation of the rectum, stricture, cancer, abscess about the rectum, and fistula in ano. Displacements and diseases of the uterus and vagina are possible causes of vesical irritability. Prolapsus uteri or vaginæ, acute metritis, cancer, and fibrous tumours of the uterus, also belong to the uterine class of local causes. Stricture of the urethra—whether organic, spasmodic, or inflammatory—is a frequent cause of this irritability, chiefly by retention of urine. Diseases of the prostate gland operate in like manner. They mechanically occasion partial retention—the urine collecting in the lower fundus of the bladder, behind the projecting prostate—while the diseased gland further excites vesical irritability. Acute inflammation and chronic enlargement of the prostate, each has this twofold operation, especially inflammatory enlargement.

---

* The Irritable Bladder: its Causes and Curative Treatment.

Lastly, diseases of the bladder—cystitis, cancer, a calculus loose or encysted, are local causes of its own irritability; but, these morbid conditions being identified with the organ affected, can scarcely be regarded as appropriate illustrations of the Etiological Principle I am advocating.

The Genitals in Men.—At the time of puberty, not only the voice, but the whole body undergoes a sensible change, which is probably owing to the stimulus communicated to the nerves of the genital parts by the semen; for we certainly know that other stimuli applied to the nerves of the nose or stomach, according to their nature, will either instantaneously impart new vigour to the whole body, or soon occasion a general stupor and debility. By sympathy with the glans penis the vesiculæ seminales are contracted in time of coition, and when the lower part of the urethra is stimulated by the semen, the acceleratores urinæ are excited to convulsive action.

The Uterus.—Some of the symptoms of hysteria are expressions of nervous sympathy with the womb; but the most obvious expressions of this sympathy are those elicited by pregnancy. From the moment of conception the uterus announces to every part of the body the changes that are going on within itself. At all times a vascular organ and plentifully supplied with nerves, the gravid uterus becomes more vascular, and *perhaps* its nerves enlarge: possessing these vital endowments, the uterus communicates through the nervous and vascular systems with organs most distantly removed from its own locality. A slight rigor, or else a feeling of faintness, may be the earliest constitutional symptom of pregnancy; and a slight febrile paroxysm may ensue: very soon every part of the body responds to the vital activity of the uterus. So all-pervading indeed are the nervous endowments of the gravid uterus, that in this respect it is almost equivalent to a second brain, influencing and regulating the most distant organs during the whole period of pregnancy. Headache frequently supervenes. The natural temper and disposition of the woman may be altogether changed: the gentle and subdued become irritable, and

the melancholy cheerful. Severe pains dart about the body, now affecting a single tooth, possibly itself sound, and anon settling for a time in the ear and face, or shooting through the breasts. The heart not unfrequently palpitates and distresses, while a troublesome spasmodic cough announces the sympathy of the respiratory organs. Very shortly the digestive organs sympathize. The most extraordinary perversions of taste may occur, with a desire for cheese, pickles, or even for chalk and other unnatural substances. The mouth sometimes overflows with saliva, and the well-known nausea and morning sickness of pregnancy express the intimate sympathy of the stomach. No less are the intestines engaged, for a troublesome and perchance dangerous diarrhœa is a not unfrequent complaint. And lastly, there is more or less irritability of the urinary bladder.

About the time of menstruation, headache, pain in the back and bowels, &c., arise, ceasing as the uterus is relieved and the menstrual discharge passes off. The breasts enlarge and become tender at the menstrual period, and subside again when it is over.

The Extremities.—Straight shoes give some people a headache; while sinapisms applied to the soles of the feet, or blisters to the legs, often lessen, and possibly remove, delirium. In obstinate constipation, cold water dashed on the feet and legs has sometimes excited peristaltic action of the bowels, after many internal remedies have failed. Tickling the soles of the feet excites the muscles of the whole body to convulsive action. Tetanus may be occasioned by a wound in the sole of the foot,—as by a nail driven into that part.

The following noteworthy cases of nervous and muscular affections pertaining to the extremities occurred in the practice of Sir B. Brodie.* The Principles of Prevention are implied thereby, and appropriate Preventive measures also.

A man complained of a severe pain at the inner side of his knee. The joint was carefully examined, but no marks of disease

* Local Nervous Affections, 1837.

could be detected. In the thigh, however, an aneurism of the femoral artery was perceptible, the size of a small orange, which had scarcely attracted the patient's notice. Sir Everard Home applied a ligature round the femoral, in the upper part of the thigh. Immediately the tumour ceased to pulsate, and the pain in the knee ceased also. The patient died about four or five days after the operation. On dissecting the limb, the aneurism was found reduced to one-half of its former size; some branches of the anterior crural nerve which passed over it, and which must have been kept on the stretch previous to the operation, were found to terminate in the part to which the pain had been referred,—on the inner side of the knee; and thus the cause thereof was sufficiently explained. It was, in fact, a sympathetic pain, without any disease of the part affected, and due to pressure on the nerves above. No application to the part itself would have been of the slightest avail while the cause (above) continued in operation.

An analogous case is recorded by Dr. Denmark.* A sailor was shot in the arm; the wound healed, yet the man complained of an agonizing pain, beginning in the extremities of the thumb and fingers, except the little one, and extending up the fore-arm. Amputation gave complete relief; for, on dissecting the limb, a small portion of lead, apparently detached from the ball when it had struck against the bone, was found imbedded in the fibres of the radial nerve.

In each of these cases, the cause of irritation (observes Brodie) was detected in the *trunk* of the nerve belonging to the part to which the symptoms were referred. Similar effects are produced when the actual seat of the disease is that more essential part of the nervous system in which the nerve itself originates,—*i.e.*, the *brain* or *spinal cord*. Thus, caries of the dorsal vertebræ, irritating the spinal cord, produces pains and muscular spasms of the lower limbs; and the same disease affecting the superior cervical vertebræ, produces corresponding symptoms in the upper limbs.

---

* Med.-Chir. Trans., vol. iv., p. 51.

Sympathetic affections may be due to an impression transmitted from one nerve to another through the medium of the central nervous axis. Such are instances of *reflected* sympathy. Nerves connected together by a plexiform arrangement more especially share each other's woes. Pain induced by irritation of one nerve so connected, will probably be referred to others proceeding from the same plexus.

A gentleman laboured under a scrofulous disease of the hip, producing caries of the bones and suppuration within the joint. The following symptoms were presented, in addition to those usually exhibited :—The slightest motion of the thigh brought on an attack of excruciating pain amounting to agony, attended with violent spasmodic contraction of the muscles which move the thigh. The limb was jerked in a most remarkable manner for several minutes, and the volition of the patient had no control over these distressing and extraordinary movements. After some time a tumour began to show itself externally on the anterior part of the thigh, raising the femoral artery, which lay pulsating on its surface. At length the patient died,—the attacks of pain and spasm having ceased for six or eight weeks before this event took place. Brodie examined the diseased hip and parts connected with the greatest care. The articular portions of bone were much softened, the cancelli contained a yellow "cheesy matter," and the cartilages had been destroyed by ulceration. The 'tumour' was abscess situated among the muscles of the thigh on the anterior part below the hip-joint, but communicating with it. Two lymphatic glands, enlarged to the size of large walnuts, were found beneath the skin on the anterior part of the thigh, below the outer extremity of Poupart's ligament. It so happened that a considerable branch of the lumbar nerves lay over either of these enlarged glands, being thus kept stretched and tense, as the strings of a violin are stretched over the bridge of the instrument. These nerves had the same origin with those supplying the muscles on the anterior and inner part of the thigh; their tension, therefore, sufficiently explained the peculiar symptoms in this case.

Nerves not connected together, being nevertheless connected through the medium of the central nervous axis, express sympathy between *distant* parts of the body. Consequently, sympathetic affections may arise from internal causes, remote from the parts wherein their operation is thus declared.

A gentleman complained of pain in one instep. The pain was severe, causing lameness; but neither swelling, nor, except the pain, any sign of inflammation. Sir B. Brodie prescribed some remedies, which were of no avail. One morning the patient called, still suffering from the pain in the foot, and so lame that he could not get out of his carriage and walk into the house without the assistance of his servant. Now, however, he complained of another symptom,—a difficulty in making water, and a purulent discharge from the urethra. He had laboured under a stricture of the urethra for many years, and had occasionally used bougies. Of late the stricture had caused more inconvenience than usual. A bougie was introduced, which penetrated the stricture and entered the bladder. Immediately the pain in the foot abated, and in less than a quarter of an hour he left the house free from pain, and walking without the slightest difficulty. Since then, whenever the pain in the foot returned, nothing but the introduction of a bougie relieved it.

An analogous case is related by the same author. A lady was subject to attacks of severe pain, beginning in the left ankle, extending along the instep towards the little toe, and into the sole of the foot, without any swelling or tenderness of the skin. The pain was due to internal piles; for in proportion as they were reduced by appropriate treatment, it abated.

The foregoing series of sympathetic affections—nervous and muscular—further illustrate the (first) Etiological Principle advanced in this chapter;—that local morbid conditions may proceed from *local* internal causes.

In respect of inflammatory diseases, they proceed from the direct extension of inflammation, continuously through the texture originally affected, to contiguous textures.

In respect of nervous and muscular affections thus arising, the nervous system is the medium of sympathetic connexion, and they are manifested in parts more or less remote from the causes themselves.

But whether exhibited in parts contiguous or remote, the prevention of the secondary condition, whatever it be, obviously presupposes a sufficiently early and exact detection and discrimination or diagnosis of the primary local morbid condition, as the cause in operation.

CONSTITUTIONAL MAY PROCEED FROM CONSTITUTIONAL MORBID CONDITIONS; OR, THE RECIPROCAL RELATION OF MORBID CONDITIONS OF THE NERVOUS AND VASCULAR SYSTEMS.

General Theory of Fever propounded by reviewing Constitutional Irritation—shock with reaction, as compared with Inflammatory fever, Eruptive fevers, and Endemic fever—intermittent and remittent.

The Pathological Principles which guide the Preventive view of these Diseases, concluded.

This, as usual, presupposes a sufficiently early Diagnosis.

Preventive Measures, concluded.

On taking an analytical review of the three Etiological Principles already considered, a more comprehensive generalization than either appears;—that the *blood* and *nervous system* are essentially concerned; either as sources from whence morbid conditions emanate, or as centres to which they converge; or else that these systemic textures, for so they may be regarded, are the media of extension and transmission, on the part of morbid conditions.

Thus, local diseases proceed from morbid conditions of the blood or nervous centres,—as constitutional causes; or, conversely, are themselves the causes of constitutional morbid conditions of the blood or nervous system; lastly, these systemic textures are the media whereby morbid conditions are extended and transmitted, respectively, to parts contiguous or remote.

These, then, are the *elements* to which such Etiological Prin-

ciples can be reduced; these the pivots, so to speak, on which the Principles of *internal* Etiology turn.

Pursuing the generalization, there yet remains for consideration the *mutual relation* of Constitutional morbid conditions,—*i.e.*, those of the blood and nervous system. Their relation will be at once perceived by reference to the subject-matter of the foregoing chapters. I need only make a brief recapitulation in the manner most suitable to my present purpose.

Observe the simplest and most obvious instance of a Constitutional morbid condition,—that in which the *nervous system* is primarily affected, as by the 'shock' of injury. It is one of nervous prostration, more or less marked. The blood's circulation is simultaneously arrested through failure of the heart's action; but *this* constitutional condition is much *less* pronounced,—there being only some loss of consciousness by *direct* exhaustion of the nervous energy, not the unconsciousness which accompanies cardiac syncope. Unlike that condition also, the reaction, denoted by a stronger and fuller pulse, is not necessarily accompanied with restoration of the nervous energy. The individual prostrated by the shock of injury, may still lie prostrated, or become restless and irritable only, with the returning circulation. 'Shock' therefore signifies failure of the nervous energy; the nervous system is *primarily* and *predominantly* affected, and exhibits this constitutional morbid condition.

Passing on to 'inflammatory fever;' it would appear that this fever primarily signifies general excitement of the nervous system, consequent on the local inflammation; and that the *blood's circulation* becomes *secondarily* affected; although this latter condition represents the *predominant* constitutional disturbance.

In both shock and inflammatory fever, the nervous system is *first* affected, and thence the sanguiferous system. The morbid (constitutional) condition of the latter proceeds from the former. So far shock and inflammatory fever are *analogous*, both denoting an impression, although of an opposite character, made on the *nervous system*, and thence continued to the sanguiferous

system; but, in respect of the former constitutional condition, the nervous system is predominantly affected; while in respect of inflammatory fever, the blood's circulation represents the predominant constitutional disturbance. Shock with imperfect reaction, lasting continuously, together represent 'constitutional irritation,' which thus contrasts with 'inflammatory fever.'

On the other hand, 'hectic' and 'gangrenous typhoid' fevers would appear to begin each as a morbid condition of the blood by absorption of poisonous matter, which thence engages the nervous system. In both *these* fevers the *blood* is *primarily* affected.

Comparing their pathology with that of inflammatory fever, we are led to conclude that the blood and nervous system *mutually* stand in the relation of cause and effect to each other, in respect of their morbid conditions; and that Fever may arise by perturbation either of the blood and its circulation, or of the nervous system; although, when established, both are engaged. Fever is therefore a *compound* constitutional morbid condition; its *essential* constituents being represented by the blood *and* nervous system: and when we consider how generally these systemic textures are distributed, as the components of organs, throughout the body, we are not surprised at the extent and variety of that series of functional disturbances which collectively represent the subordinate *symptoms* of Fever.

Other fevers, besides those I have mentioned, have their origin in the *blood*. The 'eruptive fevers' begin essentially as blood-diseases. The nervous system is secondarily affected, although apparently simultaneously. This order of succession is clearly shown by the phenomena which accompany the course of these fevers. During the *incubatory* period, the blood is silently undergoing its own peculiar morbid changes, and at that time the symptoms of nervous depression are far less predominant than when 'febrile oppression' supervenes, which denotes the engagement of the nervous system, and announces the accession of fever. The various eruptive fevers—typhus, typhoid, and relapsing fevers, measles, scarlatina, small-pox, chicken-pox,

erysipelas, and plague—are therefore severally 'blood-diseases,' *essentially* and *primarily*, although the nervous system soon becomes engaged.

Guided by this analysis of the pathology of these fevers, their phenomena can be interpreted.

The blood is to the various textures of the body what the soil is to the products of vegetation. And as the blood's composition and properties are primarily perverted in exanthematous fevers, so likewise are the products of textural nutrition abnormal. Hence, the peculiar 'eruptions' which overspread the skin, and the inflammatory affections of the mucous membranes.

The nervous system, in turn, exhibits its peculiar phenomena. While the morbid blood-condition is yet brooding, they are less definitively expressed. Lassitude, weariness, restlessness, wandering pains, especially in the back and limbs, thirst, inappetency, nausea or vomiting, headache, troubled dreams, and even delirium; these and similar symptoms bespeak some exhaustion, or exhausting excitement of the nervous energy; but, as the blood-condition becomes more predominant, the nervous system is proportionately subjugated, and acknowledges its subjugation, through the muscular system, by 'rigors' of longer or shorter duration. Thus the first avowed declaration of fever; and, the blood-condition still gaining supremacy, this subjugation of the nervous system is acknowledged by exhaustion more profound. Against such oppression the heart struggles, by restoring the circulation, to eliminate the blood-poison, in the appointed order of Nature. The fever runs its course; and if 'reaction' be victorious, the disease is brought to a successful issue. If 'febrile oppression' be overwhelming, the blood-poison is then so overpowering as to at once extinguish the nervous energy. Hence 'malignant' measles, 'malignant' scarlatina, 'malignant' small-pox, &c., are emblems of death.

This remarkable sympathy of the nervous system with a primarily morbid condition of the blood, giving rise to *compound* constitutional disease, is the pathology of other 'infectious' diseases.

In hooping-cough, influenza, and cholera, respectively, the nervous system soon concurs with the blood-condition; and thus the phenomena which characterize these diseases are *partly* of a *nervous* character; witness hooping-*cough*, and the fearful *exhaustion* of influenza and cholera. In like manner other essentially blood-diseases, *not* of infectious origin, assume this compound character; as shown by the intense nervous depression accompanying cellulitis and carbuncle, and by the peculiar premonitory symptoms of gout.

Respecting certain blood-diseases, represented by an excess of lithic acid, phosphoric acid, oxalic acid, or sugar, in the blood; and those that are occasioned by the retention of excrementitious matters, as urea, sweat, or hydro-carbonaceous matters—normally eliminated by the liver or lungs;—the symptoms are *partly* referrible to the nervous system.

And respecting those 'contagious' diseases which arise from the introduction of poisonous matters into the blood; the nervous system, sooner or later, becomes engaged: witness the phenomena of hydrophobia, and the kind of constitutional disturbance induced by snake-bites, by malignant pustule, by glanders, and by hospital gangrene.

If, then, in the course of most blood-diseases, the nervous system becomes affected, and these two constitutional elements be associated, forming essentially the same pathology, what is the *distinctive* pathology of Fever?

Obviously, the order in which its constituent elements arise is immaterial. The nervous system, probably, is primarily affected in the development of inflammatory fever; the blood, probably, takes precedence in all other fevers. The distinctive characteristic of Fever would appear to be this:—the *reactionary* operation of the heart, and consequent acceleration, &c. of the blood's circulation. The whole sanguiferous system manifests, by characteristic pulsations of the arterial vessels, this increased rapidity, and possibly the increased force of the heart's action; its regularity or irregularity also of action, and the tension of the vessels themselves.

But if all the phenomena I have now adverted to—nervous and vascular—are due to the altered *quality* of the blood in Fever, the symptoms of this compound constitutional condition are *further* complicated by the altered *quantity* of blood proportionately distributed to the various textures and organs throughout the body. The balance of the blood's circulation is disturbed, and some parts receive more, some less, than their healthy proportionate average quantity. Now, the quantity as well as the quality of blood proper to each part lies at the root of all structural maintenance and of every functional manifestation. Consequently, in Fever the whole of this economy must necessarily be perverted. Besides the leading phenomena immediately connected with the sanguiferous and nervous systems, other subordinate symptoms arise.

The circulation being accelerated, the whole mass of blood is more frequently passed through the lungs; and as the respiratory movements are also accelerated, the whole blood is more aerated than in health. The temperature of the body is increased proportionately; hence, 'heat' and 'fever' are indissolubly associated. The absolute increase of temperature varies from 4 to 6, or even 8 degrees Fah., compared with the average healthy standard, 98 degrees Fah. Some increment of heat is a constant and peculiar symptom of fever; yet it is only a symptom, and subordinate to acceleration of the blood's circulation. 'Secretion' being dependent, as is this function, on the quality and quantity of blood supplied to the various organs thus endowed, and regulated by the nervous system, the secretions themselves undergo corresponding changes in respect of their quality and quantity. The sweat and urine are *most* evidently affected. The excretory secretions are sometimes more abundant, sometimes less so, than in health.*

The diminished or increased amount of sweat-excretion should be considered in estimating the increment of heat actually generated by fever. Copious perspiration cools the body by evaporation, although much heat may have been generated; while a much

* Pyrexia—Gulstonian Lectures, E. Parkes, M.D., Med. Times and Gazette 1855, vol. xxxi.

smaller increment will be more perceptible when the perspiration is suppressed and the skin dry. A *dry*, hot, or burning skin, therefore, apparently symptomatic and expressive of Fever, is no indication of a raging fire within. The actual blood-heat can only be measured by the thermometer placed within one of the internal cavities of the body—as the mouth, or, as in Hunter's experiments (p. 456), in the vagina or rectum.

To sum up the leading results of this analysis, I would define Fever to represent a compound constitutional disturbance, of which the essential elements are phenomena referrible to the *blood* and its circulation, and to the *nervous system;* either of which may be primarily affected, but *generally* the blood first: and that the subordinate symptoms of this disturbance are an increased production of heat and altered secretions. When a patient has headache and prostration, a rapid pulse, a hot skin, and secretions vitiated in respect of quality, quantity, or both, then I would say he has Fever of some kind or other.

In further illustration of this theory, I may refer to the phenomena of Endemic or Malarious fever—*i.e.*, intermittent, remittent, and yellow fevers. They are essentially and primarily blood-diseases, by which the nervous system becomes engaged; in each also the production of heat is remarkable, and alterations of the secretions characteristically displayed. These fevers may run their course without any apparent alteration of structure being induced; and the patient often dies from the severest forms, with scarcely a trace of discoverable disease. In their milder forms, however, a greater number of tissues and organs are disorganized than perhaps in any other disease. Thus, the spleen, liver, lungs, heart, brain, the mucous and serous membranes, severally become permanent records of the all-prevailing blood-disease which has been at work.

Intermittent fever or ague is distinguished by febrile phenomena, occurring in *paroxysms*, and observing a certain *regular succession*, characterized by coldness, heat, and cutaneous discharge, which prove a temporary crisis and foretel a remission.

These phenomena are developed in an uninterrupted series or succession, more or less regular, and passing into each other by insensible steps (Aitken).

Remittent fever is distinguished by febrile phenomena with *exacerbations* and *remissions*. This fever is characterized by great intensity of headache, the pain darting with a sense of tension across the forehead. The symptoms rise and fall in daily succeeding paroxysms, representing a stage of remission and a stage of exacerbation (Aitken). Yellow fever is simply remittent fever accompanied by jaundice (R. Williams).

Distinguished, however, as these fevers are—the one by paroxysms with distinct intermissions, the other by exacerbations with remissions only—they nevertheless alike conform to all other fevers, in the *kind* of phenomena by which they are manifested; these phenomena being referrible to the blood and nervous system, as the constitutional elements from whence they proceed. Beyond analysis thus far, I am not disposed to venture at present.

The essential pathology of Fever is referred by some authors to an 'increased destructive metamorphosis of tissue.' Such is Virchow's theory :—

"Fever consists essentially in elevation of temperature, which must arise from an increased tissue-change, and have its immediate cause in alterations of the nervous system."

A critical survey of this theory has been made by Dr. Parkes,* who arrived at the following conclusions respecting "the various influences which seem to be active in Fever, and by the combined effect of which its complex phenomena may be supposed to be produced.

"Firstly. The entrance into the blood of a morbific agent, and the alteration of the blood to a certain extent under its influence. Perhaps this occurs during the incubative period, when often there is no rise of temperature, *i.e.* no fever, and when no appreciable alteration of the general health can be discovered. The nature of the change in the blood is unknown.

---

* Gulstonian Lectures; Lecture iii.

"Secondly. When the change in the blood has reached a certain point, the nervous system, or rather that part especially connected with nutrition and organic contractility, begins to undergo changes in composition, which probably impede or destroy the normal molecular currents. When this occurs, the nervous symptoms of weakness, depression, rigors, and contraction of some parts and vessels, speedily followed by relaxation, mark the stage of invasion.

"Thirdly, and simultaneously. Various parts, especially the muscles, and probably some of the organs, deprived in greater or less degree of nervous influence, begin rapidly to disintegrate, and by their disintegration produce supernatural heat.

"Fourthly. This metamorphosis is aided in most cases by the condition of the vagus and vasimotor nerves, which cause increased action of the heart and dilatation of the vessels.

"Fifthly. The contamination of the blood, already produced by the morbid agent, is increased by the check which the normal extravascular currents experience by the pouring into the blood of the rapidly disintegrating tissues, and by the continued action of the morbid agent, which in almost all cases appears to act more rapidly and more powerfully in blood rendered impure in any way, either —as shown by Carpenter—by retention of excretions, absorption of septic substances, or, as in fever, by the too rapid metamorphosis of tissue.

"Sixthly. The various organs suffer (apart altogether from specific changes), and must, one would think, produce increased deterioration of the blood. Thus, the lungs are congested in so many cases, that we can scarcely suppose proper aeration to go on; the liver would seem, from Frerichs' observations, to be, in some cases at any rate, in a most abnormal condition, and to produce compounds, such as leucin, unknown in health; and the spleen in many fevers, if not in all, enlarges (in persons of a certain age), and is congested, possibly even to extravasation.

"But to these complex conditions another must yet be added; food is almost entirely withdrawn, and the various alkaline and

neutral salts, unless supplied in the form of medicines, no longer pass into the system. And as in the excretions these salts are continually passing out and are not restored, there must at last, in fevers, be a most unusual disproportion between the organic and the inorganic constituents of the frame. The blood will show this the latest, for it seems to maintain its composition, as far as the salts are concerned, with great tenacity; and it probably borrows from the organs the ingredients it loses by the excretions. The exact influence of this loss of salts is not certain. The blood seems certainly to become less alkaline, and it is by no means improbable that this may render oxidation less complete than it should be, and thus cause some of those instances of retention of effete materials to which I (Dr. Parkes) have formerly referred.

"Thus, the blood is contaminated by primary action of the agent, the products of metamorphosis of tissue, the loss of the salts, and by the altered action of organs; the nervous system is, therefore, day by day, constantly more affected, and reacts still more on metamorphosis, the heat increases, the heart's action still quickens, and the fever reaches its acme."

Subversive of this theory, and that of Virchow more especially, is the fact that an increased destructive metamorphosis of tissue is *not* the primary and essential pathological change in the production of Fever. Undoubtedly, any waste tissue entering the circulation in greater quantity than can be duly eliminated by the excreting organs, will poison the blood, and possibly induce reaction—*i.e.*, acceleration of the circulation, &c., or Fever. But abnormally rapid destruction of tissue *presupposes* its more rapid oxygenation, and therefore a more rapid circulation; in other words, presupposes that very condition of the circulation which waste tissue, being absorbed, is supposed to induce. The result is assumed to be the cause of more rapid tissue-change.

Increased destruction of tissue may, indeed, become a *secondary* cause of reactionary excitement of the circulation, and thus a secondary cause of Fever. So, likewise, the retention of excrementitious matter—through failure of the function of secretion, and

the privation of salts—through failure of the appetite for food—are both secondary to some morbid condition of the blood, and secondary, therefore, as causes of Fever.

Increased tissue-change, suppressed excretion, and privation of food, are probably concerned in *maintaining* Fever. A morbid blood-condition is generally the primary and essential cause in operation. This is plainly the origin of Fever by infection and by malaria. The whole tribe of infectious and malarious fevers originate by the inhalation of poisonous matters, and thence the production of blood-disease. The same mode of origin is equally obvious in the production of Fever by contagion—*e.g.*, puerperal fever. Fever of the inflammatory type is perhaps the *only* exception to this law; and here the acceleration of the circulation originates, apparently by irritation from the part inflamed, transmitted through the nervous system to the heart. In all other forms of Fever, acceleration of the circulation, and the other phenomena of fever, originate from some morbid condition of the *blood,* as the *primary* cause in operation.

The Prevention of Fever has been sufficiently considered in connexion with the etiology of the various species of this compound constitutional disorder (p. 308 *et seq.*). It only remains for me to notice those preventive Principles, and measures, which preclude 'malarious fever.'

The circumstances under which malarious effluvium is engendered are, a concurrence of heat and moisture impregnated with decomposing vegetable matter. Marshes, jungles, and similar localities, are hotbeds for the production of malarious poison.

In proportion as the temperature increases, so is this poison generated. But a certain amount of moisture is also necessary. If the heat be sufficient to dry up and parch the surface of the soil, such soil ceases to yield the poisonous miasm. If, again, the moisture amount to a swamp, it is proportionately less productive. Consequently, a very dry season, or a very wet season, is equally free of malarious fever. A porous, permeable, and constantly damp soil, exposed to solar heat, is most prolific, and especially if the heat continues to brood over it. Thus, malarious fever is most

rife in the Maremme of Italy, in parts of North America, the south of Spain, and in the lake districts near Varna, in Bulgaria.

Yellow fever—a variety of paludal fever—is in some respects peculiar, with regard to the geographical and meteorological circumstances under which it arises. "Topographically it is a disease not proved as yet to be one *sui generis*, endemic only in low districts on the sea-coast, but under certain circumstances sporadic in other places; never appearing beyond 48 degrees of north latitude, nor without a temperature of 72 degrees Fah. at least, nor above the elevation of 2500 feet above the level of the sea."*

The 'infecting distance' of the paludal poison, no less than its source, is a question pertaining to the prevention of its effect—malarious fever.

The *altitudinal* range of this poison has not yet been determined. An elevation of 2000 to 2500 feet affords either total exemption from paludal disease, or else so modifies it, that the mortality from all causes will not, on an average of a series of years, materially exceed that of an equal number of British troops, for example, when on duty in the capital of their native country. In towns partially freed from marsh miasmata by extensive drainage, a few feet only perpendicular height makes an almost inconceivable difference in the liability to paludal disease. One preventive measure is suggested by this fact, and extensively employed in India. The houses are built upon stakes, and thus raised a few feet above the ground.

The *lateral* spread of marsh miasmata is even a more difficult question than that of the altitudinal range.

If water intervenes—then, in Europe, the horizontal range is less than 3000 feet over *fresh* water; and in tropical climates the range over *salt* water is greater.

If land intervenes, the question of lateral range is still more complicated by the different degrees of affinity which either the poison, or the vapour holding it in solution, has for the many substances over which it is diffused.

* Statistical Report on Sickness of Troops in W. Indies. Humboldt, p. 103.

Trees remarkably intercept the transit of the paludal poison. Different soils also attract or repel, and affect its transmission. Buildings interrupt its passage, according to the kind of materials of which they are constructed.

Preventive measures will consist in avoiding those localities which engender malaria. The more wealthy inhabitants of Rome forsake the city during the summer months, when the Pontine marshes are exhaling their poisonous effluvium. Change of residence may, however, be impracticable. Then, the *night* air should be avoided; for, impregnated as it is with the effluvium exhaled during the day, and condensed by a colder temperature, the pestiferous evening dews are falling. This precaution is especially incumbent on those who, having had fever, are liable to relapse. Debility predisposes to malarious fever, and even the strong should avoid any occasion of fatigue to mind or body during the prevalence of paludal disease.

Acclimatization is preventive. Therefore a new comer to a fever locality should, if possible, arrive in the interval of the fever seasons. In Jamaica and other West India Islands, January is the month most free, and the right time for becoming prepared to undergo the subsequent trial of the malarious season in autumn.

The *avoidance* of malaria is surpassed and superseded by preventing its *production*. Drainage, or retaining the waters of a fever district up to a certain mark, are the only radical principles of prevention; but the contrivances accordingly, by drains, floodgates, &c., belong to the province of the civil engineer.

SUMMARY OF ETIOLOGY, AS GUIDED BY PATHOLOGY.

The association of 'internal causes'—their various co-operations and orders of succession in the animal economy.

The Preventive view of such causes is consummated by the earliest and most exact detection of their *manifold* relations.

In concluding our analysis of 'internal' causes, it is highly important to view them *synthetically*, *i.e.*, in respect of the various

combinations and orders of succession in which they may possibly occur. To fully realize this synthetic view of internal causes would necessitate the investigation of *all* their relations to each other—an inquiry no less extensive than the whole of Pathology, and far beyond the range of our present knowledge. Nevertheless, I may enumerate and illustrate those general combinations and orders of succession which are of *most frequent* occurrence in the practice of Medicine and Surgery.

They are six in number, and as follows:—

Firstly. Constitutional morbid conditions frequently coexist.

Secondly. A constitutional morbid condition having caused some form of local disease, such local condition reacts in many cases, and induces a constitutional morbid condition of different character to the original.

Thirdly. Local morbid conditions frequently coexist.

Fourthly. A local disease, or an injury, having caused a constitutional morbid condition, such condition is reflected in many cases on the original disease or injury.

Fifthly. The local origin of a constitutional morbid condition, and thence of another local disease of different character to the former, is an order of succession also of frequent occurrence.

Sixthly. The local origin of local disease, with, or without, a constitutional morbid condition supervening, is another order of succession of frequent occurrence.

These abstract propositions admit of many familiar illustrations.

Of 'constitutional morbid conditions coexisting,' the combination of morbid conditions of the blood and nervous system is daily exhibited by Fever, every variety of which is referrible to this head.

Fevers themselves occasionally coexist, and also with blood-diseases not specially febrile.

Thus, typhus and typhoid fever may each coexist with erysipelas; but the probability of their concurrence is very unequal. In Jenner's series of cases, erysipelas occurred in 7 of the 23, or

nearly $\frac{1}{3}$ of the cases of typhoid fever; and in 2 only of the 43 cases of typhus fever, or in less than $\frac{1}{20}$ of them.

Measles may coexist with small-pox—the latter supervening (Bateman. Willan*). This combination generally proves fatal (Macbride). Small-pox may precede measles, and coexist; of which concurrence, cases are recorded by De Haen, Vogel, and Horne. The coexistence of cow-pox and of hooping-cough with measles is not unfrequent.

Scarlatina may coexist with vaccinia, with erysipelas, and probably with most other poisons.

Small-pox may coexist with scarlatina, or with hooping-cough (Dessessarz); with measles (Cruikshank); with syphilis (Dimsdale); and with intermittent fever (Heberden). In one case small-pox, measles, and hooping-cough coexisted; and all ran their course together (Ring). Small-pox and vaccinia being introduced into the system together, the one may precede the development of the other, or both coexist. But either disease having run its course, precludes the other with few exceptions.

Varicella may coexist with cow-pox, small-pox, and perhaps with many other morbid poisons.

Erysipelas may coexist with typhus, with typhoid fever, with small-pox, and with syphilis—primary or secondary.

Plague, probably, may coexist with any kind of blood-poison, for no known one precludes this disease.

Hooping-cough may coexist with small-pox (Willan); with measles (Bateman. R. Williams); and with intermittent fever of every type (Desruelles).

Influenza may coexist with measles, with scarlatina, with typhus, with syphilis, and probably with any other blood-poison. Paludal fever—intermittent and remittent—probably may coexist with many poisons. Intermittent fever with small-pox was noticed by Heberden.

The combination of blood-diseases, not specially febrile, is not unfrequent.

---

* See Edin. Med. and Surg. Journ., vol. xv., p. 314.

Thus, cholera may coexist with syphilis, besides coexisting with febrile diseases—with typhus, with small-pox, or with intermittent fever.

The coexistence of non-febrile blood-diseases is frequently illustrated by scrofula with syphilis. So prevalent indeed is this relationship as to suggest the possibility or even the probability of scrofula being the offspring of syphilis. Such at least is the result of Erasmus Wilson's large experience,* respecting the origin of scrofulous *skin* diseases.

My second general proposition expresses an 'order of succession,' and one very commonly observed in the operation of internal causes. It is this:—'A constitutional morbid condition causes some form of local disease, and this reacts so as to induce another constitutional morbid condition of different character to the original.'

Thus, inflammation arising from a blood-poison reacts and induces inflammatory fever. The secondary fever that occurs after the eruptions of exanthemata is an instance of this kind.

Secondary fever is most marked in small-pox. The 'specific fever' terminates as the eruption appears; but when it becomes mature, inflammatory fever supervenes about the eighth day. A similar reactionary fever ensues in the course of other exanthems.† So also scrofulous and syphilitic eruptions, the fruit of 'specific' blood-disease, are usually chronic and persistent; but if beset with inflammation, they induce the constitutional disturbance of inflammatory fever. Scurvy is a blood-disease, attended with local extravasations of blood, during which the pulse is generally slow and feeble; whereas, in the advanced stage of this disease, the pulse rises to 120 or so per minute, a hot skin, &c., supervenes—in fact, inflammatory fever. *Hot* scurvy was the name formerly given to this condition by way of distinguishing it from the former, or cold scurvy; and pending the transition from cold to hot, the

---

* Syphilis, Constitutional and Hereditary, &c., 1852.

† See Eruptive Fevers, Gregory.

effusions of blood and fibrin, forming hard tumours, become very painful and hot, coincident with which inflammation, its symptomatic fever arises.* Malignant disease, in the course of its development, undergoes changes by disintegration and softening, possibly without concomitant inflammation. In many cases, however, inflammation of neighbouring textures and parts is superadded, being apparently excited by the presence of cancer. Thus, vaginal discharge may accompany a non-ulcerated scirrhus of the uterus; pleurisy may be associated with mammary cancer, inflammation of the peritoneum with ovarian cancer. Febrile action of low type exists—observes Walshe†—in the majority of cases, probably in about three-fourths of the total number. Fits of shivering, followed by heat and perspiration, occur in some instances; they rarely constitute a well-defined paroxysm, and do not recur at fixed intervals. Febrile action is more common and severe in cases which run their course rapidly than in those of tedious progress.

'Local morbid conditions frequently coexist.' This proposition is constantly illustrated by the association of inflammation with every kind of injury, with wounds, burns, fractures, dislocations, &c.

'A local disease or an injury having caused a constitutional morbid condition, such condition is reflected, in many cases, on the original disease or injury.'

Morbid conditions of the nervous system thus induced are reflected on the part affected. For example, tetanus arising from an injury of any part of the body, may react on that part, causing it to assume an unhealthy condition. After a strangulated femoral hernia, for which I operated, no bad symptom ensued for a week; then, tetanus supervening, the wound immediately opened and became distinctly gangrenous.

Morbid conditions of the blood, arising from local disease, are reflected on the part affected. Thus, an inflamed ulcer having induced inflammatory fever, this reacts on the ulcer, aggravating

---

* Lind, ed. 2, p. 390.

† Nature and Treatment of Cancer, 1846.

the local inflammation even to suppuration and sloughing. So also a suppurating wound having caused pyæmia, this morbid blood condition is reflected on the part affected, as well as causing secondary abscesses in other parts of the body.

'The local origin of a constitutional morbid condition, and thence of another local disease of different character to the former, is an order of succession also of frequent occurrence.'

This is a variation of the order of succession expressed by the previous proposition. Illustrations abound in the shape of diseases affecting any organ concerned in maintaining a right state of the blood. Diseases therefore of the organs of digestion and excretion, including those of respiration, induce morbid blood conditions, and these are reflected on all parts of the body, any portion of which is liable to become the seat of some disease of nutrition. Diseases of the 'digestive organs' more particularly—or at least some functional derangement of these organs—in their relation to 'primary assimilation,' and thence through the blood to distant parts, assume a pathological importance of daily interest to the practitioner, but which was not appreciated prior to its elucidation by Abernethy's observations.

Primary syphilis in its relation to the blood, and thence to secondary syphilitic affections, is another familiar example of the same order of succession.

'The local origin of constitutional disease,' alone, or reflected, represents the Etiological Principle of the two previous propositions, and I reiterate this principle, because it is one of very frequent occurrence, especially in surgical practice, but apt to be overlooked in the more prolific, if not more frequent, 'constitutional origin of local disease.'

'The local origin of local disease, with, or without, a constitutional morbid condition supervening, is an order of succession which also frequently occurs.' It is well illustrated by inflammation and its consequences—local and constitutional. Inflammation affecting any one texture, travels to adjoining textures,—as from the articular heads of bone to the cartilages adjoining, or from the

synovial membrane to these cartilages: cotemporaneously (with this local production in one texture of (local) disease in another) hectic fever supervenes as the constitutional disorder induced.

The local origin of a local affection, *without* any constitutional disorder supervening, is illustrated by the origin of many nervous and muscular affections.

These are, in my judgment, the most frequent combinations of 'internal causes,' and the orders of succession that most frequently occur; of which, however, more abundant illustrations might be adduced.

The Preventive aspect of such causes will be consummated by the earliest and most exact detection of their *manifold relations*, which implies an adequate knowledge of Pathology—the functional consideration of diseases and injuries; while the earliest and most exact detection and discrimination or corresponding diagnosis of diseases and injuries—the *causes* themselves, in operation, implies that application of Morbid Anatomy, during life, which I have named Clinical Pathological Anatomy.

THE PRINCIPLES OF PROGNOSIS,

OR

OF THE EARLIEST AND MOST EXACT FOREKNOWLEDGE OF THE COURSE AND TENDENCY OF DISEASES AND INJURIES, INDIVIDUALLY, TO OR TOWARDS A FAVOURABLE OR AN UNFAVOURABLE ISSUE.

"Nescire quid antea quam natus sit,
Acciderit, id est semper esse puerum."—CICERO.

PRELUDE.

The earliest and most exact foreknowledge respecting the course, tendency, and issue of injuries and diseases, can be *concluded* only by the experience derived from special 'clinical' observation of their continued functional manifestations; but such Prognosis implies a previous Etiology, in respect of internal causes, and a prior Diagnosis, therefore, each of similar quality. Hence the guidance of Clinical Pathological Anatomy indirectly to Prognosis.

I NOW approach the most important yet most difficult preliminary 'Department' of Surgery. Most important, because a sufficiently early and exact foreknowledge of the course and tendency of morbid conditions, individually—*i.e.*, before 'complications' ensue—can alone determine the *earliest* period when therapeutic assistance is solicited, and the *least* amount therefore, no less than the *kind* required; but most difficult is *such* foreknowledge, because obscured by certain circumstances which influence the probable course and tendency of all the various morbid conditions to which the body is liable. How often will it happen that when a sufficiently early and exact diagnosis and knowledge of the origin of a disease, as being local or constitutional, its dependence or not on any 'internal cause,' and its own operation as an internal cause of other morbid conditions; when I say all this preliminary knowledge has cleared the way for a corresponding anticipation of the

future course and tendency of such disease from that date, our anticipations are *not* realized—being favourably reversed, when prognosis had been unfavourable, and disappointed, when a happy issue was most expected.

No sure foundation therefore for (rational) Therapeutic Treatment is thus provided.

The truth is, that although Pathological Anatomy supplies the *basis* of the earliest and most exact prognosis (through a similar diagnosis, and identification of internal causes; both of which departments of Surgery are built, as we have seen, on Pathological Anatomy *applied* during life), yet such prognosis, or foreknowledge of the course and tendency manifested by morbid conditions of structure, can alone be *concluded* by long experience, through 'clinical' observation of the diseased *living* body, in all its moods and declensions. Prognosis, no less than Etiology, in respect of the *operation* of internal causes, is an application of pure Pathology; and depending immediately on a right interpretation of *functional* disturbance, is necessarily obscured by its *indefinite* character. Adequate prognosis thus becomes the most difficult department of Surgery, or at least as much so as etiology, respecting the *operation* of internal causes.

While, however, the source of difficulty in either case—the variability of functional indications—being inherent, cannot be altogether avoided; yet this difficulty may be reduced by establishing *these* departments of Surgery, no less than Diagnosis, *as much as possible* on a pathologico-anatomical foundation. By constantly referring functional disturbance to alterations of structure—effects to their causes—Prognosis will acquire a *rational* character, instead of being merely an empirical estimation of symptoms, as 'good' or 'bad,' according to *experience*.

The nature, situation, and extent of a morbid condition of structure having been ascertained by diagnosis, the dependence or not of such condition on an internal cause or more—operating through functional ties—being also known; and its own operation as the immediate cause of the symptomatic *functional* disturbance,

being determined, (generally, *not* determinable—Chapter II.); Prognosis, thence derived from Pathological Anatomy, will no longer be the expression of an empirical estimation of symptoms as determined by experience alone, but a *rational* and intelligible foreknowledge, of the earliest and most exact character also, by virtue of this its ultimate source.

This consummation would be the very antithesis of that *symptomatic* Prognosis which Hippocrates introduced, and the "Prognostics" of the Empirical or Symptomatic School contrast unfavourably with those drawn from Pathological Anatomy.

But, "Systems of Medicine and Surgery" which in any way acknowledge the therapeutic guidance of Prognosis, are modelled after the "Prognostics" of Hippocrates, who, says his learned commentator,* had for his object "such a general description of the phenomena of disease, as would apply to all the disorders of the animal frame." In fact, *general* Prognostics, rather than 'Principles' of Prognosis, were sought to be established. "With this intention, he brings into review the state of the countenance, the position of the patient in bed, the movements of the hands, the respiration, the sweats, the state of the hypochondria, dropsies which are the consequences of acute diseases, the sleep, the urine, the alvine dejections, the vomitings, the sputa." These and such like prognostics do no more for Prognosis than as symptoms of *general* significance, respecting the probable course and tendency of most diseases, and 'Systematic Works' have *hitherto* gone no further. I shall here endeavour (in this as in the other 'Departments' of Surgery) to establish certain fundamental Principles guiding *all* Prognosis. My object will be by an original analysis, systematically conducted, to evolve the *essential* conditions which represent the course and tendency of *all* injuries and diseases. These essential conditions will be found to signify Pathological Anatomy as regulating, but pure Pathology—functional disturbances—as determining, Prognosis.

* The Genuine Works of Hippocrates, translated for Syd. Soc., by Francis Adams, LL.D., 1849, vol. i., p. 231.

A critical exposition of the comparative guidance of structural and functional conditions, in the earliest and most exact Prognosis, is expressed by the terms of the following propositions.

Firstly. Persistence, or not, of the immediate cause or causes, and therefore, the question of an 'internal' cause, or more, being in operation, is the *immediate* foundation of the earliest and most exact Prognosis—unfavourable, or favourable, respectively.

Secondly. The earliest and most exact Prognosis is *regulated* by the kind and extent of 'structural' alteration that the organ or texture has undergone, and by the period also during which such alteration of structure has been in operation, as an 'internal' cause of functional disturbance. Nevertheless an acute disease or sudden injury is, *cæteris paribus*, more unfavourable than a chronic lesion, to which the organism has become habituated.

Thirdly. The comparative 'functional' importance and influence of the organ or texture affected *determines* our Prognosis respecting the course and tendency of its morbid conditions.

Sub-Principles :—

(*a.*) Organs and textures which fulfil functions by virtue of their 'vital' properties (and 'chemical composition') suggest an unfavourable Prognosis.

(*b.*) Organs and textures which fulfil 'mechanical' functions suggest a more favourable Prognosis.

Local disease or injury, *per se*, suggests a favourable Prognosis.

Local disease or injury, *sustaining* or *sustained* by any 'constitutional disorder,' suggests an unfavourable Prognosis.

'Constitutional diseases' implying each some morbid condition of a 'texture' of *general distribution*, as well as of *predominant functional* influence throughout the body, suggest an unfavourable Prognosis.

These *general* Principles of Prognosis commend themselves to our acceptance, by virtue of their accordance with the suggestions of unanalysed clinical experience; but the guidance of each one is, I conceive, so valuable, and the ramifications of each one are so numerous, that to establish and amplify their guiding

power individually, comparatively, and collectively, as the source of clinical foreknowledge, at once the earliest and most exact, respecting the course, tendency, and issue of diseases and injuries, will be the purpose of the two following chapters.

CHAPTER XII.

THE PROGNOSTIC GUIDANCE OF CLINICAL PATHOLOGICAL ANATOMY.

Persistence, or not, of the immediate cause or causes, and therefore the question of an 'internal' cause, or more, being in operation, is the immediate foundation of the earliest and most exact Prognosis, unfavourable or favourable, respectively.

The Prognostic value of this fundamental Principle, illustrated by the career of injuries and diseases, from the whole range of General Surgical Pathology.

No Principle of Prognosis commands our assent more readily and implicitly than the above; and yet, practically speaking, it is liable to be overlooked. The value of this Principle can only be adequately estimated under circumstances calculated to exhibit its essential significance.

Persistence of the immediate cause, or causes, in operation—all other circumstances being favourable to recovery—is the surest omen of an unfavourable result; while such cause, or causes, being removed—although all other circumstances be unpropitious—is yet the surest guarantee of a happy issue.

The causes alluded to are of very many kinds, whereby the Principle itself assumes as many different aspects; in order, therefore, to a comprehensive appreciation of this Principle in all its various bearings, I shall draw my illustrations from the whole range of *general* (surgical) Pathology.

The causes in question are not 'external,' they being usually of momentary application only; whereas 'internal' causes have a

persistent character. The persistence or not of *these* causes, therefore, lies at the root of a 'rational' prognosis—earliest and most exact—because determined by reference to *primary* and *essential* conditions; as well as being—unlike functional disturbance—*definite*. Pathological Anatomy supplies such *data*.

Firstly. Take injuries of mechanism. *Wounds* are occasioned by the almost momentary application of mechanical violence. So far our prognosis of these lesions will be determined by the kind of injury to the soft parts. Adhering and sloughing wounds will be the chief ground of distinction, and to this ground of prognosis I shall recur; but the breach of continuity may be maintained—the cause may be said to continue in operation, by various circumstances which *prevent coaptation* of the soft parts that have been severed.

Foreign bodies—*e.g.*, dirt, portions of clothing, &c.—may have been thrust into the wounded textures, and intervene so as to prevent an even coaptation of the cut surfaces, and moreover excite suppurative inflammation. A man, in the act of cleaning a window, has thrust his hand through a pane of glass. The wound or wounds are perhaps extensive, yet simply incised, rather than lacerated or contused, and the man is in excellent health. All circumstances, therefore, local and constitutional, are favourable to a speedy reparation through union by primary adhesion; but if pieces of glass have remained in the wound, this condition is sure to induce suppuration. Our prognosis turns on the persistence of this cause in operation.

The *tents, syndons,* &c., formerly used in dressing wounds, were no less foreign bodies than pieces of glass or other substances accidentally introduced; and cases, taken almost at random from the earlier records of surgical practice, exhibit the persistent operation of such causes in preventing primary union and their relation to prognosis accordingly.

Among surgeons who felt misgivings as to the propriety of introducing tents into wounds, few dared to shake off the trammels of authority, and enter a protest against the prevailing barbarous

practice. "They never trepanned the skull," observes J. Bell, "where there was inward suppuration without thrusting a tent into the substance of the brain itself, though sometimes they were contented with laying in a syndon or slip of linen." "Often, I am sure," writes La Motte, "I have seen those who would have been very willing to have taken the help of a mallet to drive in their tents into the thorax. From shame only they refrained." Belloste was the first reformer, and the histories of his cases show the Prognostic bearing of foreign bodies on the course and tendency of wounds.

Thus, a soldier was shot in the belly, the ball entering near the navel, and passing out through the kidney; the entrance wound had healed in spite of all the surgeon's diligence, but the posterior orifice, where lay the injured kidney, the surgeon had contrived to keep open with a very long and hard tent, which he had thrust even into the substance of the kidney, occasioning a perpetual flow of urine from the part. Belloste closed this fistula, by withdrawing the tent and approximating the surfaces of the channel, aided by stimulating applications. The prognosis of wounded joints also, from the same point of view, is well illustrated by the course of a case, before and after the removal of the tents which were thrust into it. A wounded knee-joint had progressed favourably, when the treatment changed hands, and on Belloste seeing the case, he found in the knee six fistulous holes, each hole spiked up with a hard tent, and each tent long enough to reach to the bottom of the joint. The leg and foot were exceedingly swollen, and the body proportionably emaciated with hectic. The whole joint became surrounded with matter, the gutter leading backwards from each fistulous sore communicated with some adjoining hole, and the dressers had every morning stopped up the rest of the openings with their fingers, while they injected through some one hole a spirituous lotion, which distended the whole knee with intolerable agony. Yet these foreign bodies being removed, and the wounds dressed easily, gave immediate relief, so that the patient enjoyed sound and refreshing sleep on the night of that day.

The *great extent* of a wound is a condition which, not intercepting union by primary adhesion, is no ground, *per se*, for an unfavourable prognosis, provided only any foreign bodies are at once removed, and the wounded textures evenly adjusted in contact.

In the work last referred to, by J. Bell,* an illustrative case is quoted. A *drunk* countryman, aged sixty-six years, riding furiously along the street, pitched with such force against the sharp edge of a door-post, that about the *length and breadth of a hand* was stripped off the right side of his head, and laid down on the cheek. Some people, in the dark, took his wig out of the kennel, and, not knowing what had happened to his head, put it on full of mud, and squeezed his hat over it. He did not complain of his head, but greatly of his neck and shoulders. The dirt being thoroughly ingrained into the cellular tissue of the scalp, could not all be removed. But the flap was washed and readjusted, at the same time stuffed with soft liniments, and secured by four loose stitches. Partial union took place, and in five days all dangerous symptoms were over. The man enjoyed good health above twenty years.

There cannot be a reasonable doubt that the 'stuffing,' in this case, acted as a foreign body, and prevented complete primary union; so that this is an instructive instance, alike showing the prognostic significance of persistent foreign bodies in wounds, and the *efficacy* of at once removing this cause of interception, even although the wound be extensive, and the health disordered. In short, as with the presence of foreign bodies, Prognosis is unfavourable under the most favourable circumstances in *all* other respects, so with the removal of this impediment to the healing process, our anticipation is hopeful, even when other circumstances are unpropitious.

In like manner, extravasated blood operating as a foreign body, suggests a similar prognosis according to its persistence or removal. In loose pendulous parts the evil action of extravasated blood on the healing of wounds is most conspicuous. Shortly

---

* Principles of Surgery, ed. C. Bell, 1826, vol. ii., p. 463.

after amputation of the breast, for example, the blood being apt to ooze and collect in the basin-like cavity left by the absent *mamma*, the breast looks fuller than before its removal. The lips of the wound may have united, but adhesion gives way, the sutures threaten to burst, and the black congealed blood pouts out between them. Our prognosis of primary union is balked by this intervening foreign body coming into operation, although the character of the wound and the general health may alike be favourable. In like manner our anticipation of union is frustrated, when secondary hemorrhage occurs in the course of other wounds. Blood escapes into the scrotum after the operation for scrotal hernia, possibly giving rise to suspicion that the hernia has again descended; the soft jelly-like consistence of the swelling, albeit the scrotum is considerably distended, softer and less nodulated than the most lax omental hernia, determines the diagnosis, and warrants the immediate destruction of any partial adhesions that may have formed,—the withdrawal of the sutures and evulsion of the clot. Severe if not fatal constitutional disturbance from liquefaction and absorption of the congealed blood is thus averted, and a more favourable prognosis restored; although the wound may not again heal by adhesion.

Primary union is promoted, and a hopeful prognosis at once suggested by removing all blood from a wound, *before* adhesion is allowed to commence. This was the principle of the practice formerly in vogue, that of *sucking* wounds, and thence the assurance of an uninterrupted process of adhesion. The most formidable wounds were thus reduced to a more simple and satisfactory condition. La Motte tells us of a soldier who had been run through the breast with a fair lunge, in at the pap and out at the shoulder. Having examined the wound and noticed the length of his antagonist's sword, being well satisfied that the weapon had pierced the lungs and transfixed the breast,—La Motte states, that the drummer of the regiment, who acted as sucker on this occasion, first sucked one wound, then turning his patient over he sucked the opposite wound; he then applied a piece of chewed paper upon

each, and next day the soldier was seen walking in the streets!

Loose and pendulous parts tend also, by their *weight* and *mobility*, to disunite in the process of healing, or not to unite at all. This course is exemplified by wounds of the breast, and by deep and extensive flesh-wounds in other parts. The prognosis in respect of primary union will, however, practically depend on the question whether this condition, as the persistent cause of displacement, be, or can be, overcome by adjustment of the parts, and their steady retention in even contact, by sutures and bandages suitably applied.

Most parts of the body, when severed, are liable to displacement, not only by their weight and looseness of connexion, but owing also to *muscular* action. Thus, the process of primary adhesion is hindered in scalp-wounds by the action of the *occipito-frontalis* muscle, in wounds of the face by that of the facial muscles, and in wounds of the abdomen by that of the abdominal muscles. Flesh-wounds of the extremities are obviously affected in a similar manner. But here again, whatever part be subject to muscular action, the prognosis, respecting primary union in relation thereto, practically turns on the question of controlling this occasion of mobility and displacement. Suitable retentive appliances must be employed at an early period, otherwise the muscles will have retracted and become fixed by inflammatory adhesion; and moreover, thus leaving a gap, which, on filling up with cicatrix-tissue, entails a permanently weakened state of the muscles.

One other occasion of uneven coaptation, and prognosis accordingly, has yet to be noticed. A *lacerated* wound does not allow of even adjustment, although no other circumstance may prohibit perfect coaptation of the torn textures. Such wounds, therefore, cannot heal by adhesion throughout their extent; and our prognosis being guided accordingly, saves much useless endeavour to solicit adhesion by dragging the ragged textures together. Sloughing is inevitable. The bite of a horse is a wound in this predicament; so also wounds inflicted by various

kinds of machinery, as when a person's hand is caught up and lacerated between cog-wheels.

These are the chief circumstances which, intercepting an even coaptation of wounded parts, may be said to prevent union by maintaining their division. The original cause of severation is virtually persistent in its operation.

Burns exhibit a favourable or unfavourable tendency, and suggest a corresponding prognosis, partly according to their exclusion or not from the action of the air. Exposure is a persistent cause of irritation, and perpetuates the burn. "Hence," Dr. Thomson observes, "great care should be taken, in every instance, to preserve the raised portion of cuticle as entire as possible; the vesications should be relieved by pricking, thus allowing the serum to drain off slowly, without at the same time the entrance of any air between the cuticle and true skin." When, however, the surface is denuded of cuticle, the dressings should be removed, as Dupuytren suggested, not so as to uncover the whole surface at once, but portion after portion, as each is dressed; and this should be done as quickly as possible, and not more frequently than absolutely necessary for cleanliness. All these precautions prevent exposure; and, on the same principle, the method of dressing burns with carded cotton, with flour, or with the Carron-oil liniment, is efficacious.

Fractures are disposed to unite by a process analogous to the primary adhesion of soft textures, or to remain ununited; and our prognosis is guided no less by consideration of the causes of fracture, and of displacement, than by the kind of fracture,—whether simple, compound, or complicated by association with other injuries.

Among the causes *predisposing* to fracture, some operate by rendering the bones more brittle or softer than in a healthy condition, whereby they break when slight force is applied, either through direct violence or the ordinary action of the muscles. But such causes intercept, or at least retard, the process of union, and thus perpetuate fracture. Considered, therefore, in relation to

prognosis, they further illustrate the Prognostic Principle laid down in this chapter.

Age is a telling circumstance. The bones become more brittle as life advances, owing to the proportion of phosphate of lime in their composition increasing, while that of the animal matter diminishes, and this reduced ratio of the organized constituent retards or prevents the formation of callus. The osseous texture loses its reparative power; for, to use the expression of Boyer respecting this degeneration of bone, "its vitality is nearly annihilated under the mass of phosphate of lime which accumulates." "Intra-capsular fracture of the neck of the thigh-bone, in old people, is rarely if ever repaired, otherwise than by ligamentous union."

Certain blood-diseases seem to impair the reparative power, to perpetuate fracture, and suggest an unfavourable prognosis accordingly.

Syphilis, mercurialism, or both combined, are thus unfavourable. Scurvy, more especially, retards, or altogether prevents, the formation of callus, and even disposes to absorption of an old callus, and the spontaneous recurrence of fracture. Many cases occurred in Lord Anson's voyage. Cancer, affecting the osseous texture, certainly predisposes to fracture, but it is a disputed point whether or not this condition retards the process of union. An old woman, dying of cancer, broke her thigh-bone while turning in bed; union ensued as well as under ordinary circumstances. A lady, with cancer of the breast, had also a scirrhous affection of the collar-bone; and one day, in moving her arm, the bone broke, yet it readily united.* Other cases lead to an opposite conclusion. Desault mentions the case of a nun in the Salpétrière, whose arm broke as she was being handed out of a carriage. No union took place; and some time after, while changing her posture in bed, her thigh-bone broke. It was then ascertained that she had a cancer in her right breast. A woman

* London Med. Gaz., 1833-34, p. 56.

aged eighty-two, long afflicted with a cancerous ulceration of the mamma, felt her right thigh suddenly break as she stood. After death, the bone was found to be so flexible that no union could have ensued. Another female, aged fifty-six, had for several years a scirrhus of the left breast. It was removed, and the wound healed, but afterwards broke out in the form of cancerous ulceration. In this stage, as the patient was being placed in a cart, the right thigh-bone snapped about three inches below the trochanter. Death ensued in three months; and on dissecting the limb, the bone was found to be remarkably soft throughout its whole length; the knife could be passed through it at any part. The bone, however, was bent, rather than fractured.*

Softening of the bones may arise from other diseases, and they also predispose to fracture; but it is doubtful whether these conditions indispose fractures to unite, and perpetuate the injury, thus warranting an unfavourable prognosis.

Mollities ossium is a condition not necessarily adverse to union. An old lady broke both her thigh-bones by merely kneeling down, and on being lifted up, the humerus snapped. Hardly any constitutional disturbance followed, and in a few weeks the bones united.† *Rickets* does not apparently interfere with the formation of callus. Gibson mentions the case‡ of a young man in whom the bones of the arm, fore-arm, thigh, and leg, were all broken repeatedly, even from the slight accident of catching his foot in a fold of carpet whilst walking across the room. The clavicles suffered more than any other bone, they having been fractured eight times. Yet this youth enjoyed excellent health, and union invariably ensued without delay or much deformity. In a child five years of age, the right humerus was fractured three times, in different parts, within the short period of eighteen months; and B. Bell, who saw this case, further states that several similar cases

---

* Surg. Dict., S. Cooper, 1838, p. 599.

† Study of Medicine, Mason Good, M.D., ed. iii., vol. v., p. 332.

‡ Institutes of Surgery, vol. i., p. 370.

came under his notice; in all of them the patients seemed to enjoy robust health, were apparently untainted by scrofula, and their fragile bones united in a shorter space of time than he generally observed to be the lot of those whose bones were tougher.*

Scrofulous softening of bone certainly predisposes to fracture, and perhaps also perpetuates the injury, by retarding, if not preventing, union. Prognosis is guided accordingly when this condition is present.

Febrile disturbance unquestionably delays the formation of callus. *Erysipelas* is particularly unpropitious in this respect. Pregnancy may, or may not, retard the process of union, although in some instances it is entirely postponed until after parturition. In proof of pregnancy having a retarding influence, two cases are adduced by Fabricius Hildanus;† a third case by Alanson;‡ and a fourth by Werner.§ Sometimes, however, union is completed in the average period. A woman fractured both bones of her leg at the fifth or sixth month of pregnancy. Union took place, and in about the usual time.‖ Such also was the experience of Léveillé,¶ and of Boyer;** but in many instances fractures show no disposition to unite until after delivery.††

Besides the foregoing occasions of apprehension as to the probability of fractures uniting slowly and imperfectly, or remaining ununited—most of which, referring to the want of reparative power, are likewise predisposing causes of fracture; the various causes of *recurring* displacement after fracture disturb the coaptation of the broken portions of bone, interrupt the reparative process, perpetuate the fracture, and are sure grounds of the earliest and most exact prognosis in accordance therewith.

---

* Diseases of Bones, 1828, p. 71.
† Cent. v., Obs. 87; Cent. vi., Obs. 68.
‡ Med. Obs. and Inq., vol. iv., No. 37.
§ Richter, Chir. Bibl., b. xi., p. 591.
‖ First Lines of Surgery, S. Cooper, 1840, p. 222.
¶ Nouvelle Doctrine Chir., 1812, t. ii., p. 159.
** Maladies Chir., 1822–26, t. iii., p. 32.
†† Med.-Chir. Trans., vol. v., p. 359.

Thus, *muscular action*, if not counteracted, causes displacement of the broken portions of bone after their coaptation has been effected; and this happens again and again, perpetually disturbing the process of union. The formation of callus is retarded, or imperfect. The *lower* portion of the fractured bone *rides* upon the upper fragment, displacement being accomplished chiefly by the action of those muscles that are inserted into the lower fragment, or part with which it is articulated. For example, fracture of the femur in its middle third is attended with displacement of the lower portion—upwards, inwards, and backwards—by the joint action of the biceps, semi-tendinosus, and semi-membranous muscles. Displacement may be aided by the muscles that are inserted into the upper portion of bone. In fracture of the femur just below the small trochanter, the upper fragment is tilted forward by the joint action of the psoas and iliacus muscles. These instances sufficiently illustrate the general relation of muscular action to the prognosis of fractures.

Frequently moving a fractured limb is equivalent to muscular action in causing displacement of the fragments from time to time. The process of union is delayed. During the forced and hurried marches of a retreating army, cases of fracture have continued for weeks without evincing any disposition to repair under these circumstances. Larrey narrates, that on the retreat of the French army from Syria, many soldiers with compound fracture travelled on the backs of dromedaries and camels. Having undergone this rough riding by day and night for several weeks, these fractures failed to unite with osseous matter, and were sent to Marseilles, a year after the retreat from Syria, still uncured.

Mobility, by reason of the *direction* of fracture, will obviously facilitate the recurrence of displacement. It is unnecessary to exemplify the bearing of this contingency on prognosis, otherwise than by alluding to the tedious reparation of an oblique fracture, shifting to and fro with a see-saw motion, as compared with the speedy union of a transverse fracture in steady apposition; all other circumstances being equal in the two cases.

Apart from any cause of recurring displacement, *coaptation* of the fragments of a broken bone may be *incomplete*, owing to a portion of muscle between them; of which impediment to union two cases were seen by Mr. S. Cooper.* In another instance related by Schmucker, a piece of dead bone intervening between the fragments retarded the process of repair for eight months; when the sequestrum was removed, union ensued in three or four weeks. It is needless to urge the obvious relation of these conditions to prognosis. In all cases, of whatever kind, the persistence or not of the immediate cause in its operation rules our decision, and suggests the first purpose of rational curative treatment.

But in some anomalous cases of indisposition to the formation of callus, no apparent cause exists. The fracture remains ununited, or is very slowly and imperfectly repaired, without any *assignable* cause. An instance of this kind came under my observation when a student, and referring to my note-book, I find the following to have been the chief particulars:—

Disunited Fracture of Femur; Division of substance between ends of bone; Union; Fracture in the same situation a second time; no union.

W. D., æt. forty-eight, a bricklayer, of stout conformation and full stature; has enjoyed good health, and always had sufficient food. When at work he has sometimes drank six or eight pints of beer daily, besides gin.

July 17th, 1847.—While walking on some wet boards his feet slipped; he fell and snapped his left thigh-bone. Was taken to St. Bartholomew's Hospital. The fracture (a simple one) was set, and a long splint applied. At the end of nine weeks the fragments were still moveable.

October 8th.—Was admitted into University College Hospital. The left leg is two and a half inches shorter than the right. At the seat of fracture there is a projection at the outer side of the limb; and when he attempts to raise it in bed, an angle is formed by the upper fragment, and the heel rests on the bed.

* Op. cit., p. 222.

October 24th.—Fourteen weeks after the accident, the patient having been made insensible by ether, the substance connecting the fragments was divided with a narrow knife passed subcutaneously from below upwards between the two portions of bone. The limb was then extended to its proper length, and retained so by a long splint and starch-bandage; a short splint was also applied to the inner aspect of the thigh.

January 21st, 1848.—Three months since the operation. Union has taken place, and is sufficiently firm to warrant the removal of the splint. The opening made in October has not quite healed. There is no shortening of the limb, eversion or inversion of the foot. Phosphate of lime, twelve grains, to be taken three times a day. He moves about the ward on crutches, but cannot support himself on the injured limb.

One day he slipped down in the ward and broke his femur again, at the same point as before; and the operation-wound not having healed, the fracture was compound. This was reduced, and a long splint again applied, but soon removed, and position maintained by an *inner* splint chiefly, for the upper fragment of bone could be seen through the wound. Discharge considerable.

March 27th.—An abscess threatens to form, or matter to burrow, behind the great trochanter. There is union by thickening and consolidation of the soft parts around the fracture; still the ends of the fragments are distinctly moveable. Thus they continued.

In this case the indisposition to union could not be attributed to any apparent constitutional disorder or unusual condition of the part. It was an anomalous case.

Dislocations are, in many respects, parallel to fractures. Among their *predisposing* causes, certain conditions of the articulation itself perpetuate, as it were, a dislocation, by aiding its *recurrence* again and again; thus guiding our prognosis according as these causes in operation are, or are not, irremovable.

Ulceration of the *articular cartilages*, whether this diseased

condition be primary or the sequel of scrofulous caries or synovitis, is conducive to dislocation. Dislocation on the *dorsum ilii* is not unfrequently seen in advanced disease of the hip-joint. *Relaxation* of the *ligaments* likewise disposes to dislocation. The loose, soddened state of the lateral ligaments of a knee-joint affected with chronic synovitis allows free movement of the tibia from side to side, and is a condition, therefore, leading to lateral dislocation. Coexisting destruction of the crucial ligaments and of the articular cartilages will aid this result. But simple relaxation of the ligaments only is sufficient to occasion the perpetual recurrence of a dislocation. A dancing-girl, who, from her earliest years, had habitually twisted herself into various attitudes, eventually became *knock-kneed;* and when the rectus muscle acted on the patella, it was thrown nearly flat upon the side of the external condyle of the femur.* Accumulation of the synovial fluid reproduced this dislocation of the patella in one instance, whenever the limb was extended ; and Sir A. Cooper states† that he had seen several instances of relaxation of the ligaments from distension of the joints with synovia.

Although ligaments are the special means whereby the apposition of articular surfaces is maintained, they are aided considerably, it may be, by the tonic contraction of those muscles which act in the right direction for this purpose. Therefore, as with ligaments, so with these *muscles,*—a weakened, or possibly, in their case, a paralytic condition, disposes to the recurrence of dislocation.

Of mere *weakness,* having this tendency, two instances at least are recorded by the above-mentioned authority. One of recurring dislocation of the shoulder-joint, in the person of a young naval officer, who had been punished by having his foot placed upon a small projection on the deck of the vessel on board which he served, his arm being tightly lashed to the yard of the ship. In this position he was hung for an hour. On returning to England

---

* Dislocations and Fractures of the Joints, Sir A. Cooper, 1841, p. 13.

† Op. cit., p. 14.

he could readily dislocate that arm by raising it towards his head, but a very slight extension accomplished the reduction. The muscles were wasted, as in paralysis. Another instance was that of a man aged fifty, who could at pleasure reproduce dislocation of the hip-joint, and reduce it.

The tendency to dislocation occasioned by a *paralytic* condition was well shown in a case of spontaneous displacement backwards of the humerus, the subject being a young gentleman afflicted with paralysis of his right side. The muscles of the shoulder were wasted, and he had the power of throwing his *os humeri* over the posterior edge of the glenoid cavity of the scapula, from whence, however, it could be easily reduced.

In enumerating the 'persistent causes' of dislocation, I have to notice certain conditions which operate by *perpetuating* the dislocation itself, rather than by predisposing to its recurrence.

The history of *unreduced* dislocations discovers these causes. They are those which oppose reduction, and eventually preclude it; thus far conducting our prognosis.

Foremost is tonic contraction of all those muscles which maintain displacement of the articular surfaces. After dislocation the muscles are tolerably relaxed for a short time, and the limb tolerably moveable. Then tonic muscular contraction begins to act, and increasing more and more, firmly retains the displaced bone in its new position.

Any ligaments which may not have yielded with dislocation, now seem to actually bind the bone down. This is seen remarkably in many cases of displacement backwards of the first phalangeal bone of the thumb upon the distal extremity of its metacarpal bone; an instance of which happened to Liston, under circumstances apparently favourable to reduction. "The accident was very recent, not an hour had elapsed; the patient an old man, and very drunk; no resistance to reductive measures could have been offered by muscular energy, yet very powerful force was applied and persevered in without avail. At last the external lateral ligament was divided by the point of a very narrow and

fine bistoury, and then replacement was immediate and easy. Some inflammation followed, but was kept within bounds, and the man regained the use of the articulation."*

The peculiar *shape* of articular surfaces may perpetuate their displacement. Thus, the prominent margin of the acetabulum opposes reduction in any dislocation of the hip-joint; and the cup-shaped head of the radius is locked in front of the humerus when the former has been thrown forward on the latter.

Such are the conditions which, by perpetuating dislocation, oppose reduction; but analogous persistent causes *subsequently* come into operation, still further opposing reduction, possibly frustrating any attempt, or at least rendering it unjustifiable. The dislocation remains unreduced, and our prognosis is now *further* conducted by knowing the *additional* causes of impediment, and the *period* during which they have continued in operation. Persistent causes still lie at the root of our foreknowledge, and Pathological Anatomy, as usual, supplies the requisite *data*.

The causes in question are, as I have said, analogous to those already considered. The tonic contraction of muscles maintaining the displacement is exchanged for their permanent shortening, and their lines of action become accommodated and adapted to the (new) position which the bone occupies. Dislocation is confirmed by this structural change of the muscles, and as time elapses, it becomes more irrecoverable, and the dislocation consequently more irreducible. Guided by this pathological knowledge, our prognostic expectation of effecting reduction is less sanguine; and proportionately to the duration of muscular shortening and adaptation, our hope declines.

Next, the contiguous portions of bone adapt themselves, forming a new articulation, around which the cellular texture, becoming condensed, assumes the character and function of a capsular ligament; and this condition supervening, further establishes the dislocation. Our knowledge thereof further lessens the

---

* Elements of Surgery, 1832, part iii., p. 337.

prospect of reduction, and, I may add, even the desire to effect it. For here pathology passes into a process of reparation. It is consummated by a partial obliteration of the old articular mechanism; and, as if to guard against reduction, Nature erects a *bony* barrier, in some instances at least—for example, the hip-joint, between the new articulation and any vestige of the original one. All these changes proceed slowly, step by step; but assuredly, whenever a tolerably moveable joint is constructed, in substitution for the original one, adequate knowledge of the structural conditions which then exist, and have led to this comparatively satisfactory result, will at once suggest our prognosis,—the impossibility of an efficient reduction of the displacement,—as well as forbid any attempt to accomplish it.

Aneurisms are no less illustrative of the prognostic import of 'persistent causes.' The essential distinctions described in Chapter III., and which are indicated by the terms 'idiopathic' and 'traumatic' aneurisms, imply persistent or non-persistent conditions, respectively.

Accordingly, traumatic aneurism suggests an unfavourable prognosis, *only* in so far as the lesion itself is progressive. Idiopathic aneurism, however, presupposes some internal cause in operation, which is well known—thanks to Pathological Anatomy—to be no less than calcareous degeneration and consequent brittleness of the artery affected. This condition first induced the aneurism, and being persistent, suggests an unfavourable prognosis, in so far as it tends to perpetuate the lesion. The aneurism, from an almost imperceptible swelling, enlarges more and more, finally bursting with fatal hemorrhage; unless, indeed, its course and tendency be arrested surgically, or by the natural curative process antagonistic to the morbid condition or cause in operation. The cause itself is not and cannot be removed; but the leaky vessel may be closed up and cut off, so to speak, from the main. Even then the same morbid condition probably continues to operate in other portions of the arterial system, inducing *other* aneurisms; so that the original aneurism is only a sample, as it were, of the

fruit borne by one and the same tree, and which is not unfrequently prolific. A crop of aneurisms may be developed, and thus a *single* 'idiopathic' aneurism always suggests a suspicious prognosis. Many illustrative cases are on record. That of Parker, alluded to in Chapter III., is an instructive one. The right femoral artery alone presented four aneurismal sacs. Case No. 9* was *apparently* an aneurism of the femoral artery only; yet, *post-mortem* examination revealed three more in the cavity of the abdomen. These instances are quite surpassed by others on trustworthy observation. Sir A. Cooper† found seven aneurisms in one individual; Pelletan‡ counted sixty-three in one man alone, varying in size from that of a filbert to half a hen's egg; and Jules Cloquet§ contributes a crowning illustration. A crop of some hundreds of aneurismal tumours, ranging from the size of a hemp-seed to that of a large pea, studded the whole arterial system.

The value of the Prognostic Principle advanced in this chapter is further shown by contrasting the significance of 'constitutional' with that of 'local' causes in operation. The former are apt to be persistent; the latter, in many cases, are removable.

Take for example *Inflammation*, in relation to these two great classes of causes. Inflammation of local origin, as when arising from mechanical violence, or the application of a chemical irritant, has less significance, in so far as this morbid condition is more under control, than when the manifestation only of some 'blood-disease.'

A splinter of wood is accidentally thrust into the flesh; inflammation soon arises, proceeding to suppuration, perchance to gangrene. If, however, this removable cause be withdrawn in the first instance, or at least at an early period of its operation,

---

* By D. Monro. Obs. on Aneurism, trans. for Syd. Soc. by John Erichsen, 1844, p. 115.

† Lectures, vol. ii., p. 37.

‡ Clin. Chir., 1810, t. ii., p. 50.

§ Path. Chir., 1831, p. 86, and pl. 1., fig. 14.

then indeed the inflammation will at once subside without its otherwise inevitable consequences.

The significance of this simple instance of traumatic inflammation favourably contrasts with that which proceeds from any of those blood-diseases the operation of which was traced in a former chapter (VIII.). Syphilis, scrofula, the whole tribe of infectious diseases, with scarcely an exception, and other blood-diseases, as gout and rheumatism, manifest themselves by specific inflammations; and the significance of these inflammations is more serious in proportion as their (constitutional) causes are more uncontrollable than local causes of inflammation.

Ulceration and *Mortification*, regarded as consequences of Inflammation, are both, by virtue of their causes, illustrative of the prognostic principle under consideration. Mortification—although essentially one and the same local pathological condition—suggests a widely different prognosis, according to its etiology.

As a consequence of Inflammation, Mortification may arise from tension, or from external pressure. The former cause can be easily overcome by free incisions; and the latter easily removed by mechanical means for relieving pressure—*e.g.*, the water-bed, air-cushions, &c. Mortification, therefore, arising from these causes, is of very different significance to that consequent on various blood-diseases—*e.g.*, carbuncle, erysipelas, malignant small-pox, scarlatina, &c. In such diseases the composition and properties of the whole mass of the blood are seriously damaged; whereas in gangrenous inflammation of local origin, the undue quantity of blood supplied to the part is the morbid condition. This cause is removable, and generally, therefore, of only temporary duration; while the former is less controllable—more persistent.

The proportion of capillary blood-vessels in any part is a *textural* condition conducive to gangrene supervening on inflammation;* but whether *high* or *low* inflammation be contingent thereon, both are amenable to appropriate local measures. Deri-

---

* Chapter x.

vatives and stimulants, respectively, counteract the tendency to gangrene thence arising; and moreover, either extreme of vascularity, having reference solely to the *quantity* of blood in the part, suggests a more favourable prognosis than gangrenous inflammation depending on any blood-disease.

Similar remarks apply to Ulceration consequent on Inflammation.

Mortification and Ulceration have a far wider etiology than as consequences of inflammation, although conveniently associated therewith for practical purposes. They may severally arise—without previous inflammation—from many other causes; all of which, however, are referrible to the blood, in respect either of its defective quality or of the quantity supplied, and possibly a deficient supply of nervous energy to the part. These causes are fully considered in Chapter VIII., and by reference thereto, each will itself suggest its own relationship to the Prognostic Principle involved in persistent and non-persistent causes.

Thus, respecting 'blood-diseases' and non-inflammatory Mortification; Ergotism, for example, denotes the operation of a blood-poison,—ergot of rye having this issue. Most varieties of non-inflammatory ulcers are so many expressions of blood-diseases; thence, the irritable ulcer, the phagedænic, the indolent, the strumous, the lupous, the syphilitic, the scorbutic, the cancerous, and rodent ulcers.

All these forms of Mortification and Ulceration, arising from causes of a persistent character, suggest an unfavourable prognosis.

On the other hand, whenever the cause in operation is purely local and removable, the prognosis becomes equally favourable.

One variety of ulcer, in particular, is obviously of local origin. The varicose ulcer depends solely on a varicose state of the neighbouring veins; and this ulcer heals, in all but the most chronic cases, when its local and removable cause is removed.

Turning to Mortification, as depending on a deficient *quantity* of blood supplied to the part affected; the occasions of such

deficiency need only be enumerated in order to perceive their relation to the prognostic principle advanced.

Morbid states of the larger arteries;—represented by their ossific degeneration (and senile gangrene), occlusion of the vessel, arteritis, embolon, injury of the inner coat of a main artery, aneurism, punctured wound of a main artery. Morbid conditions of the larger veins;—phlebitis with occlusion of the vein, phlebolithes, fibrous obliteration of a vein, aneurismal varix and varicose aneurism, pressure on large veins from without, by tumour or by ligature.

Lastly, defective supply of nervous energy—*e.g.*, from pressure on a large nerve—may induce mortification or ulceration; and the persistency or otherwise of any such cause will also suggest a corresponding prognosis.

The 'external' causes,—represented by mechanical violence (with traumatic gangrene), cold, heat, chemical escharotics, and animal poisons inoculated, have less immediate relation to the same principle; but so far as mortification ensues in proportion to the *duration*, as well as the intensity of these causes, prognosis is determined accordingly.

The blood-origin of *malignant growths*, as compared with the production of innocent tumours, affords another admirable illustration of the guidance of persistent causes in prognosis.

Among the many proofs of the blood-origin of cancer, none is more cogent than that, after removal by operation, this growth is very apt to reappear *in situ*, and in distant parts, growing also in many kinds of texture simultaneously. If cancer reappeared only in the texture or organ first affected, it might be attributed to incomplete extirpation of the original growth,—that some portion having been accidentally left, had formed the germ of its successor; but the reappearance of cancer not only *in situ*, but in distant parts also, points to some cause unconnected with the particular texture or organ first or subsequently affected—points, indeed, to an error of nutrition prevalent throughout the body, and referrible only to some morbid condition of the blood, as the

parent source of the nutritive material. Cancer, then, is essentially a *blood*-disease. No wonder, therefore, that when one local manifestation is removed, another makes its appearance, *in situ* and in distant parts simultaneously.

The persistence of this blood-disease in operation suggests the grand distinction in our prognosis of malignant growths, as compared with that of innocent tumours. These growths do not *recur* when completely extirpated; *e.g.*, the fatty tumour is non-recurring: consequently they are of local origin. When once extirpated, there is an end; while the cause of cancer—the blood-disease, of which it is the offspring—being still persistent, reproduces new growths, as buds from the parent trunk.

True *degeneration* of structure likewise (not disintegration, the result of inflammation, for example) would appear to be the local expression of blood-disease; and among other reasons, because of the simultaneous manifestation of degeneration in many organs and textures *indiscriminately*, thereby precluding the hypothesis of its local,—textural origin. And with this constitutional cause in operation, the prognosis of degeneration acquires a most serious significance altogether apart from the special physiological importance of the particular organ whose degeneration may be diagnosed during life. For the great probability being that many organs—*e.g.*, the heart and liver—have undergone this destructive change of texture simultaneously with degeneration of the kidneys, overshadows our prognosis with evil omen; while this very ground of apprehension is a crowning illustration of the Prognostic Principle implied, by the grave import of persistent causes in operation.

CHAPTER XIII.

THE PROGNOSTIC GUIDANCE OF CLINICAL PATHOLOGICAL ANATOMY (CONCLUDED).

The earliest and most exact prognosis of an injury or of a disease is *regulated* by the kind and extent of *structural* alteration that the organ or texture has undergone; and moreover, by the period during which such alteration of structure has been in operation as an internal cause of functional disturbances of the system. Nevertheless, an acute disease, or a recent injury, is, *cæteris paribus*, more unfavourable than a chronic lesion, to which the system has become habituated.

The prognostic value of the foregoing fundamental Principle, illustrated by Injuries—wounds, other than simple (incised); fractures and dislocations, respectively, other than simple; and by aneurism—diffused. Also by diseases of Nutrition—results of inflammation; growths—malignant.

Pursuing a comprehensive survey of the course, tendency, and issue of injuries and diseases, chiefly of a surgical character; analysing, comparing, and generalizing as we proceed, in order to eliminate the Principles which guide all Prognosis, we arrive at one of predominant importance, next in rank to that advanced in the last chapter, and of which this is a further and full development.

The Prognostic Principle involved in 'alterations of structure' is a continuation and completion of that represented by 'internal' causes, but of less general significance, for it specifies the *kind* and *extent* of cause in operation. Abstractedly, anything having the relation of 'cause' is primarily important in prognosis; 'persistent' causes, therefore, assume this position, while 'alteration of structure,' having reference to its kind and extent, will *regulate* our prognosis.

The value of this Principle of Prognosis admits of illustration by the career of injuries and diseases indiscriminately. It will

suffice to select some of the more important which fall within the range of General Surgical Pathology.

Of *Wounds;* the punctured, contused, and lacerated, as compared with the incised wound, supply familiar examples of the prognostic guidance afforded by the *kind* of structural alteration.

An incised wound *per se,* and apart from unfavourable circumstances, independent of the lesion itself, heals by primary adhesion. This career may be forthwith and assuredly foretold. Compare with it the course and tendency of all other wounds. They slough, suppurate, and heal only by granulation and cicatrization. This widely different career may likewise be foretold with equal promptitude and certainty.

But our foreknowledge in either case is regulated solely by considering the *kind* of injury,—contusion and laceration, as compared with that implied by an incised wound.

Scarcely less significant is the *extent* of structural alteration which an organ or the textures of a part have undergone.

Compare the compound and the complicated varieties of *Fracture* with a simple fracture.

The crushing force that occasions a simple (contused) fracture may bruise and tear the soft tissues down to the seat of fracture; or again, the broken ends of bone may lacerate the soft parts and protrude through the skin. In either case a wound is made, leading from the surface down to the seat of fracture, thus constituting a compound fracture. This lacerated wound may be regarded as an *extension* only of the fracture to a similar lesion of the neighbouring soft textures; yet it rarely heals by the first intention, but is followed by suppuration, often profuse,—by sloughing, and slow formation of a uniting callus. The prognosis of compound fracture is regulated, therefore, by reference to this extent of structural damage.

Complicated fracture affords a further illustration of the same principle. The kind of lesion is essentially that of simple fracture; but the *extent* of injury—the fact of other and more important parts than bone being involved, affects considerably its

course, tendency, and issue. Thus, fracture may be complicated by the rupture of a large artery or nerve, or by laceration of the muscles, or it may extend into a joint, or be complicated by simultaneous dislocation. Internal organs also are implicated by certain fractures. Fracture of the pelvis may implicate the bladder; broken ribs may wound the lung or pericardium; and a depressed fracture of the skull will injure the brain. These and other complications—these extensions of structural damage are very unfavourable accompaniments of fracture, and regulate our prognosis accordingly.

Taking, then, these well-known instances, it is not difficult to discover the grounds of our foreknowledge. The extent, no less than the kind of structural lesion, is the Principle specially inculcated by analysing compound and complicated fractures, in relation to their prognosis.

In like manner it is not difficult to perceive that the Principle thus evolved supplies the earliest and most exact prognostics. No prognosis of fracture, for example, is so early and exact as that which is regulated by reference to the extent, no less than the kind of structural lesion, implied by compound and complicated fractures respectively.

It will be observed that the *extent* of lesion is particularly inculcated by prognosis relating to fracture. The kind of lesion is, in all cases, the same; a lacerated or contused wound, limited to the bone,—simple fracture; or extended to and through the skin,—compound fracture; or to neighbouring textures and organs,—complicated fracture.

The prognostic value of this element—the extent of structural lesion, is further enforced by compound and complicated *Dislocations*, which, being analogous to fractures, so named, have a similar relation to their prognosis. And in either case the earliest and most exact prognostics are supplied by considering the extent, no less than the kind of structural lesion.

This Principle is again illustrated by diffuse *Aneurism* as compared with the circumscribed.

The large and *increasing* size of diffuse aneurism is the consideration which regulates our prognosis, more especially respecting aneurism of idiopathic origin; for it then denotes an advanced and progressive condition of this lesion, without the degree of reparative power inherent in one of traumatic origin.

On the other hand, the prognostic value of the *kind* of lesion was equally conspicuous in the prognosis of wounds.

The combined influence of *both* these elements—kind and extent of lesion—in regulating Prognosis, is the most important aspect of Pathological Anatomy with regard to this Department of (Medicine and) Surgery.

So very many forms of injury and disease have equal claim to our consideration from this point of view, that selection is difficult. I might adduce all those forms of injury which have already been noticed. Thus the prognosis of wounds, fractures, dislocations, and aneurisms, severally exhibit the guidance afforded by a due consideration of the kind *and* extent of each of these lesions.

The results of *Inflammation* contribute a series of most apposite illustrations.

The *kind* of result, in the first place, has a most important significance. Lymph-effusion, as compared with mortification, is an event of a widely different prognostic import. Reparation, possibly by the former; destruction, certainly by the latter; and between these two extremes various subordinate degrees of difference intervene by virtue of the kind of result. Lymph, pliant and plastic, is not so damaging to structure as when it is less organizable; nay, in some cases, adhesions wrought by plastic lymph work together for good. Instances of this kind are given in the introductory Elements of Pathological Anatomy.

Then, again, suppuration, as compared with lymph-effusion, represents the transition from *some* degree of reparative effort to an inert fluid of no reparative capacity in the animal economy. Furthermore, suppuration within the substance of any part of the body itself, implies a previous coextensive destruction of texture; the formation and accumulation of pus being necessarily

preceded by the death and removal of those textures whose place it usurps. Mortification here advances hand in hand with suppuration, and *pari passu.*

But mortification may itself be the most conspicuous result of inflammation; and then, indeed, its contrast with the reparative tendency of lymph-effusion is most obvious.

Nor is the significance of these results confined only to the seat of inflammation. The prognostic importance of that fever which supervenes on prolonged suppuration, is, *cæteris paribus,* less than that which depends on mortification. Hectic fever is less speedily and surely fatal than gangrenous typhoid under similar circumstances.

The *extent* of this or that result of inflammation has likewise its share of significance.

Adhesions are of serious import, in proportion as they are extensive, by shackling the functions of whatever organs have thus become attached. At the same time, the physical character of the adhesions,—their degree of pliability and ductility, should be taken into account.

Suppuration is destructive of organization proportionately to its extent in the organ or part affected. This consideration explains the more serious character of diffused suppuration, compared with an abscess. Pus diffused among the textures detaches their continuity, and intercepts their supply of blood. The cellular texture especially is destroyed, and the muscles, blood-vessels, and bone dissected. Hence coextensive sloughing of the skin, fasciæ, and muscles; hence, moreover, necrosis. Witness the consequences of phlegmonous erysipelas supervening on a simple incised wound, and the course of certain compound fractures and dislocations.

Lastly, the prognosis of *Growths* is regulated by reference to their kind *and* extent of structure, individually—*in so far as* the same career is invariably associated therewith.

Cancer—the type of malignant growths—is infiltrating; and with the condition of structure thus produced is invariably asso-

ciated a peculiar course and tendency, which likewise exhibit the prognostic significance of the extent to which this species of growth has attained. Cancer, continuing to infiltrate surrounding textures, contracts adhesions, and tends to ulcerate. Contiguous lymphatic glands become affected; the disease is prone to appear in distant parts of the body, and to grow simultaneously.

Such course and tendency are synonymous with the term 'malignancy,' by virtue of which cancer contrasts with every other known species of growth, excepting certain recurring tumours—*e.g.*, myeloid and fibroid, which are more or less malignant, although structurally dissimilar. While, therefore, our diagnosis of malignant growths cannot be determined by any particular condition of structure, yet, given the kind of structure and its extent, we can determine our prognosis.

Now, regarding diseases of structure as 'internal causes,' their *duration* is an element to be considered in relation to Prognosis. It might be supposed that any morbid condition of structure would assume increasing importance, exactly in proportion to the period during which it has been in operation; that duration would be the measure of its importance. Analogy supports this anticipation. Mechanical causes are thus estimated. A force, represented by one pound, acting at one end of a simple lever, is equivalent to another of two pounds resisting at the other end—provided only that the one-pound force is in operation for twice the time; and so on in the same proportion. This is the principle of all the 'mechanical powers,' so called; and, by the compensative element of time or duration, forces of very different intensity may be brought into competition and balanced; or, a lesser, yet enduring force, may be more than equivalent to one of far greater intensity. The same principle of duration is also a measure of the prognostic importance of internal causes, but *less exactly*. For the bodily organism is endowed with an opposing power—that of readjusting or accommodating itself, as a whole, and each of its several parts, to whatever local morbid condition of structure may be in operation; and this accommodating toleration is almost in

proportion to the duration of the internal cause. Any such cause, therefore, loses its efficacy and importance, almost in proportion to the period during which it has been in operation. Recent causes are proportionately of greater prognostic import. An acute disease, or a recent injury, is, *cæteris paribus,* more unfavourable than a chronic lesion, to which the system has become habituated.

These abstract propositions are amply confirmed by clinical observation; and, indeed, the "toleration of disease" or injury is so familiar to common experience as to render illustration almost unnecessary. The 'toleration' of a foreign substance by any part of the body into which it has been introduced is much a question of *time.* When first a thorn is thrust into the flesh, inflammation and suppuration ensue, to dislodge and eject the intruder; this failing, then, after some time has elapsed, the thorn remains unopposed—the part has accommodated itself; and even a much larger foreign body—*e.g.,* a bullet—may become encysted, and is then tolerated, without any further opposition, as an old tenant. This innate power of readjustment sometimes succeeds in completely restoring an injured part to its original condition of health, or apparently so—a subject on which I shall enter at large in a subsequent chapter.

The *periodic* operation of an internal cause, as witnessed in the periodic return of intermittent fever, is another character of internal causes, but one less readily explained. Hence the prognosis of periodic diseases can be determined only by experience, and is empirical.

CHAPTER XIV.

THE SUPERIOR PROGNOSTIC GUIDANCE OF PATHOLOGY.

Pathology declares the course, tendency, and issue of diseases and injuries, by their continued Functional manifestations.

Principle I.—The comparative Functional importance of any texture or organ *determines* our Prognosis, unfavourable or favourable, respecting the course and tendency of any morbid condition of structure it may have undergone.

Sub-Principle I.—Organs and textures which fulfil Functions by virtue of their vital endowments and chemical composition, suggest an unfavourable Prognosis.

The Prognostic value of this Principle and sub-Principle illustrated by the course and tendency of Blood-diseases—*e.g.*, Syphilis, Scrofula, Gout, and Rheumatism; also by disorders of the Nervous System—*e.g.*, Constitutional Irritation; by disorders of the Nervo-Muscular System—*e.g.*, Tetanus; and by disorders of Secretion and Nutrition.

Sub-Principle II.—Organs and textures which fulfil Functions by virtue of their physical properties and mechanism, suggest a more favourable Prognosis.

The Prognostic value of this sub-Principle illustrated by the course and tendency of Injuries—*e.g.*, incised flesh Wounds; reduced simple Fractures and Dislocations.

Principle II.—Local disease or injury, *per se*, suggests a favourable Prognosis.

Principle III.—Local disease or injury, sustaining, or sustained by, some Constitutional disorder, suggests an unfavourable Prognosis.

The Prognostic value of this Principle illustrated by the course and tendency of unreduced Fractures and Dislocations; also by persistent Inflammation; and by poisoned Wounds.

Principle IV.—Constitutional diseases, implying each some morbid condition of a texture or textures, of general distribution, as well as of predominant Functional influence throughout the system, suggest an unfavourable Prognosis.

The Prognostic value of this general Principle illustrated by the course and tendency of the same diseases of the Blood—disorders of the Nervous System, Nervo-muscular System, and of Nutrition—already adduced in illustration of the first Principle advanced in the present chapter.

Modes of Dying.

How paradoxical it seems that Pathological Anatomy should determine the immediate cause in operation, with its structural condition and extent, most exactly and at the earliest period of disease or injury, and at the same time not determine our prognosis of its

course, tendency, and issue. Yet so it is. Pathology can alone supply this deficiency. Whence its superior prognostic guidance? Obviously, by virtue of its *significance.*

Functional disturbances have, indeed, no existence apart from the Structural conditions representing their internal causes; but alteration of Structure, physical properties, and perhaps chemical composition, singly or conjoined, have no significance apart from the disturbance of Function they induce and perpetuate,—which, representing the course and tendency of morbid conditions of Structure, &c., alone concern Prognosis. Otherwise, *they* are dead or inert, as causes.

But although Pathological Anatomy certainly bears to Pathology the relation of cause to its effect, this relation is uncertain,* as regards the kind and degree of Functional disturbance thereby induced.

While, therefore, the *data* of Pathological Anatomy *regulate,* those of Pathology can alone *determine,* Prognosis. The former guide, subject to the correction of our foreknowledge, by estimating the kind and degree of Functional disturbance manifested through the internal cause in operation.

This Principle, suggested by these *à priori* considerations, will be established, as hitherto, by analysing a series of known and acknowledged instances of Prognosis, and thence evolving the source of such foreknowledge.

The Prognostic province of Pathology will be discovered to comprise different aspects of its determining power—certain very important expressions of the same Principle; and, first in order, I have to verify the leading one advanced in this chapter—that 'the comparative Functional importance (and influence) of any texture or organ determines our Prognosis, unfavourable or favourable, respecting the course and tendency of any morbid condition of structure it may have undergone.'

What, then, is the acknowledged prognosis of all *Blood-diseases,* as gathered from the experience gained by repeated observation of

---

* Chapter ii.

their career? In two respects, at least, they all agree. The proneness of all such diseases to engage many organs simultaneously, and their liability to shift their local manifestations from one organ to another—from the skin to the lungs, to the heart, to the gastro-intestinal mucous membrane; and hence the proportionate liability of some organ becoming affected, the integrity of which is essential to life.

Syphilis, scrofula, gout, and rheumatism, are apt illustrations of this precarious state of existence. Nay, moreover, these particular blood-diseases, or their tendencies, are transmitted from parent to offspring, and their hereditary taints may cling even to successive generations.

Guided, then, by their history, as taught by experience, blood-diseases are pregnant with evil omen, and suggest an unfavourable, or at least a precarious, prognosis. But the blood ranks high indeed among the component *textures* of the body in respect of its *physiological* importance. Accordingly the Principle involved in our prognosis of blood-diseases is that which I have already enunciated. And 'the comparative Functional importance of any *organ*,' in relation to our Prognosis of its morbid conditions, is shown by analysing the career of diseases, of which the *Central Nervous System* is the primary source of all the morbid phenomena.

Shock, consequent on injury, suspends the heart's action, more or less; or, without syncope, subdued respiration is the most prominent peril. These are the most usual symptoms. From this state (of shock) both the nervous and vascular systems may revive, and reaction may reach only the healthy state of equilibrium, may fall short, or pass beyond it. Or again, a mixed state of prostration, with ineffectual reaction—prostration of muscular power—combined with nervous excitement, may supervene: and what are the perils then imminent? The heart's action is apt to fail, and death ensue, albeit almost at the moment of frenzy. But the heart and central nervous system are both organs of high functional importance in the body, and therefore exemplify the

counterpart of the prognostic principle involved in the prognosis of blood-diseases.

The prognosis of tetanus is also unpropitious, and why? Because the central nervous system—an organism of high functional importance—is the source (I do not say the cause) of all the morbid phenomena, whereby many organs are liable to consentaneous or consecutive participation. And in this wide range of participation there is danger. Life is perilled by the dominant functional influence of the central nervous system. Thus, the heart's action may be suddenly arrested by the metastasis of tetanic spasm. "From the plunge of the cold bath," writes Travers,* "I have seen the tetanic patient brought up a corpse."† Again, a gentleman under the care of Mr. Key, who had just recovered from a severe attack of tetanus, was thrown into a fit of passion by some ill-timed communication relating to his property, and died on the spot, probably from spasm of the heart. More frequently, however, the imminent danger is asphyxia, from spasm of the muscles of the glottis, and of the diaphragm.

Events such as these in the career of tetanus confirm the prognostic value of the guidance afforded by duly estimating the comparative functional importance of whatever organ is the primary internal cause of all the morbid phenomena which arise in the course of any given disease or injury. And this principle of prognosis holds good in respect of diseases of textures, as well as of organs. Morbid states of the blood, for example, considered in relation to their consequences, furnish, as we have seen, fruitful illustrations.

Diseases of the *excreting* textures and organs owe their prognostic significance to the grave functional importance of these structures.

Such diseases operate as internal causes through the medium of the blood, and their prognosis therefore is unfavourable, or, at least, precarious. Thus, rheumatism, consequent on checked secre-

---

* Constitutional Irritation. Further Inquiry, 1835, p. 301.

† See also, A Treatise on Tetanus, J. Morrison, 1816.

tion by the skin, is prone to involve the heart, and, by inducing pericarditis, assume a fatal character; while gout, arising from defective excretion of uric acid by the kidneys, may suddenly be transferred to the stomach—an equally perilous event.

In like manner, all those diseases of Nutrition which poison the blood with the products of secondary mal-assimilation, are perilous, or, at least, precarious.

Taking for granted that the comparative Functional influence of any texture or organ determines our Prognosis of its morbid conditions, there is yet another Prognostic Principle subordinate thereto, and which is implied by the instances of prognosis adduced in illustration of the first proposition.

The sub-Principle alluded to is this:—that 'morbid conditions of organs and textures which fulfil Functions by virtue of their 'vital' endowments, or by their 'chemical' constitution, suggest an unfavourable prognosis.' Thus, it is a significant fact that blood-diseases—such as syphilis, scrofula, gout, and rheumatism; morbid states of the nervous system,—*e.g.*, constitutional irritation; those also of the nervo-muscular system,—*e.g.*, tetanus; and diseases of secretion and nutrition,—all agree in two particulars. They are diseases of unfavourable, or at least precarious, course and tendency; and they are diseases of organs or textures, the functions of which are severally fulfilled by virtue of vital properties, peculiar chemical constitution, or both. Such are—the plastic power of the liquor sanguinis, and its chemical character, as the source of every structure; the vital property known as the *vis nervosa;* that also of muscular contractility; the power of secretion, and that of assimilation.

The superior functional importance of structures having these powers or endowments is not inexplicable. Without them the body would be a piece of mechanism only, of no greater functional capacities than those of any other machine constructed artificially. The superaddition and possession of vital properties, and of peculiar chemical constitution, confer the functional capabilities, which together constitute our idea of 'life.' The lifeless body may retain for a time its mechanical powers, by virtue of its mechanical con-

struction and physical properties; but the living body alone possesses *vital* powers, by which the heart beats and the blood circulates: innervation sheds its influence on every process, and the act of respiration is performed; excretion purifies the blood, and nutrition constructs and reconstructs the entire organism. All the rest is either physical, or the result of chemical processes, such as may occur in the world of unorganized matter, and can be imitated *artificially*.

The chemical changes contingent on the act of respiration are of this nature; and digestion is, chemically speaking, a process of solution, whereby the various constituents of the blood, preformed in the food, are rendered capable of absorption; while absorption itself is but a process of purely physical character—it conforms to the laws of hydraulics. Turning to the functions of animal life, witness the apparatus of the eye and ear, and their functions. They are exemplifications of the laws of optics and acoustics respectively. We recognise the triumph of mechanical skill in the osseous framework of the body and the various actions of the muscles, whereby a thousand different movements are executed with ease, strength, and precision.

But superadd to all this the vital powers of the blood and its unique chemical composition; the power also of innervation, muscular contractility; that of secretion and assimilation,—as each of these endowments are displayed by their operation in the several compound functions of organic and animal life,—and we begin to realize the conception of a *living* body. Moreover, this living body, in its various states of *disease*, exhibits the superior importance of those special and peculiar forces, to which physical properties and mechanism are subordinate and subservient. Hence, morbid conditions of organs and textures which fulfil functions by means of their vital powers and chemical constitution, suggest an unfavourable Prognosis. Such is the *rationale* of this Prognostic Principle.

It may be inferred as a corollary, that in respect of 'organs and textures which fulfil Functions through their 'physical properties and mechanism,' *their* morbid conditions suggest a *more*

favourable Prognosis.' But *this* sub-Principle scarcely admits of illustration. For the functional dependencies of all parts of the body are so intimate that the whole sympathizes with every part, and thus the disorder of any portion becomes the disorder of the whole organism. Nevertheless, an approach to isolation is seen in the healing of *incised* flesh-wounds, and that of *reduced* simple fractures and dislocations. Reparation proceeds uninterruptedly in many cases, without any notice by the system, after the first shock is over. These simple injuries of mechanism are in fact, comparatively speaking, isolated and excluded from any systemic complication; and they illustrate the comparatively more certain and favourable prognosis of morbid conditions, where the organs and textures affected fulfil their functions severally by virtue of physical properties and mechanism. Vital powers are not concerned, or, at least, not otherwise than beyond the *local* nutritive reparation of the injured part.

In like manner is demonstrated and illustrated another Principle,—that 'local disease or injury, *per se*, suggests a favourable Prognosis.'

There are certain apparent exceptions to this rule. For example, the larynx fulfils the physical (pneumatic) function of allowing the free ingress and egress of air, to and from the lungs, —a function, without the continued alternation of which, life ceases in three minutes or so. Morbid states of this organ—*e.g.* laryngitis—are most perilous. Yet the function of the larynx is purely mechanical. But this, and all other apparent exceptions to the inferior prognostic importance of such organs, can be readily explained, and indeed, when analysed, confirm the rule,—that, *per se*, their morbid conditions are less perilous.

Agreeably to the functional relationship subsisting between different parts of the body, the larynx is itself subservient to the aeration of the blood, and thus *acquires* a high degree of functional importance. As the only channel through which inspiration and expiration are naturally accomplished; the larynx, mechanical as is its own function *per se*, acquires vital importance by direct subserviency to the chemical constitution and endowments of the blood.

Our prognosis, therefore, of any morbid condition of this organ, is determined by the essential importance of its relation to the *blood*.

Lesions of a mechanical organ may thus become equivalent to disorders of vital character, and our prognosis accordingly should be estimated by the course and tendency of these *perilous* consequences.

Similar considerations establish the prognostic value of another Principle ;—that 'local disease or injury sustaining some Constitutional disorder, suggests an unfavourable Prognosis.' Examples are of daily occurrence in practice.

Unreduced fractures and dislocations owe their constitutional significance to the continuance of shock, which is sustained by the damage still inflicted on the nerves involved about the seat of injury. And if this be true of simple fracture and dislocation, how much more so of unreduced compound fracture and dislocation. Shock is still more persistent.

In Chapter IX. I had occasion to notice the persistence of shock under these circumstances ; and I then endeavoured to trace the intimate relation between it and tetanus.

But this continued prostration is more perilous than temporary shock, and tetanus is perhaps yet more perilous.

Clearly, therefore, the Principle involved in our unfavourable prognosis of such cases is this—that they are instances of a local cause sustaining a constitutional disorder.

Any occasion of *persistent* acute inflammation affords equally convincing proof of the same principle. Inflammatory fever is aggravated ; hectic, or gangrenous typhoid, supervenes ; and the prognosis becomes more unfavourable : for the local cause in operation sustains either of these constitutional disorders. Hence the prognostic significance of contused and lacerated wounds, and of compound fractures and dislocations—as compared with the simple forms of these injuries, which far more readily heal, and the accompanying constitutional disorder, or fever, subsides.

Poisoned wounds, so called, allow of the *experimentum crucis* in

proof of the Prognostic Principle I am advocating. For if the constitutional disorder, otherwise inevitably consequent on any such wound, can be prevented by timely withdrawal of the poison, or by excision of the wound itself,—*i.e.*, by removing the local cause in operation,—our prognosis at once becomes proportionately favourable. Repeated experience corroborates the *à priori* probability of a happy issue, when the perilous constitutional disturbance is no longer sustained. The results of numerous cases on record are to this effect. Respecting hydrophobia, for example:—in the experience of Professor Colles, three persons were bitten by the same dog, at the same time; two of them suffered the part to be excised, and they escaped; the third refused, had hydrophobia, and died.

The Prognostic Principle obviously implied by these and similar cases could be further illustrated, were it necessary, by the history of all other poisoned wounds; but, granting the great prognostic importance of 'local disease sustaining constitutional disorder,' it suggests the equal prognostic significance of 'local disease sustained by constitutional disorder.' Thence also we are led to consider another Principle of Prognosis,—the last of *general* application. It is, that 'Constitutional diseases,—implying as they do, each, some morbid condition of a texture or textures of general distribution, as well as of predominant functional influence throughout the system—suggest an unfavourable Prognosis.'

To rightly estimate the value of this Principle, be it observed that the leading idea which the Prognostic guidance of Pathology represents is, that the significance of Functional disturbances is to be measured by the *range* of their influence *throughout* the body. The comparative functional influence of any organ or texture, considered with reference to our prognosis of its morbid conditions, exhibited this law. The more extensive the range of its functional influence, the more important are the diseases of any part. Accordingly, organs and textures which fulfil functions by virtue of 'vital' endowments, are of superior prognostic significance to those which may be termed, by way of distinction, 'mechanical' organs and textures. Again, 'local disease sustaining some 'con-

stitutional' disorder suggests an unfavourable prognosis.' And why? Because the term 'constitutional' refers to some source of predominant functional influence throughout the body,—*i.e.*,to vital powers. But if to this be superadded the *general distribution* in the organism of the *structure* affected, we then fully realize what is meant by a constitutional disorder. Thence also their great prognostic significance. Experience amply confirms it. Diseases having this twofold character of structural extent, and functional range of influence, augur *most* unfavourably. Vital powers are, indeed, predominant over mechanical functions; but the term constitutional denoting that which is structurally prevalent, as well as functionally predominant, any disease of this twofold character is the most perilous. Such are diseases of the blood and vascular system; those also of the nervous system; and perversions of the all-prevalent function of assimilation,—they being possibly coextensive with all the textures.

If constitutional disorders are not always the most perilous, they are ever liable to become so; and are, at least, always precarious. Their prognosis is in all cases uncertain, owing to their essential characters of structural extent, and functional range of influence. Some such (constitutional) diseases fall more especially within the province of surgical practice. Scrofula, syphilis, gout and rheumatism, among diseases of the blood; shock, continued prostration, and tetanus, among those of the nervous system; and certain instances of secondary mal-assimilation among those cited and examined in Chapter X. All these diseases derive their prognostic significance partly from the fact, that 'vital' powers are disordered; but partly also from the additional circumstance, that the textures concerned are very *generally distributed* throughout the whole body.

To conclude the prognostic guidance of Pathology, it is necessary to understand the various ways in which diseases and injuries may end fatally, or the modes of dying. They are conveniently arranged under six heads.

Death may begin—

Firstly, by cessation of the circulation—Cardiac syncope, Asthenia.

Secondly, by Coma and Paralysis.

Thirdly, by Asphyxia } Necræmia, Infection,
Fourthly, by Suppressed Excretion } Contagion.

Fifthly, by Starvation—Acute Atrophy.

Sixthly, by Marasmus—Chronic Atrophy.

All these modes of dying lead to each other. Beginning therefore in any one way, death results eventually from the association and co-operation of the rest.

Or again, two or more modes of dying may commence independently, yet simultaneously, and lead to the supervention of the rest more speedily; just as a fire, lit in two or more places at once, sooner consumes the whole combustible matter. So in pathology; by fatty degeneration, for example, of the heart and kidneys, asthenia and uræmia, beginning together, co-operate, and rapidly extend their fatal influence.

Physiology explains these associations and co-operations.

All the six compound functions—circulation, innervation, respiration, excretion, digestion, and nutrition—together form a *circle*, as it were, consisting of so many links. By the failure of any one such function, the rupture of any one link, the bond of life is broken, and death beginning thence, as the starting point, entails, sooner or later, the failure of the remaining functions. If more than one such link be snapped at once, the circle falls to pieces sooner—death ensues more speedily.

But certain of these compound functions are more *essential* to maintain the circle of life unbroken; and the beginnings of death have therefore different degrees of prognostic value. They are all resolvable into two:—cardiac syncope, or asthenia, as the case may be,—implying defective circulation of a due quantity of blood; and necræmia,—implying defective quality of the vital fluid.

In whichever way death begins; in order to prognosticate the fatal termination of a disease or injury, it is absolutely necessary to know and be able to distinguish the particular functional

disturbances or *symptoms* which characterize each of these several modes of dying.

Firstly. Death by *cardiac syncope*, and consequent cessation of the blood's circulation, is manifested by sudden pallor, with arrest of the heart's action and of the pulse, immediate insensibility; the individual falls down with a gasp, the respiration has ceased, and all is over. This mode of dying is particularly an instance of *sudden* death, and the shock of severe injury a good illustration of its cause. Paralysis of the heart is immediately induced, whereby this organ loses its (vital) irritability; or tonic spasm may be induced, whereby it refuses to undergo relaxation, and remains contracted.

Asthenia denotes the *gradual* failure of the heart's action, and of the circulation. This state is preceded by symptoms of similar character and import, but of some duration. Pallidity gradually overshadows, with coldness of the extremities; the heart's beat is feeble, the pulse languid, intermittent, now slow, now quick, and the mind clouded. Occasionally a temporary reaction may ensue, like the glow of burnt embers reviving from time to time. Death is almost imperceptible; or, in some cases, life is suddenly extinguished. Death slowly accruing from fatty degeneration of the heart is preceded by these symptoms, although the act of death itself is generally sudden.

Death by *coma* implies the cessation of the cerebral functions. Insensibility, and loss of voluntary power, are therefore the primary symptoms. The heart still retains its contractile power, and the pulse-beat fails not. It is, however, slower and fuller than usual.

These symptoms may occur quite suddenly and overwhelmingly, as from depressed fracture of the skull, or from knock-down apoplexy. They may, however, supervene slowly, and by instalments. Obscurity of the mental faculties, with partial paralysis and perversions of the special senses, singing in the ears, flashes of light before the eyes, are then the primary symptoms. But in either case, the loss of the cerebral functions does

not *kill*. Insensibility and paralysis are not of themselves fatal prognostics. They become so only in proportion to the loss also of involuntary excito-motion, and especially as regards the act of respiration.

With overwhelming coma, the breathing is much embarrassed and stertorous; and besides this most perilous accessory, all other functions depending on involuntary excito-motion fail. The pupils dilate, and refuse to obey the stimulus of light; deglutition is not excited by the presence of food in the fauces; vomiting cannot be aroused by the most powerful direct emetics; the sphincter muscles yield, so that the urine and fæces escape. All these symptoms betoken the approach of death. Their significance lies in the fact, that with the general failure of involuntary excito-motion, respiration does not, or rather cannot, escape; and *this* function is essential to life.

Paralysis arising from disease or injury of the *medulla oblongata* is equally fatal, by apnœa; but this mode of dying contrasts with that beginning in the *medulla spinalis—i.e.*, by the failure of its functions. In the former, respiration is necessarily stopped; in the latter, the act of breathing may remain unimpaired. Physiology readily interprets the different prognostic importance of these two cases.

The *medulla oblongata* is that nervous centre by the integrity of which respiration is sustained, and failing which, it ceases. Moreover, if the pneumogastric, or great afferent nerve of respiration, be injured, the respiratory act ceases, or becomes insufficient to maintain life; and the same result attends any impediment to the course of reflex nervous influence from the medulla oblongata through the efferent nerves of respiration—the phrenic, intercostals, and spinal accessory.

Paralysis arising from disease or injury of the *medulla spinalis* is fatal only in so far as it involves these efferent nerves. But if not surely fatal, such paralysis acquires an ominous character, when, besides the loss of sensibility and voluntary power, the damage done to the spinal cord precludes involuntary excito-motion

also. For then, the vesical and anal sphincters ceasing to contract, involuntary micturition and defecation render life miserable and undesirable. Not only so; the excrements are now worse than excrementitious—the urine rankly ammoniacal and fetid, the fæces decomposed and putrid. Both are poisonous; the one inflaming the bladder and excoriating the skin over which it dribbles, the other generating gas and inflating the intestines. Add thereto, failure of the circulation in the paralysed limbs, whereby they become cold, livid, and ill-nourished. These are, indeed, symptoms of death (by paralysis) beginning in the spinal cord.

Excepting this mode of dying, both that by coma and by cardiac syncope, or asthenia, respectively derive their fatal import from the more or less urgent asphyxia consequent on the failure of one or other of the conditions essential to respiration. By cardiac syncope, the heart suddenly fails to propel the blood through the lungs, and an expiratory gasp succeeds; in asthenia, this requirement gradually fails. By coma, the nervous requirements of the respiratory act are wanting.

There is yet another mode of dying by *asphyxia*, properly so called:—when any mechanical obstacle prevents the free admission of air from without, through the larynx and air-tubes, to the air-cells; or when, if air be thus freely admitted, it is impure, and therefore chemically unfitted to aerate the blood. In either case asphyxia ensues. But its symptoms vary with circumstances, and chiefly according to the suddenness and urgency of this state.

Sudden and *complete* asphyxia, as by hanging, immediately induces violent, but voluntary, efforts to regain breath. If the struggle proves unsuccessful, what then happens? During this short and sharp, but ineffectual struggle for breath, the pulmonary capillaries refuse to transmit venous blood. It therefore accumulates in the right half of the heart, and in the systemic veins; hence the surface of the body, particularly of the face and neck, having first exhibited a red hue, has now become livid, and the veins turgid. The eyes are bloodshot and lustrous,

and the tongue protrudes. The natural temperature of the body begins to decline; the surface becomes cold and clammy. More perilous events press on. The brain, ever foremost in its demand for arterial blood, fails first; subsequently the heart loses its contractile power. It flaps and flutters more and yet more feebly and imperceptibly.

The cessation of respiration, insensibility, and the stagnant circulation, proclaim that life is virtually extinct. Nevertheless, the presence of black blood in the veins of the cerebro-spinal axis excites involuntary contortions, and during this apparent agony the semen is ejected. But involuntary excito-motory power declining, the sphincter muscles relax, whereby the contents of the bladder and rectum escape. One or two slow, writhing movements, and the body hangs lax and lifeless, beyond the possibility of revival.

Such are the chief symptoms that accompany and indicate sudden and complete asphyxia. It is otherwise with asphyxia *slowly* induced, as by pneumonia. The demand for air not being refused abruptly, time is allowed for the system to adapt itself by a compromise to the deficient supply. The ordinary symptoms of asphyxia are therefore less marked. There is not the sudden fight for breath; for respiration continues, although laboriously and with a sense of oppression. The *semi*-venous blood is permitted to pass through the pulmonary capillaries, whence it circulates through the body. Consequently the systemic veins are not so conspicuously bloated, yet the face has a dusky hue. Stupor and delirium, rather than insensibility, ensue; and the heart's action is enfeebled more by the mechanical impediment of pulmonary congestion, than by a deficient supply of arterial blood to its muscular substance. If, indeed, the patient outlives his pneumonic attack, there is hope that the consolidated lung or lungs may permit sufficient aeration of the blood to sustain life in a quiescent and inactive state during convalescence. The system has gradually become accommodated to a reduced respiration—asphyxia to a degree which, if suddenly induced, would have been fatal. The respi-

ratory condition is that of a reptile, yet without any urgent symptoms of asphyxia.

This accommodation is well shown in the course of many chronic lung-diseases. In advancing phthisis, for example, asphyxia slowly progresses without urgent symptoms, and respiratory accommodation is granted daily by almost imperceptible degrees of toleration.

Death beginning in the blood, or by *necræmia*, is naturally associated with that by asphyxia. The symptoms, however, are different and sufficiently characteristic. They imply the fatal failure of all the vital powers. The cerebral functions are oppressed, and, as it were, smothered. Involuntary excito-motory power is eventually overcome. Muscular strength sinks into helpless weakness. The heart loses its contractile power, and thus the force of the circulation declines, the pulse becoming very rapid, although ineffectual. Livid congestions and bloody extravasations ensue. The power of excretion is exhausted, so that excrementitious matters naturally eliminated, by the skin and kidneys more especially, are now retained in the blood.

The symptoms of necræmia are, in fact, those which denote the fatal termination of blood-disease induced by the *retention* of *excrementitious* matters; but they are most marked in the course of infectious diseases, and the blood-poisoning induced by contagion.

I need not here repeat all that was said at length in a former chapter (x.), respecting the fatal phenomena of each blood-disease.

The general symptoms of this mode of dying are thus enumerated by Dr. C. J. B. Williams:—"They are usually called typhoid, putrid, or malignant. For example, a congested appearance of the whole surface—the colour being dusky or livid, and extending to the conjunctivæ, tongue, and fauces; various slight exanthematous or papular patches on the skin, often with petechiæ; more extensive hæmorrhages in form of ecchymoses, or oozing of thin bloody fluid from the gums, nostrils, and sometimes from other passages; extreme prostration of strength, with an obtuse state of all the senses and mental faculties, occasionally combined

with delirium and twitchings of the limbs; half-closed eyes and dilated pupils; a very quick, weak, and soft pulse; frequent and unequal respiration; absence of appetite; intense thirst; a dry, brown tongue, with dark sordes on the lips and teeth; a progressive fall of temperature, from the first febrile elevation; cold, clammy, and fœtid perspiration; hiccup; subsultus tendinum, scanty and offensive urine; involuntary discharges."*

Starvation, or Inanition—Acute Atrophy, as I have termed it—is another mode of dying related to the last in this way:—Both have reference to the blood as the source of all their symptoms; but in necræmia the blood is poisoned by the addition of some noxious matter; whereas in starvation it is deprived of its *healthy* constituents. The symptoms emanating from, and denoting this deprivation, are briefly these, and occur in order as follows:—

Gnawing pain is experienced in the epigastrium; relieved, however, by pressure. In a day or two it passes off, being followed by an empty sinking feeling in the same part. Unquenchable thirst succeeds, and continues as the most tormenting symptom. The eyes look wild and lustrous, the facial expression anxious and pallid. General emaciation begins to be perceptible. A remarkable fœtor is exhaled from the skin, which exudes also a brownish and faint-smelling secretion. The muscular strength now fails rapidly, and the individual, if he move at all, prowls about with a tottering gait, and speaks, if he speak at all, with a small and feeble voice. Generally an overclouding torpor prostrates the emaciated victim; but frequently this apparent corpse revives towards the last in a state of wild delirium. Finally, death releases, either imperceptibly, by relapse into torpor ending in the sleep of death, or it may be life ceases suddenly in a convulsive paroxysm.

By *Chronic* atrophy—Marasmus—I mean that mode of dying in which the vital power of nutrition fails first, and the whole body wastes slowly. It occurs most commonly in advanced life,

* Principles of Medicine, 1856, p. 557.

and is indeed the usual mode of dying in old age; it might, therefore, be termed death by *senile* atrophy; but that chronic wasting may possibly occur at any period of life, owing to the premature decline of assimilation—premature old age. Death beginning in this way is always equivalent to that by age. Chronic atrophy is the appointed mode of passing through Nature to Eternity. Regarding it as that "degeneration of the body" by which life declines and ceases in the order of Nature, Mr. Paget adds—"It could not be without interest to watch the changes of the body as life naturally ebbs—changes by which all is undone that the formative force in development achieved,—by which all that was gathered from the inorganic world, impressed with life and fashioned to organic form, is restored to the masses of dead matter; to trace how life gives back to death the elements on which it had subsisted; the progress of that decay through which, as by a common path, the brutes pass to their annihilation, and man to immortality."*

Unfortunately, however, our knowledge of all these changes is still incomplete, and the pathological history of this natural decadence has yet to be written. Its *symptoms* are thus described by Dr. Day:—

"Under the influence of senile marasmus, the desire for food is almost lost; after partaking of it, a feeling of more or less weight and pain is experienced in the region of the stomach, and vomiting not unfrequently supervenes. There is seldom any unpleasant taste in the mouth, and the tongue either remains unchanged, or is of a bright red colour and dry. No hardness or swelling is perceptible in the abdominal region, nor is it tender on pressure. The evacuations from the bowels are dry, hard, and scanty, and there is frequently great constipation. The least exertion is followed by extreme depression, emaciation increases, and the pulse becomes very small and weak. At length the patient takes to his bed, from a feeling of intense debility. Then we usually

* Surgical Pathology, 1853, vol. i., p. 96.

observe, if not earlier, more or less febrile irritability towards evening. The palms of the hands and the soles of the feet burn, and the cheeks flush; the powers of life are gradually and almost imperceptibly extinguished, and at last, without a struggle, 'the dust returns to the earth as it was, and the spirit to the God who gave it.'"*

In seeking to discover the general Principles of Prognosis, we experience one difficulty at least throughout the course of our inquiry—the paucity of materials at our command, and the inadequacy therefore of the basis on which our generalizations are built. This deficiency arises from the fact that our present knowledge of the *natural* course and tendency of diseases and injuries is very limited. The 'natural history' of disease is yet a *desideratum;* and why? Because hitherto the career of disease has been rarely observed when *uninfluenced* by therapeutic interference of some kind or other. Consequently, being unable to foresee, in most instances, what might or would be the *natural* course and tendency of any given disease or injury, the Prognostic conclusions, or Principles, at which we arrive—by however extensive a survey of clinical observation—are necessarily ambiguous.

These remarks specially apply to each of the Principles I have drawn from Pathology properly so called, as representing 'functional' disturbances. Such Principles are supposed to express the Pathological conditions which suggest an unfavourable Prognosis. But our conclusions respecting their unfavourable character, whether perilous or precarious, are derived from observations of the functional manifestations of diseases and injuries under the influence of therapeutic interference, and not, as when left to themselves, according to the free and unfettered course of Nature.

Subject to corrections on account of this source of error, the Principles advanced in this chapter may, I trust, be accepted as the most general expressions of that 'determining' guidance which Pathology accords to Prognosis.

---

* Diseases of Advanced Life, 1849, ch. v.

CHAPTER XV.

THE RESTORATIVE POWER, ITS EXISTENCE, OPERATION, AND RESOURCES, MANIFESTED BY THE NATURAL COURSE AND TENDENCY OF INJURIES AND DISEASES, INDIVIDUALLY, TO, OR TOWARDS, RECOVERY.

This Principle illustrated by selections from the whole range of General Pathology. (See the Prospectus of Contents.)

Experience shows that certain conditions of disease and injury have an unfavourable course and tendency, and suggest, therefore, an equally unfavourable Prognosis; but that such conditions are *exceptional.*

Experience shows also that, apart from these adverse conditions, the *natural* course and tendency of diseases and injuries is to, or towards, a favourable issue; consequently, that our Prognosis may assume an equally propitious and gratifying character.

This revival and restoration from morbid conditions indicates the existence and operation of an innate Restorative Power—a property of the living body, which being adequate to the repair of injury and recovery from disease, ofttimes in spite of the most aggravating interference therewith, has commanded the tacit acknowledgment of all practitioners, however prejudiced, by misdirected education or otherwise, in every age of Medical and Surgical Practice.

Our Natures are the Physicians of our diseases, was the cardinal maxim of the "Father of Physic;" and from the period of Æsculapius down to the present time, this maxim has always obtained, in some shape or other. The "archæus" of Van Helmont, the "anima" of Stahl, the "vis medicatrix naturæ," and the "expected cure" of the French School, severally became

sectarian expressions of this conviction. Unhappily, however, many circumstances are opposed to its *practical* recognition.

Whether from any self-interest in the administration of medicines and in recourse to surgical operations, or, possibly, from the desire to possess the power of healing, or from the natural and more laudable promptings of humanity to cure disease or relieve pain—whether from one or other of such motives, we cannot say—but the practitioner is too often induced to interfere unnecessarily or prematurely; and thus he loses the opportunity of observing the natural course and tendency of disease and injury, and of witnessing the curative power of Nature.

"It is," says John Bell, "a propensity so natural with men of our Profession to rely on the resources of Art, and to mistrust Nature, that we seem almost to have forgotten how much unassisted Nature can do. It is also an error, but too common, to prize highly the learning and maxims which we have acquired with difficulty; to carry the speculations of the closet into real practice: to retain the prepossessions imbibed during our younger years after we are called to those manly duties which require the exercise of sober judgment and plain good sense. The first great difficulty we feel in acting with a just reliance on our own good sense is that of believing that anything usually and openly practised can be entirely wrong; and this, again, proceeds from our not knowing from what weak men, or from what trivial occasions, many established opinions and practices have arisen. This once explained, the younger part of the Profession would find nothing imposing in antiquity or authority, and would trust to the dictates of that ordinary experience and plain sense which is above all learning. Let us turn, then, to the works of those amongst the moderns who have studied the ancients, prized their learning, and adopted their maxims, and whose artificial practice has corrupted ours; surely it becomes us, if not to reform our opinions, at least to inquire into the truth of them, and be able to give a reason for the faith that is in us."*

* Principles of Surgery, ed. by C. Bell, 1826, vol. ii., p. 240.

Again, in the expressive language of a recent writer on "Self-limited Diseases,"* "it is difficult to view the operations of Nature divested of the interferences of Art, so much do our habits and partialities incline us to neglect the former, and to exaggerate the importance of the latter. The mass of medical testimony is always on the side of Art. Medical books are prompt to point out the cure of disease. Medical journals are filled with the crude productions of aspirants to the cure of disease. Medical Schools find it incumbent on them to teach the cure of disease. The young student goes forth into the world believing that if he does not cure disease, it is his own fault. Yet, when a score or two of years have passed over his head, he will come at length to the conviction that some diseases are controlled by Nature alone. He will often pause at the end of a long and anxious attendance, and ask himself how far the result of the case is different from what it would have been under less officious treatment than that which he has pursued; how many, in the accumulated array of remedies which have supplanted each other in the patient's chamber, have actually been instrumental in doing him any good? He will also ask himself whether, in the course of his life, he has not had occasion to change his opinion, perhaps more than once, in regard to the management of the disease in question, and whether he does not even now feel the want of additional light."

Retarded by the obstacles adverted to, and by the fetters of authority—borne on, nevertheless, by the current of accumulated experience—clinical observers, for the most part, at length professed reliance on the curative power of Nature. The most recent and unqualified acknowledgment was regarded by its author as "the legacy" he bequeathed to his younger medical brethren. I allude to Sir John Forbes's work, entitled, "Nature and Art in the Cure of Disease."

Why, then, should I advocate the existence and operation of the Restorative Power of Nature in the cure of Disease? For two

---

* Nature in Disease, 1859, p. 39, Jacob Bigelow, M.D.

reasons. Firstly, because, acknowledged by all theoretically, *practically* this Power is still by far too little consulted, if not overlooked or opposed. And, secondly, the Power of Nature to cure diseases and injuries has not hitherto been made the subject of *systematic* clinical observation, for comparatively little is known of the *natural* course and tendency—the natural history of diseases and injuries.

I therefore purpose now bringing together such a body of evidence as cannot fail to establish, not only the existence and operation of the Restorative Power, but to identify it, also, as the immediate foundation of the leading Principle of Therapeutics.

In the first place, the *nature* of this Power should be clearly defined. The power by which restoration is effected cannot be identical with that by which structures and their functions are *maintained only* in the healthy state, and disease prevented.

The Restorative Power is manifested in all those nutritive processes and functional actions whereby structures and their functions, when disorganized and disturbed, are *recovered* to, or towards, a healthy state, and readjusted. In respect to the recovery of parts injured, it may be termed the Reparative Power.

As regards this latter, Mr. Paget affirms that it is identical with that power by which structures are maintained, and in the particular state of integrity proper to the time of life. "Thus, when in an adult animal a part is reproduced after injury or removal, it is made in conformity not with that condition which was proper to it when it was first formed, or in its infantile life, but with that which is proper according to the time of life in which it is reproduced; proper, because like that which the similar part had, at the same time of life, in members of former generations. In the reproduction of the foot or the tail of the lizard, they grow, as it were, at once into the full dimensions proper to the part according to the age of the individual. Spallanzani expressly mentions this: that when a leg is cut from a full-grown salamander, the new leg and foot are developed, as far as form and structure are concerned, just as those of the larva were; but as to size, they from the begin-

ning grow and are developed to the proper dimensions of the adult. The power, therefore, by which this reproduction is accomplished would seem to be, not the mere revival of one which, after perfecting the body, had lapsed into a dormant state, but the selfsame power which, before the removal of the limb, was occupied in its maintenance by the continual mutation of its particles, and is now engaged with more energy in the reconstruction of the whole.*

Granting, for a moment, that these two powers are identical, yet, according to the above statement on which this conclusion is grounded, it should be observed that the dimensions only of the part destroyed were restored, not its former structure, which retained that of the embryonic state. If, therefore, the reparative power be identical in its nature with that by which structural integrity is maintained, it certainly falls short of its *developmental* productiveness. Growth or size *only* is reproduced, but not the former mature condition of development.

The difference between 'simple maintenance' and 'restoration,'—in the sense of *reparation,* is most clearly seen when structures in the state of health are suddenly damaged and their functions abruptly impaired.

A man in the prime of life and of mature health sustains a compound fracture of the thigh-bone. There is considerable contusion also of the surrounding muscles, but the main artery and nerves have escaped laceration. With the shock of injury the pulse is immediately subdued, and the skin speedily becomes blanched and cold as collapse ensues. In a few hours, however, the heart has regained its wonted vigour, and the pulse is now full and bounding: this 'reaction' at length declines, and the *balance* of the circulation is again restored.

The fracture having been reduced, and the wound closed with a simple pledget of wet lint, we now daily perceive the restorative power in operation to unite the broken bone, and this process of

---

* Surgical Pathology, 1853, vol. i., p. 151–2.

reparation may possibly be conducted to complete recovery without any interruption.

In such a case the restorative power is operating under conditions most nearly resembling those of health; yet surely the reproduction of new bone, and this accompanied by disturbance in some degree of the circulation and other functions, is a *process unlike* the molecular renewal of bone wasted in the ordinary course of nutrition, and renewed by a placid circulation of the blood.

The one is simply the maintenance by healthy nutrition of healthy structure; the other, the restoration of that which was lost.

Nor can I perceive the *necessary* identity of the reparative and 'primary formative' powers. Original development and growth from the embryonic condition is one process, reproduction another; and their structural results are different.

"There is," writes Mr. Paget, "in every considerable process of repair, a remaking of a part; and the new materials assume the specific form and composition of the part they replace, through the operation of no other or otherwise directed force than that through which that part was first made. For in all grave injuries and diseases the parts that might serve as models for the repairing materials to be assimilated to, or as tissue-germs to develope new structures, are lost or spoiled; yet the effects of injury or disease are recovered from, and the right specific form and composition are regained."*

But here also the *processes* by which repair is effected differ remarkably from those of original development and growth. The process by which a fractured bone is united differs from that by which the bone itself was first formed. The degree of structural development attained is also different and inferior in all cases of reproduction. New bone or callus, for example, is less perfect bone than the original portion destroyed by fracture.

Moreover, assuming that parts injured are "lost or spoiled,"

* Op. cit., vol. i., p. 151.

and incapable therefore of assimilating, or of furnishing the germs of new tissues, this at least is an additional fact adverse to the identity of the reparative power with that by which nutritive maintenance is accomplished. The power by which reparation is effected cannot reside in parts that are lost or spoiled; yet these very parts previously maintained themselves by their inherent power of self-assimilation, and are now possibly repaired.

Passing on from structural reparation, and extending our view to all those other processes and functional actions which indicate the existence of a general power of restoration prevalent throughout the living body, what conclusion as to its nature does this wider survey of Pathology suggest? The distinctive character of the Restorative Power becomes even more apparent; and such will be the impression unavoidably conveyed by the many and varied instances of its operation adduced in the course of this chapter.

The Restorative Power—including its reparative phase—with all the *special* phenomena of its operation, could never have been *predicated* by the most minute and extensive knowledge of Physiology. The idea of 'reparation,' for example, is not involved in that of 'nutritive maintenance,' nor in that of 'growth' or increase of quantity, nor in that of 'development' or the 'original formation' of structure. The production of structure by development and growth combined, and its subsequent maintenance, would not and could not ever lead us to infer the existence of the power of reproduction, much less could such knowledge enable us to foresee the processes by which it operates. The existence, operation, and resources of the Restorative Power can therefore be discovered and appreciated only by vigilantly and repeatedly watching its curative agency—as displayed naturally in the course and tendency of injuries and diseases.

Less conclusive, because less demonstrative, are certain *arguments* of an *inferential* character.

This species of indirect evidence is abundantly supplied by the learned author of the work already mentioned. Sir John Forbes takes his stand on arguments such as these:—

Firstly. The pathology of animals.—Wounds of wild animals, even of the most desperate kinds, are often repaired by natural processes, as proved by *post-mortem* examination of the bodies of these animals when subsequently killed or found dead in their native haunts. Wild animals affected with epidemic disease of fatal character, more frequently recover than die; and all those restored to health must have been restored by the curative power of Nature alone. This also is true of domestic animals—horses, cattle, sheep—when no treatment was employed.

Secondly. The medical history of savage or uncivilized nations —as reported by travellers and residents—shows, that of the diseases occurring among such people, the greater portion which have a favourable issue (and the number is great) must owe this result entirely to the inherent power of the body.

In other cases, where some kind of treatment was employed, the recovery could not be ascribed thereto. Superstitious charms, inert herbs, invocations, sacrifices to various deities, witchcraft, &c., could have had no avail. Nature must have been the Physician.

Thirdly. The order of Physicians did not exist among many civilized nations through a considerable period of their history. Among the Israelites, the Greeks, and Romans, for a long time, the treatment of diseases, being left to the priests, was, generally speaking, of such a nature as could hardly have had any beneficial influence.

Even in the school of Hippocrates much of the treatment was regiminal, and auxiliary therefore only to the efforts of Nature.

Yet there is no ground for supposing that the mortality from ordinary or extraordinary diseases was much greater in ancient times than now.

Fourthly. Isolated individuals, or isolated bodies of men, have been attacked with disease under circumstances in which no medical aid or medical appliances were procurable, and, nevertheless, have recovered; for example, shipwrecked mariners thrown upon a savage coast. In commercial vessels also, without surgeons, re-

covery from sickness proceeds under Nature's care, or in spite of the heroic treatment employed by the captain.

Fifthly. Inert methods of treatment are followed by recovery.

Sixthly. Quack methods of treatment are on the safe side—inert. Homœopathy is of this kind, and yet frequently successful. Nature alone is here the physician. The so-called "system" itself is virtually—*do nothing* medicinally or "expectant," although the aid of both regimen and faith is surreptitiously invoked.

Seventhly. The most opposite methods of treatment may be equally successful, and the mortality also equal. Witness the results of the same epidemic disease, and during the same epidemic. For example, the results of very different modes of treating typhus, small-pox, &c.

In all such cases the curative power of Nature can alone account for equal results under dissimilar modes of treatment.

Eighthly. The autocracy of Nature in the cure of disease and injury is supported by the concurrent testimony of a large section of the medical profession, including many of its senior and most distinguished members.

Sir John Forbes thus concludes his arguments:—"that of all diseases which are curable and cured, the vast majority are cured by Nature, independently of Art; and of the number of diseases that, according to our present mode of viewing things, may be fairly said to be curable by Art, the far larger proportion may be justly set down as cured by Nature and Art conjointly. The number of diseases cured entirely by Art (of course I omit in all these statements *surgical* art), and in spite of Nature,—in other words, the number of cases that recover and would have died had Art not interfered, is extremely small."

This species of evidence is advantageously followed by that of a less general and more demonstrative character.

The actual course and tendency of injuries and diseases prove the existence of the Restorative Power, by illustrating its operation in various *nutritive* processes and *functional* actions, conducive to the repair of structure and the readjustment of functions.

I proceed, therefore, to analyse the general clinical history of natural recovery from many and different kinds of injury and disease, to, or towards, the healthy condition, structurally and functionally.

First in order may be taken the constructive power of the *materials* supplied for the repair of *injuries*.

Coagulable lymph and, possibly, blood are the materials employed, and their constructive power is exhibited by the organization they both undergo spontaneously.

John Hunter, so far as I know, first advocated the possibility of blood undergoing this change; and it was with him the mode of 'union by the first intention.'* Subsequently, the organizability of blood was disputed by Mr. Travers† and other observers. Now, however, it is amply confirmed by the microscopic observations of Zwicky,‡ Paget,§ and Dr. W. T. Gairdner,‖ not to mention others.

I allude to observations such as these:—the organization of blood effused in serous sacs, particularly in the arachnoid; of clots in veins being converted into fibrous cords, or having evinced less constructive power, degenerating into phlebolithes; clots forming distinct tumours in the heart and arteries; and the clot above a ligature on an artery becoming part of the fibrous cord which constitutes the impervious portion of artery.

Mr. Paget thus estimates the function of blood in the repair of injuries:—

1. It is neither necessary nor advantageous to any mode of healing.

2. A large clot, at all exposed to the air, irritates and is ejected.

3. In more favourable conditions, the effused blood becomes enclosed in the accumulating reparative material; and while this is organizing, the blood is absorbed.

---

* Blood, Inflammation, and Gunshot Wounds, p. 193-4.
† Physiology of Inflammation, and the Healing Process, 1844, p. 162.
‡ Die Metamorphose des Thrombus, 1845.
§ Surgical Pathology, vol. i., 1853, p. 174.
‖ Edinburgh Monthly Journal, 1851, Oct., p. 392.

Lastly, it is probable that the blood may be organized and form part of the reparative material; but even in this case it probably retards the healing of the injury.

Mr. Paget then traces the process of organization which coagulable lymph undergoes. Fibro-cellular or connective tissue is formed, but in either of two different ways, or by both processes of development simultaneously; and the particular mode of lymph-development is determined chiefly by the circumstance of exposure or not to the air. Lymph effused for the repair of open wounds generally developes itself into fibro-cellular tissue, through nucleated cells, which elongate into filaments; while that effused for the repair of subcutaneous wounds as generally developes itself into this tissue, through the medium of nucleated blastema—by the nuclei developing themselves into fibres (Henle), or by the blastema itself undergoing fibrous transformation (Paget).

The same state of organization—fibrous tissue—is thus attained, by either of these different processes of self-development, or, as I have said, by the association of both in some, probably many, instances of reparation.

Coagulable lymph possessing an inherent self-constructive power, I proceed to show *how* this power is exercised in the repair of injuries.

The healing of *Wounds* may be effected in one or other of four different ways, first clearly distinguished by Macartney,* and subject to certain modifications;—the distinctions he drew are still tenable.

First. Immediate union, without any intervening substance, such as blood or lymph. (Union by the first intention, and through the medium of blood—Hunter).

Secondly. Union by the medium of coagulable lymph, or a clot of blood—mediate by lymph or blood. (Union by adhesion, or adhesive inflammation—Hunter. Union by first intention, as now commonly understood. Primary adhesion—Paget).

---

* Inflammation, 1838, p. 48.

Thirdly. Reparation by suppurative granulations.

Fourthly. Healing under a scab. (The modelling process of Macartney?)

Each of these processes of healing claims some further notice, in reference to the power of reparation indicated thereby.

'Immediate union' is effected only under certain circumstances. Incised wounds will thus unite when the cut surfaces are immediately replaced in close contact, so that no substance of any kind intervenes. The blood itself is pressed out of the wound, the divided blood-vessels and nerves are brought into perfect contact, and reunion ensues by the opposed surfaces simply growing together. This process of repair is very speedy. It will take place in two or three days; possibly in almost as few hours. No intermediate substance exists in a wound thus healed; consequently no cicatrix or mark remains. Nature undertakes and may accomplish the immediate union of any incised wound, irrespective of its extent, provided only the conditions mentioned are fulfilled.

'Mediate union' by coagulable lymph is effected through the formation of nucleated cells, which develope themselves into fibres, this fibrous or fibro-cellular texture constituting the medium of union. Adhesion, therefore, is said to take place in such case; for the opposed surfaces of the wound are rejoined by an intermediate substance. But, as Mr. Paget remarks of this mode of union, it is less desirable than the immediate; the formation of lymph or exudation cells being a process so indefinitely separated from that of pus-cells, that union thereby is much more likely to pass into suppuration than any process in which no lymph is formed; then again, it is probably not so speedy in most cases; and finally, if accomplished, it is not so close, a scar therefore always remaining by the organization of the new intervening substance.

'Reparation by suppurative granulations' is that process by which all other than incised wounds are healed.

Hemorrhage ceases, and the local circulation is suspended. A thin coagulable lymph oozes from the surface. This organizes itself

into a fibrinous film, in which is imbedded an abundance of white corpuscles of the blood. But the influence of 'texture' in regulating the effusion is clearly seen; for scarcely any, if any, lymph is yielded by fat or bone. Otherwise, the whole surface of the wound becomes glazed over. Then follows a period of inaction, lasting from one day to ten, or more; and varying with each particular texture in the wound. Concurrently, however, the same fluid continues to ooze, and the film to slowly increase in thickness, so that at length the surface is covered with a yellowish-white layer of buffy coat. This period of repose is aptly named by Mr. Paget "the brooding time for either good or evil." The healing may proceed underneath the film of lymph, and be soon completed; or granulations begin to form when the brooding time is about to terminate.

A distinct afflux of blood more than restores the circulation around the wound. Inflammation has supervened, and it announces the commencement of reparation by suppurative granulations. Then, the organizable material or lymph is effused, out of which granulations are formed. This lymph mingles with or displaces that which hitherto glazed the wound. It undergoes the process of organization already described—namely, self-development into fibrous or fibro-cellular tissue. The deepest cells are most advanced; they elongate themselves nearly into filaments: the superficial ones remain in a rudimental state, or along the margin of the granulating surface, and acquire the character of epithelial cells. Blood-vessels shoot up from below; but I shall recur to them presently. Nerves and lymphatics do not apparently enter the substance of granulations.

Such being their minute structure, they arise, if healthy, in the form of small conical papillæ, having a glistening red colour, when free of pus.

But the formation and growth of granulations are determined by the particular textures constituting the surface of the wound. Skin and muscle are soon overspread; fat and bone are comparatively barren, and, for a longer time, remain bare.

The various appearances which granulations assume by disease are contingencies unconnected with the process of formation and the reparative power evinced.

This process of repair is effected by *suppurative* granulations, and the relation of pus thereto would appear to be twofold. The cells of pus are either degenerate, or ill-developed, granulation-cells. Regarded as these cells in a degenerate state, pus represents the superficial portion of organized granulation-matter, which having already lived its time, passes away, just as epithelial cells disintegrate, and detach themselves from the skin or mucous membrane. Regarded as ill-developed granulation-cells, pus represents only the superfluous portion of organized granulation-matter, which has never reached maturity. This latter is, I suspect, the chief relation of pus to the granulating process. Consequently, we are not surprised to observe that pus-cell formation ceases when the granulations gain the level of the skin. The chasm being now filled up, no more organizable material is needed.

Possibly, in the course of reparation, granulating surfaces meet together; when, if the granulations be healthy, and are retained in easy contact, they coalesce. This disposition of granulations to unite was noticed by Hunter.* It is accomplished by the same process of organization as that of primary adhesion: the development of fibres from nucleated cells, and the interchange of blood-vessels through this medium of communication. For the sake of nominal distinction only, Mr. Paget proposes to designate the union of granulations 'secondary adhesion.'

Whether by the development and growth of granulations from the bottom of a wound, or occasionally by adhesion in the course of their progress, the breach is repaired. But ere the skin-level is thus attained, another supplemental force comes into operation. The granulations contract, slowly yet surely, so as to effectually bring down the marginal skin to their own level, and diminish the area of the open sore. This contractile force continuing

* Works, vol. iii., p. 493.

day by day, lessens the extent of new skin required, while the granulations apparently rise to the surface. Partly therefore by their actual growth, and partly by the constant exercise of their contractile force, granulations and skin at length become even.

Then *cicatrization* commences, its purpose being to cover the granulations with skin.

The marginal skin acquires a paler tint, and a bluish-white line appears in advance, and still advancing, over the granulations, around the circumference of the sore. It is new skin, covered with new cuticle. In some cases, the marginal granulations are not the seat of cicatrization. The new skin forms in detached spots on the surface of the sore, which is thus beset with little islands of skin. Eventually, in either or both ways, the whole surface presents one cicatrix or scar; the process of cicatrization is completed.

When newly-formed, healthy cicatrix-tissue is thin, has a red, stretched, shining aspect, and is not so supple and elastic as true skin, it is also depressed, sometimes elevated, but always less moveable. The scar itself contracts for a long while and with considerable force, especially after burns, and acquires a pearly-white colour.

These physical characters change somewhat with the lapse of time; for cicatrix-tissue gradually approximates to the structure and appearance of healthy skin; while the new subcutaneous cellular texture—by which the scar was at first bound down—relaxes its hold. The cicatrix more and more resembles true skin—is supple, elastic, and moveable as a part of the integument. Nature has healed and almost effaced the wound.

'Healing under a scab' ranks higher, in respect of its result, than that by suppurative granulations. Open wounds, and superficial burns, may heal in either way. But the cicatrix formed under a scab more nearly resembles the natural textures, and being also less contractile, is less disfiguring. Yet the process of healing in this way is more liable to miscarry. Inflammation is apt to supervene, and discharge accumulating under the scab, the healing

process is again and again delayed, or recommenced. No such impediment interrupts the progress of suppurative granulations,—or the course of an *open* sore.

The scab itself is formed of dried blood, lymph, or pus; but the precise nature of the reparative process underneath is concealed from our view. So far—observes Mr. Paget—as one can discern with the naked eye, the wounded surface forms only a thin layer of cuticle on itself; no granulations, no new fibro-cellular tissue, appear to be formed; the raw surface merely skins over, and it seems to do so uniformly, not by the progressive formation of cuticle from the circumference towards the centre, as is usual in open wounds.*

Healing by the 'modelling process' is somewhat similar. It takes place under a scab or protective film. But that "natural growth" of textures proper to the part, by which it was said to be remade or remodelled, and which was thought to be the special and characteristic feature of the modelling process, is probably only the growth of granulations, *without* suppuration, because not exposed to the air.

This mode of healing, like the last, is often witnessed in the wounds of animals. Macartney first described its characters, and I state them on his authority.

"The pain arising from the injury soon ceases. No tumefaction ensues, separating the edges of the wound; and its surfaces are not only disposed to lie in contact, but even to approach each other so much, that they cannot be kept asunder by mechanic restraint; there is therefore no necessity for the effusion of lymph; and as there is no cavity to be filled up, granulations are not formed. The surfaces of the wound, although they come into contact, do not unite by vessels shooting across; they are smooth, red, and moistened with a fluid, which is probably serum, and present the appearance of one of the natural mucous surfaces of the body. If any parts have been killed by the injury, they are detached

* Lectures, vol. i., p. 227.

by simply as much interstitial absorption as may be sufficient to set them free. The wound is finally healed by the same means which determine the shape of the natural parts of the body. It gradually diminishes in extent until obliterated; or it may be cicatrized before the surfaces are abolished, after which the same process of natural growth goes on until no part of the original wound remains. The cicatrix which succeeds the cure of injury by this modelling, or growing process, is small, pliant, free from callous adhesions to the parts underneath, and morbid sensations, that so often belong to cicatrices, which have for their bases deposits of lymph, or the new-formed structures, called granulations.

"When the modelling process, or cure by natural growth, goes on perfectly, there is no inflammation in the part, and the patients are so entirely free from all uneasy sensations, that I (Macartney) have known instances of their being ignorant of the real site and extent of the injury until they had examined the part with their hand, or seen it in a looking-glass.

"It might be anticipated, that as this mode of reparation bears so strong a resemblance to the natural formation and development of parts, it is the slowest mode; but this is of little account, when compared with its great advantage in being unattended with pain, inflammation, and constitutional sympathy, and leaving behind it the best description of cicatrix. It constitutes the nearest approach, in the higher classes of animals, to the regenerative power exhibited by some of the inferior tribes."*

Hitherto I have described the various processes by which wounds heal with the view only of illustrating the *operation* of the Reparative Power. The *resources* of this power are manifested in the healing of wounds under circumstances of difficulty or complication. The healing of wounds in different *regions* of the body exhibits these resources.

Wounds of the head are prone to heal in healthy subjects,

---

* Inflammation, pp. 53–4.

although the lesions themselves be most extensive, and the mutilation most destructive of texture. One such example is narrated at p. 703. Another, and more remarkable, exemplification of healing power will now suffice.

A man was brought to the Royal Free Hospital in the year 1862, having fallen from the box-seat of a van, and pitched on the right side of his head. He was faint, but without any symptoms of concussion of the brain. The whole of the integument belonging to the right half of the skull was torn off, and hung as a flap over the face. The peduncle of this flap extended from the external angular process of the frontal bone to the nasal eminence; so that it was connected only by this limited portion of integument. The temporal muscle was detached entirely down to the zygoma, and lay in the flap, full of grit. The corresponding portion of skull was so entirely bare of periosteum, that I could plainly see the numerous foramina in the temporal bone, from which small (fibrous) processes of this membrane had been torn, when it was peeled off. Below the zygoma, the upper half of the masseter muscle was exposed.

I removed the temporal muscle, and replaced the flap of integument. It united by adhesion throughout its whole extent, excepting over the zygoma and masseter muscle. There, healthy granulations sprang up, and, by their growth, apparently incorporated the portion of temporal muscle beneath the zygoma. Not one unfavourable symptom occurred in the course of healing, excepting a heavy aching pain, now and then, over the temporal and parietal bones. The parts entirely united, and the granulations below the zygoma cicatrized soundly. The only obvious disfigurement then was the remarkably flattened appearance of the right side of the head, occasioned by the absence of the temporal muscle—a bulky one.

Four indications of strong reparative power were evinced by the progress of this interesting case—namely, union by adhesion of nearly the whole flap of integument, although ill-supplied with blood through its narrow peduncle; union of the integument and

bare bone, without any exfoliation; union of the cartilaginous portions of the ear; and lastly, the formation and growth of granulations, and the course of cicatrization over the masseter muscle, although from time to time displaced by movements of the jaw.

Passing from wounds of the head to those of the thorax, we observe in the healing of penetrating wounds equally unequivocal evidence of the curative efficacy of Nature under circumstances of difficulty and complication. A penetrating wound of the chest, involving the lung, exhibits the same strong disposition to heal. The thorax has been actually transfixed, and yet the whole track of the wound has healed kindly. One such instance occurred in the person of a sailor, whose case is recorded in the catalogue of the Museum of the Royal College of Surgeons. I had an opportunity of examining this man many years after the accident. He was then in good health, and without thoracic inconvenience of any kind. I communicated the particulars of the examination I made to the "Lancet" (about 1859), the exact date of which, however, I cannot find. Another case of thoracic transfixion by the shaft of a chaise, followed by recovery of eleven years' duration, is also to be found in the same Museum.

But the healing of a penetrating wound which involves the lung teaches more than the efficacy of the Reparative Power. We observe also how the *resources* of this power are *specially* adapted to meet and overcome adverse circumstances.

A penetrating wound of the chest, involving the lung, is in this predicament:—the lung falls collapsed after a few inspirations have inflated the pleura, and lying in this state beyond the reach of art, air and blood might continue to escape from the lung-wound into the cavity of the pleura, thus further embarrassing the respiration and encouraging hemorrhage. But with collapse of the lung there is safety. The pleural bag being fully distended with air, remains so, if only the external flesh-wound be closed, and accurately compressing the lung, as an air-tight cushion, precludes any further escape of blood or air from the rent in the lung. Collapsed and inactive, therefore, as this organ is, the pulmonary pleura is also at rest. Healing of the rent by adhesion speedily

ensues, and is facilitated by the strong disposition of the (pulmonary) membrane to throw out coagulating lymph under the slightest irritation. Thus the smallest wound of the lung having arrested the expansion of this organ, the inflated pleura serves as a *compress* to stop hemorrhage and any further escape of air; the rent in the pulmonary pleura is also at *rest*, and strongly disposed to adhere and unite by virtue of the reparative material being so readily supplied. Surely this compress almost immediately placed over a wounded part, itself beyond the reach of art, this rest of that part, and this strong disposition to heal in an emergency, are so many adequate and beneficent resources of the reparative power.

They were first clearly explained by John Bell,* who also showed how, by analogous resources, penetrating wounds of the abdomen, involving the viscera, are safely healed. That the abdominal viscera are equably and adequately supported as a whole by the action of the abdominal muscles, resisted by the diaphragm; the viscera, therefore, are severally retained in position, and move much less, relatively to each other, during life, than would appear from their looseness, as seen after removal of the abdominal walls. Consequently, when, by a penetrating wound, any portion of bowel, for example, is opened and threatens to protrude, the abdominal muscles still exercising their wonted upward pressure, tend to close the aperture (in the bowel), thus precluding the escape of fæces and blood. Even more is accomplished by this resource. For the relative position of the viscera being still nearly retained by the external pressure is a provision by which the wounded bowel remains as nearly at rest and adjusted to the external wound. Lastly, the strong disposition of the peritonæum to exude coagulating lymph comes into operation as that provision by which the two wounds are speedily united, thus closing the breach and triumphantly completing the efforts of the reparative power. Or should this most perfect intention be not fully realized, then at least the open bowel remains favourably placed by adhesive contact with the external wound, allowing free

* Principles of Surgery, ed. C. Bell, 1826, vol. i., p. 440 *et seq.*

vent to fæcal matter. To speak paradoxically, an '*artificial* anus' is made and finished by Nature *alone*.

The repair of *Fracture* displays most conspicuously the unbounded *resources* of the Reparative Power. So manifold, indeed, is its adaptation to the peculiar circumstances and exigencies of each case, that I shall select the typical process only or chiefly, whereby the fracture of a long bone is repaired.

Omitting any consideration of the damage done to the surrounding soft textures and the disposal of the extravasated blood, as not essentially connected with the question of 'fracture-union,' it is necessary to bear in mind the precise kind of injury about to be repaired.

When a long bone is broken, its medullary web—endosteum—is torn, and, in most instances, completely across; but the periosteum is rarely much damaged. "It is seldom stripped off the broken ends. Commonly it is cleanly rent across at the same level as the bone is broken, and maintains its close union, having only its fibres somewhat frayed or pulled from their natural direction. Sometimes, indeed, it remains entire, even in extensive fractures; and in this case, thickening, it contributes to the security of the repair of the injury." This integrity of the periosteum is an important fact on the authority of Mr. Paget,* to whose special observations I am also indebted for the particulars of the supervening process of repair, which is briefly as follows:—

Inflammatory lymph, in small quantity, is first exuded round about the seat of fracture, infiltrating the neighbouring textures, "so that the cellular tissue in and near the seat of injury appears more succulent than natural, being infiltrated with a serous-looking fluid, in which are cells like those of granulations or lymph."

But inflammatory lymph is apparently incompetent to unite the fracture. The swelling caused partly by this lymph and partly by the blood extravasated—which is chiefly subcutaneous—subsides gradually. No more inflammatory lymph is exuded, in most

* Surgical Pathology, 1853, vol. i., p. 241.

instances, after the second or third day. There follows a period of inactivity, of uncertain duration, but which in the adult is rarely less than one week, or more than two. Then the proper reparative material begins to flow.

In the human species, and when the fracture is neither disturbed much nor the bone diseased, this *ossifying* lymph is deposited—not, however, abundantly—between the fractured ends of bone, supposing them to be in apposition. The reparative lymph, thus placed, constitutes a thin layer, limited only to the area between the fragments, and not extending beyond in any direction otherwise than as a slight marginal fringe externally, and less so within the medullary canal. It is as if the fractured end of either portion of bone had been impressed upon the soft material interposed between them, and the two ends meeting in apposition, had squeezed out the superfluous matter around the area of compression; just as a stamp or seal, impressed on soft sealing-wax, expels a small marginal quantity from between the seal and paper; in other words, there is an interposed layer of lymph, eventually forming, when ossified, an 'intermediate' callus, but little or no "external' or 'internal' callus. The two fractured ends of bone are united by a process analogous to 'primary adhesion;' for Nature requires no more than an interposed layer of lymph, and accordingly no more is supplied than this *modicum* of organizable material. Such is the mode of union by the *normal* process of repairing fracture in the human species. And it is remarkable to observe how unexceptionally this *limited* amount of organizable material, forming only an intermediate callus, is supplied, even when the fractured ends of bone are not in apposition, but displaced so as to overlap. In such case, the callus is interposed between the *overlying portions* of compact bone. Nature still works upon the same plan, but with that degree of deviation which is absolutely necessary to firmly unite the fragments when thus displaced. The organizable material is still interposed only between the adjacent surfaces of bone; but it is interposed more abundantly wherever the strongest union is wanted;—" wherever would be the

weak part if unhealed, there is the new material placed, in quantity as well as in position just adapted to the exigencies of the case, and restoring, as much as may be, the original condition and capacities of the bone." No external or internal callus is formed.

Should, however, this process of union be disturbed by repeated movement of the fragments, or should it be perverted by disease of the bone itself; then, indeed, the reparative process resembles that by which bones of the lower animals are commonly united. It was *this* process which was traced experimentally by Dupuytren, and by other observers of experiments on animals, and which has been erroneously imported into surgical works as if applicable to the human species, and as if the pathology of man in this and other instances of supposed resemblance were the counterpart of animal pathology. But it is only when coerced by similar circumstances the pathological condition of the human body temporarily resembles that of an animal—itself not remote in the scale of organization; that then, for a time, disease or injury assumes a somewhat similar, or analogous, course and tendency.

Thus, in the event of a fractured limb being subjected to *movements repeatedly,* as the limb of a dog might be without the protection of splints; Nature, ever provident, soon forms a splint in the shape of an external callus, or bony clasp, around the seat of fracture; and, moreover, an internal callus, or splint, within the medullary canal. The same assistance is also given when the bone, being *diseased,* is indisposed to form an adequate intermediate callus. But the callus placed in either of *these* situations is only temporary or *provisional.* The lymph deposited speedily undergoes ossification, and either callus is formed some time before the intermediate or *permanent* (definitive) one begins to ossify.

"Ossification of the ensheathing (external) callus is accomplished chiefly, or solely, by outgrowth of bone from the fragments on which it is placed. Here, also, the same method of progress is observed, in that the formation of new bone extends gradually towards that part of the callus which exactly corre-

sponds with the plane of the fracture. This part of the callus is last ossified; but at length, its ossification being complete, the fragments are combined by and within a sheath, or ferrule, of new bone. The internal callus, ossifying at about the same time, consolidates the cancellous tissue of the fragments, and at a later period unites them. The walls remain still longer disunited."*

What are the *minute* structural changes whereby the reparative lymph, wherever placed—intermediately, externally, or internally—is developed into bone, or callus? A nucleated blastema is its first phase, which, in turn, developes itself into fibrous tissue, as already explained in respect of subcutaneous wounds. This fibrous, or fibro-cellular, tissue ossifies. Or, the lymph may deviate from its usual order of development, by passing at once into the structural condition of nearly perfect cartilage, and this ossifies. Possibly it assumes a structure intermediate between the fibrous and cartilaginous. "In either case, ossification may ensue when the previous tissue is yet in a rudimental state, or may be delayed until the complete fibrous or cartilaginous structure is first achieved."

"The new bone, through whichever mode it is formed, appears to acquire quickly its proper microscopic characters. Its corpuscles, or lacunæ, being first of simple round or oval shape, and then becoming jagged at their edges, subsequently acquire their canals, which appear to be gradually hollowed out in the preformed bone, as minute channels communicating with one or more of the lacunæ. The laminated canals for blood-vessels are later formed. At first, all the new bone forms a minutely cancellous structure, which is light, spongy, soft, and succulent, with a reddish juice rather than marrow, and is altogether like fœtal bone in its first construction. But this gradually assimilates itself to the structure of the bone that it repairs; its outer portion assuming a compact laminated structure, and its inner or central portion acquiring wider cancellous spaces, and a more perfect medulla. It acquires also a defined periosteum, at first firm,

* Op. cit., p. 768.

thin, and distinctly lamellar, and gradually assuming toughness and compactness."*

Resuming the visible changes seen in the repair of fractures, it is truly admirable to observe how skilfully any superfluous reparative material now organized is removed, so as to remodel or fashion off the bone again to nearly its original symmetry of form.

External and internal callus, if present, are, as I have said, provisional only. They therefore are absorbed; yet cautiously, as either can be spared.

After union with displacement of the fragments, we notice also—the removal of sharp projecting points and edges from them; the closing or covering of the exposed ends of the medullary tissue; the forming a compact external wall, and cancellous interior, for the reparative new bone; and lastly—triumphant consummation—the making of these continuous with the walls and cancellous tissue of the fragments. Thus, nearly complete restoration is accomplished. Little remains after the lapse of years to indicate the scene of former injury; as if Nature, ever seeking to preserve or restore her perfect work, were unwilling that any evidence of past imperfection should eventually remain on record.

Compound fracture unites by the same process, only modified by inflammation. Suppurative granulations are formed by that portion of the reparative material to which air has access, and these are developed into bone; but more slowly, and less certainly, than by the ossification of ordinary fibrous or cartilaginous texture.

The Reparative Power as evinced by fracture-union is adequate to meet most exigencies. This self-sufficiency is manifested whenever, in fracture, a portion of bone has been removed by accident or by art. Its reproduction, even to a considerable extent of bone, is witnessed in not a few instances.

Gulliver mentions a case in which Syme removed from the tibia of an adult male about an inch of its entire diameter, and

---

* Ibid.

this was completely reproduced. Another example, where Bushe, of New York, performed a similar operation on the femur of a young man, without subsequent shortening of the limb equal to the portion of bone removed.*

In other cases, this reproduction of bone does not supervene; but firm osseous union takes place, and within the usual period.

One such case, under my care at the Royal Free Hospital, was accompanied with other additional circumstances of interest in relation to the repair of fracture, and is recorded in the "Lancet," July 26, 1862. The particulars are briefly these:— A widow, without family, aged forty-eight, was admitted to the hospital with a compound fracture through the middle of the tibia and fibula, the result of direct violence. Two small wounds at the seat of fracture communicated with the bones. The tibia was much curved forward (as was that also of the other leg) owing to previous rachitis, so that the broken ends of this bone threatened to protrude. There was considerable contusion of the soft textures of the injured leg, which rapidly became swollen to a large size. The patient's habits were intemperate, and she was intoxicated at the time of the accident. Her stature was diminutive; and in early life, having been subject to rickets, several bones had been broken,—namely, those of the other leg, and of both arms.

The limb was reduced, and put up in the ordinary manner; but in a few days suppuration ensued at the seat of fracture, and the broken portions of the tibia protruded. The patient's general health was well sustained by food and her accustomed stimulants, with opiates nightly. Every chance was given for union, without further interference; and the progress of the case showed that this result would have been accomplished; but the arched shape of the tibia disarranged the even and quiescent apposition of the fragments, and perpetuated their protrusion.

Under these circumstances,—six weeks having elapsed without union, although the patient's general health had much im-

* Edin. Med. Journ., No. 124, p. 46.

proved,—I considered it advisable to excise the broken ends of the tibia, and sufficiently so to enable me to reduce the leg to a better shape than before the accident. Accordingly, I enlarged the wound freely to expose these portions of bone. The leg being then bent at the seat of fracture, to evert the ends of bone, they were sawn off, each to the extent of about an inch. The leg was then reduced to a far straighter shape than before this operation, and the case afterwards treated as an ordinary compound fracture. In the course of two months, firm osseous union had ensued, and the patient left the hospital in good health, with a sound limb, about two inches shorter than its fellow. A high-heeled shoe enabled her to walk with comfort and facility.

This case shows how predominant the Reparative Power may be, even when counteracted apparently by constitutional maladies of grave importance;—namely, the morbid blood-condition, and failure of nutrition, consequent on habits of intemperance; and the remnants, at least, of rachitis. On the other hand, the latter disease, when in active operation, may retard or arrest the bony union of fracture; syphilis, cancer, scrofula, scurvy, fever, erysipelas, starvation, and pregnancy, may severally have the same effect.

Subject, therefore, to so many occasions of hindrance, it becomes difficult to determine the period in which osseous union, or callus, is securely completed. It ranges probably from the twentieth to the seventieth day. In compound fracture, the formation of a secure callus may be indefinitely delayed beyond this period; and its uncertainty is made more uncertain by the possible operation of any one of the retarding causes adverted to, or of other such causes, at present unsuspected.*

---

* For further information—pathological and experimental—on the repair of Fractures, see Lebert, Sur la Formation du Cal, Physiologie Pathologique, 1845, t. ii.—Howship, Med.-Chir. Trans., vol. ix.—Liston, Edin. Med. and Surg. Journ., No. 78, p. 47.—Breschet, Recherches sur la Formation du Cal, 1819.—Stanley, Illustrations of the Effects of Disease and Injury of the Bones, 1849, p. 27.—Dupuytren, Exposé de la Doctrine de M. Dupuytren sur le Cal, par Sanson, Journ. Univ. des Sciences Médicales, t. xx.—Villermé, Journ. Univ. des

Dislocations exhibit, even more conspicuously than Fractures, the efficacy of the Reparative Power.

A *simple* dislocation reduced is rarely the scene of any permanent disorganization of the surrounding textures. The torn ligaments and tendons, if any be ruptured, as that of the subscapularis muscle, with dislocation of the humerus downwards into the axilla, are readily reunited by adhesion, provided only the lacerated parts be allowed to remain quiescent during the process of repair. Lymph is effused, which speedily undergoes development into fibrous tissue, through the medium of nucleated blastema. The textures quite regain their wonted strength; any extravasated blood and superfluous effusion are absorbed, and the swelling subsides. Thus, all vestiges of the injury are gradually effaced, and the mechanism, being adequately repaired, is at length restored to nearly its originally perfect construction. So much for reduced simple dislocation. But an unreduced one is not forsaken. The resources of the Reparative Power are now displayed, and most remarkably. A new joint is constructed.

The process by which this result is accomplished was briefly described in a former chapter. I here reintroduce it, to carry on the series of illustrations pertaining to the present subject.

If the dislocated bone happen to be lodged upon *muscle*, it gradually burrows for itself a convenient nest, the two surfaces become mutually adapted to each other, a capsular ligament is formed of condensed cellular texture, and an imperfect joint is established. Should the bone have found a resting place on *bone;*

Sciences Méd., vol. xxviii.—Cruveilhier, Anat. Pathologique, 1829–42.—Voetsch, Die Heilung der Knochenbruche, 1847.—Bishop, Lettsomian Lectures on the Physical Constitution, Diseases and Fractures of Bones, 1855.—Syme, On the Power of the Periosteum to form New Bone, Trans. Edin. Roy. Soc.—Malgaigne, Traité des Fractures et des Luxations, 1847, t. i.—Gulliver, Edin. Med. and Surg. Journ., July, 1835.—B. Cooper, Guy's Hospital Reports, part iv.—Flourens, Sur le Développement des Os et des Dents, 1842.—J. Bell, Principles of Surgery, ed. C. Bell, 1826, vol. ii.—Miescher, De Inflam. Ossium, etc., 1836.—Scarpa, De Anat. et Path. Ossium, 1827.—Meckel, J. F., Path. Anat., 1818, vol. ii., pt. ii. p. 62.—Marrigues, Sur la Confor. du Cal, 1783.—Dehtleff, Dissert. seu Ossis Calli generat. etc., 1753.—Duhamel, in Hist. et Mém. de l'Acad. des Sciences, 1739–43.

then, the one loses its periosteum, and the other its cartilage,—a receptacle is gradually excavated, suitable to the impression of the dislocated articular facet, a bony rim or lip is thrown up by the periosteum around the margin of this newly-formed articular cavity, the surrounding cellular texture acquires the character of a capsular ligament, thereby completing the provision against any displacement, and a far more perfect and secure articulation is constructed.

In either case, the muscles which act on the dislocated bone further retain it in its new position, and becoming permanently shortened, their lines of action get accommodated thereto. The natural articular cavity, from whence the bone was dislodged, loses its cartilage, and closes in. It is at length partially obliterated by a dense fibrous deposit. But very slowly indeed do these destructive changes proceed; Nature reluctantly closes the original cavity, and with it the opportunity for reduction, only when long disappointed by delay and wearied by the lapse of time.

Compound dislocation is analogous to compound fracture, and their course is analogous.

In either case, if the wound be closed immediately, the injury is at once converted into a simple one of its kind—fracture or dislocation. Healing by adhesion is then possible, and should be attempted in every instance. Failing this, repair is effected by suppurative granulations and cicatrization; a slower process, and precarious.

Reviewing the operation of the Reparative Power as exemplified by the healing of wounds, fractures, and dislocations, we observe that either of these kinds of lesion may heal without inflammation, or with it and its consequences. Incised wounds, simple fracture, and dislocation, evince the former disposition; contused and lacerated wounds, compound fracture, and dislocation have the latter character. So far, it would appear that the *kind* of injury alone determines this difference. But further observation disproves the *absolute* truth of this explanation. A simple dislocation—involving as it does a lacerated wound of the textures, ligaments, and

tendons surrounding the joints—nevertheless heals readily, without inflammation necessarily supervening.

The true explanation of the difference in question lies *partly* in another direction. By comparing the condition of simple dislocation, that of simple fracture, and that of simple or incised wound, having its surfaces in even apposition, we at once recognise their resemblance in one important circumstance;—the injured textures are not exposed to the *air;* whereas, contused and lacerated wounds, compound fracture, and dislocation are, by their nature, unless subject to surgical appliances, unavoidably exposed. We perceive, therefore, the justice and importance of the distinction originally made by Hunter between 'subcutaneous' and 'open' lesions; "injuries of the first division, in which the parts do not communicate externally, seldom inflame; while those of the second commonly both inflame and suppurate."* The access or exclusion of atmospheric air, as well as the kind of injury, *partly* determines its inflammatory or non-inflammatory character.

There is yet another generalization respecting the repair of injuries, but suggested by comparing the *development* of the reparative material—coagulable lymph, in subcutaneous and open lesions. In both kinds of lesion it is converted into the same fibro-cellular or connective tissue; but this state of organization is reached, in subcutaneous injuries, through the medium of nucleated *blastema;* in open lesions, through that of nucleated *cells.*

Subsequently, the development of new *capillary* blood-vessels is a most convincing illustration of the Reparative Power. How, then, does the new fibro-cellular tissue become vascular? How are granulations, for example, supplied with blood-vessels?

Each new capillary vessel is constructed by the outgrowth of two pouches from a parent vessel; which pouches, crammed with blood-corpuscles, extend upwards, and, at the same time, curve inwards; still converging, they never fail to meet exactly in apposition,

* On Blood, Inflammation, &c., 1794, p. 191.

without over-shooting. Having met together, these vascular segments coalesce by absorption of the partition formed of their closed ends. A complete vascular arch is thus constructed, through which tube the blood, diverging from the main current, and then rejoining it, is continuously propelled. In constructing any one such vascular arch, the pouches, from whence it springs, sometimes burst. What then?—is the arch not built eventually, or is it formed imperfectly? Assuredly not. The blood-corpuscles having escaped, are propelled through the new tissue by the force of the current of blood from the parent vessel; and this propulsive force is directed so skilfully, that the corpuscles channel for themselves a curved passage, and eventually complete the process of construction.

Thus, each new capillary vessel is constructed on an engineering principle of apparent simplicity, the repeated and unerring application of which, and upon a very minute scale, indicates the perfection of constructive skill. Contemplating its marvellous accuracy, Mr. Paget remarks:—"We admire the intellect of the engineer who, after years of laborious thought, with all the appliances of weight and measure and appropriate material, can begin at points wide apart, and force through the solid masses of the earth one tunnel, and can wall it in secure from external violence, and strong to bear some ponderous traffic; and yet he does but grossly and imperfectly imitate the Divine work of living mechanism that is hourly accomplished in the bodies of the least conspicuous objects of creation—nay, even in the healing of our casual wounds and sores."*

Equally wondrous are the processes by which blood-vessels become *obliterated*, and hemorrhage prevented, that might prove fatal.

The spontaneous cure of *Aneurism* affords an excellent illustration. The *essential* character of this process is coagulation of the blood within the aneurismal sac; for although such coagulation

---

* Op. cit., vol. i.

may be induced by Nature in four or five different ways, they alike tend to thus obliterate the aneurism.

The *principle* of cure will be readily understood. Aneurism arises whenever the force and velocity—the *impetus* of the blood's motion through any given artery—is no longer counterbalanced by the elasticity and contractile force—the resisting strength of the walls of that vessel. Coagulation of the blood within the aneurismal sac tends to restore this resisting strength, and is therefore proportionately conducive to the cure of aneurism.

The details of this process, and the external signs of its progress, are briefly these:—The blood continuing to flow through the aneurism, leaves upon the interior of the sac a thin layer of coagulum, upon which another is superimposed, and so on, forming a lamellated coagulum—the outermost portion of which, attached to the sac, acquires considerable firmness and resisting strength. It eventually becomes somewhat friable, and resembles boiled beef in colour. The portion of coagulum next in order has the consistence and appearance of damson-cheese; while the innermost portion—in contact with the flowing blood—is semi-fluid, like currant jelly. By this successive deposition of coagulum the sac is gradually filled up to the level of the artery; which still remaining pervious, the surface of coagulum exposed to the current of blood acquires a smooth and membranous appearance.

In a large artery, through which the blood flows with most force, coagulation within the *sac alone* may be the whole extent of spontaneous cure. Its external signs are:—gradual solidification of the aneurism; less and less forcible expansion; the pulsation is that of a solid tumour; and lastly, the swelling cannot be reduced by pressure applied to the artery above, or by compressing the aneurism.

In aneurism of an artery of the second or third magnitude, through which the current of blood flows less forcibly, coagulation advances from the sac into the *vessel*, which gradually becomes plugged up, above and below, with a coagulum, extending to the next important branch above and below the aneurism. This

advance of coagulation is accompanied with the cessation of pulsation. Eventually the aneurism contracts and dwindles into a comparatively small solid swelling; while the artery, to some extent, above and below it, is converted into an impervious fibrous cord. During this process of obliteration, compensation has been gradually made for the loss of the original supply of blood through the artery. The collateral branches above the unobliterated portion of artery enlarge with the additional flow of blood through them, and becoming equal to their extra duty, at length convey as much, or even more, blood than the aneurismal artery formerly supplied. The circulation is adequately restored, and nutrition efficiently sustained, by this compensatory supply of blood.

Coagulation of the blood within an aneurismal sac may be induced by Nature in four or five different ways.

Bearing in mind the cause of aneurism, as already explained, and also the physiological fact that the coagulation of blood is favoured by *rest*, we at once perceive how anything which retards the flow of blood through an aneurismal sac tends to restore its resisting strength, and is therefore proportionately conducive to the cure of aneurism. The same effect is produced by anything which *otherwise* favours coagulation of the blood within the sac.

Firstly. The flow of blood may be retarded by a less forcible propulsive power of the heart; in which case coagulation is possibly induced without any co-operative condition incidental to the aneurism itself. The sac is gradually filled up to its brim; then, possibly, the artery is plugged, and finally obliterated. This is the usual mode of spontaneous cure.

Secondly. The flow of blood through an aneurismal sac may be retarded by a piece of clot dislodged from the sac, and washed into the mouth of the distal portion of artery, or impacted within it some distance off.

Thirdly. A piece of clot may be floated down to the aneurism from one higher up, or the retarded flow of blood above may induce coagulation in the lower sac.

Fourthly. The flow of blood may be retarded by the aneurism

overlapping and compressing the portion of artery immediately above or below itself.

Fifthly. Coagulation of the blood within an aneurismal sac may be induced otherwise than by any occasion of a retarded flow of blood. Adhesive inflammation possibly, and certainly sloughing, obliterates the sac, and leaves the artery impervious.

Each of these modes of spontaneous cure is worthy of remembrance by example. Coagulation was induced in the first way—by a less forcible circulation of the blood, in the following case:—"A publican consulted Mr. Stanley in October, 1847, for a large pulsating tumour in the right breast, then *rapidly increasing*. He was ordered perfect quietude. He lay in bed for six months, and was fed only with beef-tea, milk, and light pudding. At first the swelling seemed to enlarge, but from Christmas of that year its walls gradually got harder, the pulsation diminished in force, and by March, 1848, was no longer perceptible. He then left his bed, having become by this time very pale, emaciated, and feeble. He lived very abstemiously for four years and a half, when a general election 'excited him to deviate from the tranquil and abstemious habits he practised.' He died of acute pleurisy after exposure. A large sac was found communicating with the aorta by an opening one inch by one and a half inch, just below the innominate artery, in the posterior wall of the vessel. There remained only a cavity large enough to contain a horse-chestnut; the rest had filled with firm coagulum. The tumour had been of enormous size—seven and a half inches from side to side, and four inches vertically. The disease which proved fatal was apparently quite unconnected with the aneurism."*

This case well illustrates the mode of spontaneous cure in question; for the sac was very large, and exposed to the full force of the heart's propulsive power. Similar instances are detailed in the writings of earlier authors. Desault† examined a

---

* System of Surgery, ed. by T. Holmes, 1862, vol. iii., p. 365.

† Journ. de Méd. de Paris, t. lxxi., p. 430.

popliteal aneurism which had recently undergone this spontaneous cure. He found the sac filled with layers of coagulum, and a plug also, extending to the breadth of three fingers, within the popliteal artery. Petit* examined the remnant of an aneurism of the right carotid artery, which, some years previously, had undergone the same spontaneous cure. The tumour, originally as large as an apple, was at the time of examination not larger than an olive. In the same artery, nearer the heart, was another aneurism, and in this also a similar process had commenced. The latter was nearly filled with concentric layers of coagulum which had a fleshy appearance, whilst the former was converted into a solid ligamentous knot. Baillie† met with an aneurism of the right carotid artery, completely filled with fleshy layers of coagulum. In the left carotid of the same subject was a larger aneurism, in which the deposition of coagulum was proceeding to close the passage through the sac. Mr. Ford‡ found the sac and popliteal artery, some weeks after the spontaneous cure of aneurism in the ham, filled with a firm, hard substance; and Guattani,§ at a still later period of time, describes it as having degenerated into a solid ligamentous cord.

Hodgson‖ refers to all these cases, and mentions others of like character from his own observation. *They* are peculiarly valuable as demonstrating the spontaneous curability of aneurism by coagulation within the *sac alone;* the artery remaining free to convey blood as heretofore, without supplemental collateral circulation. This more simple mode of spontaneous cure is that alone by which an aneurism of the largest arterial trunk can be closed so as not to endanger life. Were the parent artery occluded, the collateral circulation could scarcely become sufficient compensation,

---

* Acad. Roy. des Sciences de Paris, an. 1765.

† Trans. of a Society for the Improvement of Med. and Chir. Knowledge, vol. i., p. 119.

‡ London Medical Journal, vol. ix.

§ De Externis Aneurismatibus, Hist. iv., p. 16, tab. 1, fig. 2.

‖ Diseases of Arteries and Veins, and on Aneurisms, 1815.

and thus the very process of restoration would have a fatal tendency.

Several remarkable instances of spontaneous cure by closure of only the sac are recorded in Mr. Hodgson's work. Not only aneurisms of the aorta (p. 119 *et seq.*), but also those of smaller arteries, *e.g.* the brachial and anterior cerebral (p. 130 *et seq.*), were thus effectually closed, without any obliteration of the aneurismal artery. For similar instances, where the aneurisms were due to unskilful blood-letting, the reader is referred to the works of Scarpa,* Petit,† Foubert,‡ and Saviard.§

Instances of spontaneous cure accomplished in the second and third ways specified must be extremely rare. I have not hitherto met with a satisfactory case of either on record, and no such case has occurred in my own experience. I have regarded them as natural modes of cure only on the authority of standard surgical works.

It is easy to conceive *how* the impaction of a piece of clot in either the distal or cardiac side of an aneurismal sac would, by retarding the flow of blood, induce further coagulation, and eventually obliterate the sac; the usual compensation being made simultaneously by enlargement of the collateral vessels to restore an adequate circulation. The same effect might be accomplished were the flow of blood retarded by the resistance of another aneurism of the same artery higher up in the course of the arterial current.

The flow of blood through an aneurismal sac may be retarded by the aneurism overlapping and compressing the portion of artery above, or below, itself. This natural mode of cure has unquestionably occurred in a few cases. It was known to Hunter, who witnessed it in more than one instance. Hodgson gives the

---

* Treatise on Aneurism, trans. Wishart, p. 351.

† Mém. de l'Acad. Roy. des Sciences de Paris, an. 1735.

‡ Mém. de l'Acad. Roy. de Chirurgie, t. ii., p. 535.

§ Journ. des Sçavans, an. 1691.

particulars of a case* in which the femoral artery was obliterated *above* an aneurism of this vessel by compression between the sac and the femur. The aneurism remained stationary for twelve years, when it began to enlarge, and was attended with a dull pain after violent exercise. Then the tumour gradually increased; and twenty years after its commencement, had attained an immense size, but no longer possessed any of the characters of aneurism. It had a firm fleshy feel, and was without pulsation. Eventually, sloughing exposed the interior of the sac, yet without hemorrhage taking place. On *post-mortem* examination, the femoral artery above the sac was found obliterated to the extent of *three inches*.

Obliteration from pressure of the aneurism *below* itself is a similar mode of spontaneous cure occasionally discovered. It is admirably illustrated by a specimen in the museum of St. Thomas's Hospital. An aneurism of the femoral artery, just below the *profunda*, descended in the form of a large long sac for several inches below its communicating opening, and completely compressed the artery, which was full of clot even into the ham.† Hodgson‡ met with a case in which the left subclavian artery, having a small aneurism at its origin, was obliterated by the pressure of an aneurism of the aortic arch.

There are yet cases where coagulation of the blood within an aneurismal sac is induced *otherwise* than by any occasion of a retarded flow of blood. Adhesive inflammation possibly, and certainly sloughing, obliterates the sac, and leaves the artery impervious. If obliterated by sloughing, the sac discharges a quantity of coagulum, which, extending as a firm plug into the artery for some distance, closes it. No hemorrhage ensues. An aneurism of the femoral artery in one instance, and a popliteal aneurism in another, were thus cured by Nature (Hodgson).§ Guthrie‖ saw

---

* Op. cit., p. 107.

† System of Surgery, Chelius, trans. South, 1847, vol. ii., p. 206.

‡ Op. cit., p. 110. § Op. cit., pp. 103–5.

‖ Diseases and Injuries of Arteries, 1830, p. 96.

three cases of inguinal aneurism attacked with sloughing, and one of them recovered.

Such are the natural or spontaneous modes by which aneurism is cured. They each imply a course and tendency of this disease to or towards recovery by coagulation of the blood within the *sac*, either through some impediment to the flow of blood or the supervention of gangrene, which directly induces coagulation. If induced by any impediment, the *artery* may or may not be also plugged with coagulum; if induced by gangrene, it is assuredly plugged.

In the event of an aneurismal dilatation of the *whole circumference* of an artery, the cure by coagulation is just possible, only modified in a remarkable manner to meet the exigencies of the case. Nature's resources are now, as ever, ready, as occasion requires.

In one case, recorded by Sir A. Cooper, the femoral artery, from its origin to the extent of more than three inches, was dilated into a sac, which was lined throughout with very firm layers of coagulum, having a fleshy appearance. But this deposition did not completely obstruct the passage through the sac; for an irregular canal, in some places larger than the natural bore of the artery, still remained through its centre. The coagulum that formed the immediate boundary of this canal was more condensed than any other portion of the whole, and had a membranous appearance. Here, then, while coagulation had effectually *strengthened* this important artery, its *continuity* was preserved by an adequate channel through the coagulum. Only so much, and no more, coagulum had formed as was absolutely necessary to accomplish this twofold purpose.

Aneurism sometimes terminates safely in another way; but, not implying the favourable course and tendency of this disease, it must be regarded rather as an accidental mode of recovery, by an evil occurrence, than as a mode of natural cure, although no assistance is given by art. I allude to the bursting of an aneurism under a tight fascia, or other resisting structure; and compression

of the sac and artery, even to obliteration, by the extravasated blood.

One example is related by Sir A. Cooper. An aneurism of the femoral artery, just below Poupart's ligament, having burst, the thigh became enormously swollen. For three days afterwards, pulsation was perceptible over the aneurism. Then, however, it ceased, and the size of the limb began to diminish. At the end of four months, the aneurismal swelling had considerably subsided; the patient could use his limb, and in less than six months he quitted the hospital. Subsequently, he died from the rupture of an abdominal aneurism, and *post-mortem* examination showed that the femoral artery was obliterated by the pressure of the large quantity of blood effused.

Another mode of accidental recovery through a circumstance itself morbid, is by the pressure of an aneurism of a neighbouring artery, or by that of a tumour not aneurismal. Liston mentions the instance of a subclavian aneurism, which on dissection was found solidified by the compression of another aneurism springing from the innominate artery.

Allied to the cure of aneurism spontaneously, is the healing of *wounded* arteries, whereby hemorrhage is arrested and life preserved. In both processes, coagulation of the blood at the seat of injury is the starting-point of repair; but, whereas in the former instance this is the only condition necessary to complete the process, in the latter, primary adhesion or adhesive inflammation, as the case may be, invariably supervenes. These are the two stages of repair in the healing of arteries, however much its details may vary with circumstances; and *they* are—the kind of wound, incised or lacerated, either of which also may be partial or complete division of the artery or arteries concerned.

An incised and partial division only of an artery is the simplest instance; but owing to the elasticity of arterial tissue, the *direction* of such a wound is an additional circumstance which much affects its healing.

A longitudinal or oblique incision, extending partly through

an artery, more readily closes than a transverse cut, the edges of which gape and do not fall into apposition.

Supposing the incision to be one of small extent, and its direction longitudinal, or only slightly oblique; then the blood effused betwixt the vessel and its sheath coagulates and forms a compress over the aperture, extending also, more or less, above and below it. This is assisted by the compression of any coagulum which may have formed outside the sheath over the aperture in it. But the relative position of the apertures, respectively in the artery and its sheath, are altered by the effusion of blood, and the formation of a clot between the two.

This clot-compress is a temporary provision only. The wound underneath subsequently heals permanently by primary adhesion of its edges, they being in contact in this case.

Supposing, however, the more complicated case,—that the direction of a small incised wound is transverse—across the vessel. The artery contracts longitudinally, by virtue of its elasticity; and the edges of the wound no longer remaining in apposition, primary adhesion cannot ensue. The wound now heals by the effusion and organization of lymph *within* the vessel, which thus becomes impervious and obliterated. The artery, indeed, is lost in this instance; but hemorrhage of a fatal character is arrested. Nature is still victorious.

The *size* of the artery will, however, affect the issue. According to Guthrie's observations,* an artery, like the temporal, with a longitudinal slit, heals without obliteration of its canal. An artery of larger size, and similarly wounded, becomes impervious and obliterated. Then again, the ultimate success of Nature's effort is apt to be marred, even when the wound is longitudinal or oblique; for the plastic lymph is effused only by the external coat of the artery; or, at least, its internal and middle coats do not unite firmly; and this defect predisposes to aneurism.

Complete division of an artery by incision, is healed by a

* Diseases and Injuries of Arteries, 1830, p. 213.

modification of the same process. The artery is closed temporarily, by coagulation and the formation of a plug; permanently, by adhesive inflammation.

Immediately after its division, the artery *retracts*, by virtue of its elasticity, within its sheath, so that this latter projects loosely beyond the artery. Simultaneously the aperture of the artery *contracts*, even to a pin-hole opening, by virtue of its muscular contractility. The retracted portion, thus contracted, becomes conical; or—as Guthrie remarks—assumes the shape of a Florence oil-flask, or French claret-bottle. In some instances—observes Paget—the contraction is narrowly funnel-shaped, and the end of the artery may be open, while, at a little distance within, its canal is closed or much narrower. In some, the exterior layer of muscular fibres seems to contract rather more than the interior, and the end of the artery appears prominent or pouting.

This contraction of the otherwise open aperture of a divided artery may suffice to arrest the hemorrhage. Small-sized arteries, being stimulated by the injury of division, and by exposure to the air, contract sufficiently to preclude any further flow of blood. This natural mode of arresting hemorrhage is witnessed almost immediately after amputation. Comparatively few of the vessels which bled in the first instance, require ligature; they have severally receded into their sheaths, and contracted even to closure.

The retraction of a divided artery varies according to the degree of looseness with which the vessel is attached to the tissue in which it lies imbedded; according also to the capability of such tissue to retract, and the extent to which it is free to do so. Ligamentous structures retract but little themselves; arteries therefore, when divided in them, appear to retract more than those of muscle, or of an elastic structure; either of which tissues itself retracts considerably when divided.

An artery having retracted, coagulation is induced by the *filamentous* character of its sheath, which entangles the fibrin of the blood as it flows. This arrest of the fibrin, and the formation of a clot, will therefore vary with the degree of retraction of the artery; but it

varies also with the degree of obstruction offered to the escape of blood from the vessel, and from the external wound. In proportion as contraction of the aperture of the artery suffices to prevent hemorrhage, so is the formation of a clot unnecessary, and does not take place. If blood continue to escape freely from the vessel and external wound, then another resource of Nature comes into play. Cardiac syncope supervenes; the heart's action failing, hemorrhage ceases; and the blood being stagnant, coagulation is induced.

By these means the blood loiters about the aperture of the artery, and especially within the projecting cellular sheath. The clot forming there is at first pervious, and transmits a small stream of blood. Hemorrhage gradually ceases, as coagulation proceeds concentrically, and soon forms a solid plug, which completely blocks up the sheath, and closes the aperture of the artery. Still enlarging, it passes into the bore of the vessel for a short distance, and rises in the shape of a small cone, the base of which corresponds to the aperture of the artery. This conical portion of the clot is named the 'internal coagulum' (bouchon—Petit), and the portion beyond, in the sheath, the 'external coagulum' (couvercle—Petit). Its shape is less defined; for a small portion is insinuated between the sheath and artery, beyond where retraction has ceased, thereby further compressing the arterial aperture, while a still larger portion, of an irregular shape, projects beyond the aperture of the sheath. The external and internal portions of clot, taken together with the arterial aperture, are somewhat like a glass stopper placed in the neck of a decanter; to which the whole clot is compared by Professor Gross.* Nature thus stops up the mouth of a divided artery, and precludes further hemorrhage. But this is only a temporary provision, designed to meet a pressing demand.

The *permanent* closure of the divided artery is an affair of time, and is accomplished apparently by adhesive inflammation.

---

* System of Surgery, 1859.

Plastic lymph is effused, in situation corresponding to the external and internal coagula—*i. e.*, at the aperture around it, and extending into the vessel. This lymph is in contact with the vessel, and intervenes, therefore, between it and the coagula. The external portion of lymph eventually becomes a permanent substitute for the corresponding coagulum. The internal portion (of lymph) appears as a small button, effused *into* the base of the coagulum corresponding to it. Hence this portion of clot acquires a marked difference of density and colour at different parts of its extent. The base, consisting chiefly of fibrin, is firm and of a buff colour; beyond, the clot has more the characters of ordinary coagulum. The plastic base of this clot helps to permanently seal the aperture of the artery, while its prolongation within the vessel serves only as a breaker to resist the dash of the wave-currents of blood, and so far prevent hemorrhage recurring.

Respecting the closure of the *lower* end of a divided artery, Guthrie's observations* led him to believe that the distal end retracts and contracts less than the cardiac end, and not so permanently; and that the internal coagulum is, in many instances, altogether absent or very imperfectly formed.

A *lacerated* wound of an artery, or arteries, is either partial,—one or more of the three coats being torn,—or complete laceration of all three coats; the artery is completely torn through. The former injury is never attended with *immediate* hemorrhage; the latter may have this effect, but more frequently does not occasion hemorrhage, immediately or *subsequently*. There is also the more rare form of injury—that of an artery *spoilt* by a blow, but without laceration of its coats, individually or collectively, partially or completely. Yet the structural damage done is irreparable. The artery contracts at its damaged part, and permanently so. Gangrene is imminent; and a fatal case is on record.† In some such cases coagulation takes place in the contracted

---

* Op. cit. See cases, pp. 233 and 251.

† Surgical History of the British Army, vol. ii., p. 343.

portion of artery, of which a fatal instance is mentioned by Guthrie at page 22 of the work already quoted.

Partial laceration of an artery may extend through the external, and more or less of the middle coats, leaving only a thin undivided inner membrane, which, however, still continues the canal of the vessel. Hemorrhage is imminent. A fatal case is narrated by Guthrie at page 78 of the same work.

Partial laceration of an artery may extend only through the inner and middle coats, leaving the outer cellular coat untouched. Such cases also are apt to terminate fatally—possibly by gangrene, probably by secondary hemorrhage. A fatal instance which came under the care of Mr. Shaw, at the Middlesex Hospital, is related by Mr. Moore.*

Fortunately, however, in numberless instances of the latter kind of injury, the reparative power is equal to the occasion, and a happy issue may be confidently anticipated, provided only that the two inner coats of the artery are so divided as to be *cut*, rather than torn through. Such is the kind of injury inflicted by the surgical application of 'ligature,' which leaves the external coat undivided; and although the portion (of external coat) embraced by the ligature suffers continued compression, and the ligature itself is noxious, as a foreign body, yet an artery properly ligatured rarely fails to safely undergo a process of repair, terminating in the permanent occlusion and obliteration of the vessel.

The steps of this most interesting process are these. As might be expected, the ring of external cellular coat, being tightly compressed by the ligature, sloughs entirely; while the puckered portion around (this slough-ring), being less tightly compressed, ulcerates—or undergoes molecular sloughing. Thus the ligature is set free together with the included slough-ring of cellular coat. Hemorrhage would be inevitable but for the simultaneous adhesion of the two inner coats, across the area of the vessel, above and below the ligature. At these points they curl inwards, when

* System of Surgery, ed. Holmes, 1860, vol. i., p. 672.

divided, and converging, meet together. This condition was well seen in a femoral artery which I examined five days only after I had ligatured it, at the time of amputating the thigh. The *post-mortem* appearances of the stump are described in the "Lancet," December 28, 1861.

The fringed edges of the two inner divided coats are thus brought together, above and below the ligature, by a simple natural provision. Plastic lymph is then effused, and their adhesion readily ensues. A process of organization, therefore, above and below the ligature, accompanies the destruction and detachment of the ring of external coat embraced by the ligature. The artery is severed by sloughing, and either end being set free, is securely sealed, and the life-blood preserved.

Accessory, but incidental only, to the prevention of hemorrhage (when the ligature is detached) are certain changes by which either portion of artery, contiguous to the ligature, is obliterated.

The vessel ceased to convey blood when the ligature was applied; and the blood having since been stagnant, above and below, to the nearest collateral branch of artery, has been gradually coagulating in the shape of two conical clots, the bases of which accurately plug the artery, opposite the ligature, on either side. The apex of the clot on the cardiac side tails off, usually opposite the first collateral branch, through which the stream of blood, thereby diverted from its course, is carried from the main. The distal clot is less defined. At first, either clot has the ordinary colour and firmness of recently coagulated blood; then, it becomes mottled with paler spots, and its substance porous; eventually, it acquires a buff colour, firm consistence, and fibrous texture. Blood-vessels usually enter these organizing clots from the vessels formed in the lymph effused within the artery, just above and below the ligature. Hence they enter the *base* of either clot, and gradually extend towards its apex.

Finally, these organized clots are incorporated with the organized lymph adjoining the ligature; the coats of the unused

portions of artery degenerating, assume a fibrous character, and the whole is converted into a small, firm, impervious, fibrous cord, which extends to the first collateral branch above and below. Nature having safely severed the artery, and securely sealed either end, has now obliterated the useless portions.

It will be readily imagined that this process, simple as it appears, is not accomplished in a day or two. Plastic lymph is effused almost immediately within the vessel adjoining the ligature; for it is imperatively demanded to meet the loss occasioned by sloughing of the cellular coat, and consequent imminent danger of hemorrhage. Coagulation within the artery, above and below the ligature, begins after a variable period of from one to eighteen hours. Fibrous degeneration of the unused portions of artery dates probably from the organization of the two clots, and their incorporation with the organized lymph adjoining the ligature. The residual substitution of an impervious fibrous cord for the artery is an affair of time.

Such being the history of this process, an accurate comprehension of which is so important to the surgeon, there yet remain two or three points of an exceptional character, with which, nevertheless, he should be familiar.

A ligatured artery does not invariably contract as far as the first collateral branch, nor only so far as this branch, and never beyond. Exceptions to the rule were determined by Mr. Guthrie's observations. Contraction may fall short of, or pass beyond, the first collateral branch.

The falling *short* of this point—implying, I presume, a correspondingly diminished length of coagulum—shows that obliteration of the vessel is not an essential part of the curative process, but incidental only, although accessory. Organized adhesion of the coats divided by the ligature is the essential means which Nature employs to prevent hemorrhage when the ring of cellular coat is removed by sloughing. Hence, the presence of a collateral branch immediately above the ligature does not necessarily interfere with occlusion of the artery. It *may impede* the process of adhesion,

and for a time render it less safe, or *possibly preclude* it altogether: but Mr. Guthrie affirms that in so many instances arteries have healed after division close by a considerable branch, as to justify the conclusion that they are always *capable* of doing so, provided only they are sound.

The contraction of a ligatured artery *beyond* the first collateral branch depends apparently entirely on the (functional) importance which that branch then assumes. After amputation of the arm, through its middle third, the brachial artery goes on diminishing in size, as high as the large subscapular branch; the small circumflex branches—although nearer the ligature—also diminishing proportionately, owing to their then being so much less functionally important than before the operation.

The rule, therefore, would appear to be this:—that a ligatured artery (or an artery otherwise occluded) contracts as far as the first collateral branch from it, which *assumes* importance after the flow of blood is diverted from the main vessel.

An artery or arteries torn *completely* through, heals without hemorrhage, or scarcely any. This is accomplished by another natural provision, similar to that by which hemorrhage from an artery cut completely across may be spontaneously arrested. The cellular sheath, and outer cellular coat, or both together, are drawn out, from off the two inner coats, which then retract. The blood escaping, is entangled within the canal thus formed of loose cellular tissue, and coagulates there, giving a bulbous-shaped appearance to the lacerated extremity of the vessel. The clot or plug here formed of blood intermixed with cellular tissue is sufficient to prevent hemorrhage. It is larger than that external coagulum which forms over the mouth of an artery, when cut across; for, by laceration, the cellular texture is drawn out, and makes a much longer canal to receive this coagulum, than when the receiving canal is made by the retraction alone of the two inner coats. The bulb-shaped extremity, consisting of cellular texture and clot projecting beyond the inner coats, may extend to half an inch or an inch in length. It is scarcely worth consider-

ing any extension of this clot as an internal coagulum within the canal of the artery; or possibly a small portion insinuated between the retracted coats and cellular texture, thereby compressing the aperture of the canal of the artery; or lastly, any portion of clot extravasated in the cellular tissue, beyond the termination of the bulb-shaped plug. Such details are comparatively unimportant. Hemorrhage is effectually prevented, in many cases, without these accessory, but only incidental aids.

The bulb-shaped plug is the immediate provision of Nature for preventing hemorrhage. I am not aware of any sufficient observations showing how an artery thus closed is permanently secured. Probably, however, by adhesive inflammation. The efficacy of this provision—this plug, is admirably illustrated in the following case by Houston,* also adduced by Crisp, and of which the chief particulars are these:—A man, aged thirty-three, had his arm torn off at a point corresponding with the insertion of the deltoid muscle, by the revolving strap of a corn-mill. No hemorrhage occurred, except a little oozing from a few small vessels. The torn end of the brachial artery was dark coloured, smooth, and tumefied—the very extremity being largest. When pressed between the finger and thumb, it felt soft and elastic, as if tensely filled with half-fluid, half-coagulated blood, and was distended and jerked by pulsations synchronous with those of the heart; yet not a drop of blood issued from, or could be squeezed out of, its truncated extremity. The man died from the severity of the injury; and it was then proved to demonstration that Nature had closed the artery in the way already described.

Another case happened to Mr. Newbigging. The arm of a man was torn off by the roller of a mill; and the extremity of the brachial artery immediately became swollen into a bulb. Other accidents of a similar description are recorded by Larrey, La Motte, and Carmichael. Several cases might be mentioned, in which, by this provision of Nature, the artificial application of

* Dublin Journal, vol. xxiv.

ligatures was unnecessary; in which also no ligatures were used, and recovery took place, without hemorrhage, either primary or secondary. A few such instances will suffice.

Cheselden relates the case* of a miller, whose arm was torn off by a rope. The arteries and veins were drawn out of the axilla by this forcible extension; they were simply replaced, and the man perfectly recovered.

In a similar case, by Scarnell,† the entire arm was roughly torn from the body, and no ligatures were applied. The man's general health remained good; he walked at the end of a fortnight, and on the third Sunday went to church.

A man having incautiously placed his hand on one of the cylinders of a bark-crushing machine, the arm was gradually drawn in as far as the shoulder. The humerus being crushed in several places, he was liberated by dividing only the integuments. On admission into King's College Hospital, where he was placed under the care of Mr. Fergusson, about three inches of the main artery was hanging out of the wound, and the vessel pulsated violently to within one inch of its torn extremity; nevertheless, not a drop of blood was lost.

Another instance is given by Benomont.‡ A boy, about eleven years of age, had his leg torn off above the knee by the spokes of a coach-wheel; no hemorrhage ensued from the femoral artery, and no ligature was applied. Division only of the bone and soft parts was necessary, and the patient perfectly recovered.

Veins heal by processes apparently analogous to those which wounded arteries undergo.

There is, then, abundant and unequivocal evidence that the bodily organism is endowed with the (inborn) power of repairing

---

* Anatomy of the Human Body, 1778.

† Lancet, April, 1832.

‡ Mém. de l'Acad. de Chir., tom. iii.

the various kinds of injury to which it may be subjected. The existence of this Power is manifested by its operation—or the course and tendency of various kinds of injury—*e.g.*, the healing of wounds of all kinds; the repair of fractures and dislocations,—whether reduced or unreduced, and simple or compound; the development of new blood-vessels; the spontaneous or natural cure of aneurism; and lastly, the healing of wounded arteries (and veins), whether cut or torn, and partially or completely divided.

This innate Restorative Power represents a certain, but as yet unknown and inadequately appreciated, amount of reserve force, on the part of Nature; ever able and willing to be drawn upon by the practitioner, as occasion may require, in his therapeutic management of injury or disease.

Investigated by this light, the course and tendency of injuries and diseases are discovered to be, in numerous instances, processes of cure. Conditions themselves morbid, and not therefore credited with any curative power, assume this character as their course and tendency expand, under the watchful observation of the enlightened practitioner. Ever ready then is he to assist, ever unwilling to disturb, the Natural process of Restoration.

The course and tendency of injury or disease declare the *intention* or purpose of Nature, towards recovery or not. Guided by its intention, I venture to regard *inflammation* as salutary in many cases; namely, whenever an extra and pressing demand is made on the function of Nutrition—either to restore some loss of structure, or to construct some new mechanism. The part is thus either restored to its original mechanism (although not to its original perfection of structure), or it becomes adapted to the new requirements imposed on it.

John Hunter inculcated the salutary nature of inflammation *itself*. "Inflammation," said he,* "is to be considered only as a disturbed state of parts, which requires a new but salutary mode of action to restore them to that state wherein a natural mode of

* Works, 1837, ed. Palmer, vol. iii., p. 296.

action alone is necessary. From such a view of the subject, therefore, inflammation in itself is not to be considered a disease, but a salutary operation, consequent either on some violence or some disease." But the essentially morbid character of inflammation is chiefly shown by one important fact;—that the full development and appropriation of inflammatory lymph is prevented by the persistent determination of blood, and consequent engorgement of the capillary vessels. In fact, the characteristic act of inflammation, and that which ensures the most abundant supply of organizable material, disturbs the process of its organization. Inflammatory determination of blood must cease, ere the lymph effused will undergo any degree of organization approaching to that of the normal tissues.

Inflammation is, therefore, itself essentially a morbid condition; yet the *purpose* of inflammatory lymph-development, as declared by the course and tendency of this process, may be a manifestation of restorative power in many cases of inflammation. But if inflammatory lymph-development be salutary in many cases, this process is itself consequent on (the act of) inflammation. Practically speaking, therefore, the whole continued series of changes—persistent determination of (arterial) blood, followed by lymph-development—constitutes a *restorative* process, whenever its *purpose* is beneficial.

The *purpose* or intention of Nature is an acknowledged ground of distinction, and the chief or only one, between many things healthy and morbid. Sleep is a salutary condition, because obviously intended by Nature to reinvigorate and restore the functions of animal life; while *coma*, not having any such intention, is a morbid state. Then, again, the alternate systolic and diastolic action of the heart is undoubtedly salutary, by virtue of the purpose it fulfils; *palpitation*, however, being an irregular action without any physiological intention, is a morbid condition. The act of respiration is, in like manner, a healthy one, by virtue of its physiological purpose; but this act being performed more *quickly* than usual, and *irregularly*, without such purpose, as

in some kinds of Fever, becomes a morbid symptom. Touching the functions of digestion and secretion, respectively, similar remarks are applicable. The healthy character of these functions is acknowledged; their morbid character is announced by their altered intention.

So it is with nutrition. As intended to repair the waste of the body, this is a salutary function; it acquires a morbid character when no such purpose is intended.

The absence of any healthy or physiological purpose in the development and growth of Tumours is the chief evidence of their morbid character. By virtue of their minute structure, all Tumours, known as Growths, much resemble the developmental conditions of their analogous healthy tissues; but, in respect of their (functional) purpose, they fulfil no physiological intention. Conversely, inflammation, by virtue of its intention in numerous instances to accomplish some *salutary purpose*, assumes a curative character.

Inflammation is a manifestation of the Restorative Power, whenever intended to meet some extra demand on Nutrition—either to restore some lost portion of structure, or to construct some new mechanism. And, as I have added, the demand then made is pressing. Inflammation, as a restorative effort, is intended to meet some demand on nutrition beyond that of mere nutritive 'maintenance.' Accordingly, the characteristic act of inflammation is a persistent determination or persistent flow of arterial blood. The extra supply required of organizable lymph is thus conveyed to wherever the nutritive demand is made, and there effused, ready to undergo whatever degree of organization may be necessary to fulfil either of the purposes referred to. Nature thus conveys and deposits the raw organizable material. Then lymph-development begins, and advances proportionately to the *decline* of inflammatory hyperæmia. This process is analogous to that of non-inflammatory lymph-development in the process of nutrition. And the same conditions are necessary in both cases:—a due supply of appropriate blood, of nervous force, and the influence of a healthy condition of whatever texture in which, or in

connexion with which, the inflammatory lymph has been deposited.

Fibro-cellular or connective tissue is the typical organized result of this process. False membranes are formed, constituting adhesions; for example, of the pleura and those of other serous membranes.

Other and more highly-developed tissues are reproduced. Many such instances of inflammatory lymph-production are adduced by Paget.* Adipose tissue is perhaps a slight step in advance. It may be formed, if not directly from inflammatory lymph, yet in the fibro-cellular tissue of completely organized adhesions. Elastic tissue is sometimes abundantly formed in the adhesions developed from inflammatory lymph, and particularly in those of the pleura. Epithelium covers the surfaces of well-formed adhesions. Fibrous tissue is produced from the development of inflammatory lymph, interstitially deposited in any fibrous tissue; as in ligaments, capsules of joints, &c. Bone is often formed, either as a late transformation of inflammatory lymph, which had become organized into perfect fibrous tissue—*e.g.*, osseous plates in false membranes of the pleura, and in those of the pericardium, which plates are not true bone; or new bone appears in the form of ossific deposits, connected with inflamed bone or periosteum. Cartilage is possibly formed in chronic rheumatic arthritis.† This new cartilage is prone to ossify.

Blood-vessels are found in organized inflammatory lymph, but probably they are not formed in it; they project into the new tissue from the structure on, or in, which it is placed. Lymphatics were discovered by Schroeder van der Kolk‡ in false membranes. Nerve-fibres have been twice seen by Virchow§ in adhesions; one of the pleura, the other of the peritoneum between the liver and diaphragm.

---

* Op. cit., vol. i.

† Trans. Path. Soc. Lond., vol. iii., 1851, by W. Adams.

‡ Spec. Anat. Path. de Vasis novis Pseudo-membranarum, 1842.

§ Würzburg Verhandlungen, i., 144.

It yet remains for me to illustrate the restorative power of inflammation by certain remarkable exemplifications of its operation. These examples are not limited only to the results of inflammatory adhesions; they extend to the consequences of inflammation. The results of suppurative granulations and of sloughing, alike illustrate this restorative power in some cases.

The reparative power of adhesive inflammation is manifested by the healing of incised wounds in those cases where the effusion of lymph is induced by inflammatory hyperæmia. The repair of simple fracture and of simple dislocation, when reduced, is an analogous process, and commenced probably by inflammatory hyperæmia consequent on the injury. So, also, wounded arteries are permanently closed by adhesion of an inflammatory character in some cases; and the healing of ligatured arteries is certainly effected by adhesive inflammation, which seals the divided coats of the vessel above and below the line of ligature. Penetrating wounds of the thorax and abdomen, implicating the viscera, afford ample and varied evidence of the reparative power of adhesive inflammation. Wounds of the lung are healed by adhesion of the pulmonary pleura consequent on inflammation. Wounds of the abdominal viscera are healed in like manner wherever the peritoneal investment extends; and the visceral reflexion of the peritoneum is very apt to adhere to the parietal reflexion of this membrane opposite the external wound. These results were explained in describing the healing of wounds of the abdomen and thorax respectively. In certain cases the peritoneum is purposely injured by the surgeon, so as to establish adhesive inflammation. This constitutes the radical cure of hydrocele and that of hernia; the former being accomplished by a stimulating injection; the latter by long-continued pressure with a truss; or the radical cure of hernia may be accomplished on the same principle by the successful operation recently introduced by Mr. Wood. Lastly, among the most familiar examples of beneficial adhesive inflammation, I may mention the well-known fact that foreign bodies, long imbedded in the living tissues, thus become enclosed in

organized lymph, and their presence rendered comparatively harmless. An encysted foreign body is quiescent.

Reviewing these and similar results, there appears to be sufficient reason to justify Hunter's view of the use or purpose intended by adhesive inflammation. "It may be looked upon as the effect of wise counsels, the constitution being so formed as to take spontaneously all the precautions necessary for its defence; for in most cases we evidently see that adhesive inflammation answers wise purposes."*

In conformity with this design, we observe inflammatory adhesion preventing suppuration, or confining its extent—in the pleura, in the peritoneum, occasionally; in the synovial capsules, and determining the boundary of an ordinary abscess; beyond which circumscribed limit, in any case, the pus formed cannot pass. Then, again, the same process prepares the way for the discharge of pus, without infiltration of the textures during its passage or escape into any natural cavity through which it may traverse. Any collection of matter having become circumscribed—as an abscess—by the deposition of inflammatory lymph, is gradually conducted through the surrounding textures at their most yielding point; the pathway having been previously paved securely for this purpose by their adhesion. The pus is still watchfully circumscribed during its passage, until at length the contents of the abscess are discharged. An abscess is thus advantageously opened by Nature. Absorption makes the channel, and in the direction of least resistance; but adhesion makes its banks secure. With this precaution a collection of matter is safely conveyed from a great depth in the body, and by an otherwise dangerous route. Witness the evacuation of pus from the pleura (in empyema), through a large and pendulous mamma. Adhesions of other serous surfaces favour the escape of matter. When a student, I once saw an abscess of the liver which pointed and threatened to burst externally, but which eventually relieved

* Works, vol. iii., p. 399.

itself by perforating the diaphragm and right lung successively, a continuous expectoration of pus being provided for by adhesion of the pleura where perforation had taken place. Sometimes, by a similar provision, an abscess of the liver discharges itself externally, or into the stomach, the duodenum, or the colon. An abscess of the kidney will also thus communicate with the colon; and an abscess in the right iliac forsa will be emptied into the colon, cæcum, or bladder.

These are instances of a new mechanism being constructed for some new, but perchance temporarily useful purpose, which is perhaps less frequently the intention of inflammation than that of permanently restoring some lost part, and of thereby reinstating a former function.

This latter, or reparative purpose, is *further* illustrated by the healing of open wounds and ulcers, *e.g.* certain burns, through the medium of suppurative granulations; also by the healing of compound fracture and dislocation in like manner, and by the growth and contraction of suppurative granulations formed by the pyogenic membrane of an abscess after its contents have been discharged.

Lastly, should inflammation terminate by sloughing, even then this disorganization and destruction of parts may prove beneficial. Although not fulfilling any constructive or reparative purpose, sloughing may nevertheless be a manifestation of restorative power by virtue of its purpose. Nature thus operates possibly to recover the condition of health. A remarkable instance this of the proverb—that good sometimes comes out of evil. The spontaneous cure of aneurism by sloughing is one example; the unloosening and expulsion of a foreign body by sloughing of the part in which it is imbedded is another instance of the same mode of natural cure.

Keeping in view the purpose of inflammation, as shown by its career from first to last, we are led to regard this morbid condition as an expression of restorative power in many cases. Inflammatory determination of blood may prove serviceable, and its

consequences beneficial. Among the latter we find inflammatory lymph-deposit possibly undergoing development suitable to various requirements—reparative and constructive.

Other lymph-deposits, of a *non*-inflammatory origin, exhibit an opposite course and tendency. They are prone to degenerate; yet their *degeneration* may in like manner be a manifestation of restorative power,—by virtue of the *purpose* that Nature thus endeavours to accomplish. Tuberculous matter tends to degenerate and become quiescent. Grey (miliary) tubercles wither. They lose their lustrous appearance, and shrink into yet smaller fibrous masses. If deposited in the lung, the portions of pulmonary texture in which these remnants of grey tubercles are imbedded, also dry, shrivel, and acquire a dark colour from the deposition of pigment. Yellow tubercles are apt to undergo calcareous degeneration, whereby they become quiescent. This petrifaction may occur, not only in recent yellow tubercles; it may overtake those which have softened; the degeneration in either case being accompanied usually with pigmental degeneration of the surrounding pulmonary texture. Lastly, the softening of tubercle—accompanied, as this change is, with the disintegration and destruction of lung-tissue—is nevertheless not without its restorative character. The purpose of Nature is obvious. Liquefaction favours the expectoration of the tuberculous matter, and leaves the cavity, or *vomica*, formed in the lung-texture, free to contract, by the deposition of organizable inflammatory lymph around the cavity, which moreover is then likely to cicatrize.

Obliteration of a cavity, possibly a large one, in the lung, does actually occur, and far more commonly, than pathologists were formerly inclined to believe. *Phthisis pulmonalis* is *prone* to undergo this spontaneous cure. We owe its discovery—a grand one in restorative pathology—to an admirable series of observations made by Professor Bennett. During a period of five years, as Pathologist to the Royal Infirmary of Edinburgh, he performed and recorded the results of more than two thousand *post-mortem* examinations. "Gradually one great fact became im-

pressed upon his mind,—viz., that all organic diseases occasionally presented a tendency to spontaneous cure. He was repeatedly meeting with instances where, although death was occasioned by disease in one organ, there were others which presented traces of previously existing lesions which in some way had healed. In no organs were such appearances more common than in the lungs, and of no disease was evidence of a spontaneous cure more frequent than of Pulmonary Consumption."* Only one condition seems necessary—the arrest of any further deposition of tuberculous matter. But contraction and cicatrization may ensue, with or without the complete evacuation of all the tuberculous matter from its containing cavity. Organizable inflammatory lymph—instead of more tuberculous and non-organizable lymph—is deposited in the pulmonary tissue forming the walls of the cavity. Its boundary thus becomes defined, and extends no farther. Then follows contraction, and finally cicatrization; the only remnant of the original cavity being a linear cicatrix in the substance of the lung; and, as the surface of the corresponding portion of pulmonary texture is drawn in, it there presents a characteristic stellate puckering. Should, however, the cicatrix be superficial, the pleura over it becomes thickened, and adherent to the costal pleura; then, as contraction proceeds, the cicatrized portion of lung is drawn closely to the wall of the thorax, from which it cannot be detached without great violence. Any tuberculous matter which may have been retained in the cavity, during this process of healing, still exists in the cicatrix, but in a quiescent state,—it having undergone either fibrous or calcareous degeneration, and possibly have become encysted.

There is ample evidence to show that in all cases the natural tendency of phthisis is to, or towards, recovery—provided only, that the further deposition of tubercle is arrested; and there seems no reason why cavities, possibly of large size, in the lung, should not heal with the same frequency as ulcerations or abscesses in

* Pathology and Treatment of Pulmonary Consumption, 1859, Preface.

other internal organs. Professor Bennett supports this—his conclusion—by an indisputable series of cases. The following one will convince the most sceptical:—A man, aged twenty-two, laboured under all the symptoms of advanced phthisis, and his life was despaired of. A large cavity had formed. Subsequently, however, he recovered his health, and continued well. Eventually he died, at fifty years of age, from *delirium tremens*. The *post-mortem* examination discovered a cicatrix, three inches long, in the apex of the right lung, and cretaceous concretions, with puckering, at the summit of the left lung.

Laennec, Andral, Cruveilhier, Kingston, Pressat, Rogée, Bondet, and others, have also published cases where all the functional symptoms and physical signs of phthisis, even in its most advanced stage, were present, and yet the individual survived many years, ultimately dying of some other disease; and on dissection, cicatrices and concretions were found in the lungs.

Not only *Deposits*, but *Growths*, are liable, and even prone, to undergo certain morbid changes, which, by virtue of the *purpose* intended by Nature, may be regarded as manifestations of curative power. Tumours *grow*, indeed, without any seeming physiological purpose; but their pathological changes are, possibly, expressions of restorative power, in disguise.

Non-malignant, or innocent growths—*e.g.*, fatty, fibrous, cystic, and cartilaginous tumours—severally exhibit this character in some cases. It is unnecessary to specify instances, because more conclusive illustrations are afforded by the natural course and tendency of that kind of growth which is not commonly supposed to exhibit much, if any, inclination to spontaneous cure. I allude to Cancer-growth. Malignant as is its character, this species of growth has been no less indiscriminately maligned. Its career is not invariably an evil one; there is an effort made, and a striving towards a happy issue. This is evinced in various ways. Cancer-growth may cease spontaneously, and the tumour remain in a stationary condition for years or for life, and the individual die eventually from some other disease of a totally different

character. Two cases are mentioned by Walshe,* in both of which female patients having cancer, advanced to ulceration, succeeded nevertheless in concealing their disease from their husbands; and in one of these cases, the husband being a medical practitioner, such concealment was surely a special guarantee that neither the local suffering nor the constitutional disturbance was severe or obvious. Mr. Cooke, my colleague, and of the Cancer Hospital, speaking of the arrest of cancer,† states, that experience has led him to believe that in a great number of these tumours there is a natural ebb-tide. At first the tumour grows rapidly, after a time slowly; it then remains stationary, and at last begins to waste, until gradually it almost disappears. These favourable cases are seen in persons of cheerful temperament.

Arrest of cancer in the aged is not an uncommon event. Two remarkable cases are recorded in the "Lancet" (July 17th, 1858). One—at the Cancer Hospital—was that of a healthy-looking old woman, with a ruddy complexion, and aged eighty-two. Scirrhus of four years' duration existed in her left breast, and she had been an out-patient at the hospital just twelve months. Under treatment of a palliative character only, the disease had become quite stationary, the tumour moveable, and somewhat diminished in size. The other case was that of an old woman, aged seventy-seven, a patient in the University College Hospital. A rather prominent cancer had formed in the left breast, which many surgeons had seen, all of whom recommended its removal by the knife, to which the patient would not consent. In this instance the disease had existed for seven or eight years, and was progressive only during the last eight or nine months.

Other modes of spontaneous cancer-cure are specified by Walshe in the work already referred to.

Thus, cancer-growth may terminate by "resolution and absorption." Weller‡ states that a medullary cancer of the eye of

---

* Nature and Treatment of Cancer, 1846, p. 134.

† Lancet, June 5th, 1858.

‡ Handb. der Augenheilk., S. 414, 1830.

an infant ultimately disappeared, and was followed by dropsy and atrophy of the eyeball. Travers also relates an instance of absorption, under circumstances which left no doubt as to the nature of the disease, and where no treatment worthy of the name had been employed,—only a deodorizing solution of chloride of lime. Yet scirrhus of the breast disappeared in a lady whose other breast had been extirpated for this disease. Not long after, she died of asthma, and a *post-mortem* examination showed that scirrhus had formed in several of the abdominal viscera.

Suppuration is another mode of spontaneous cure. M. Lévêque Lasource* cites an example. A man had a large scirrhous tumour removed from his back by Boyer. The disease returned. No operation was again performed; but subsequently, violent inflammation supervened; an abscess followed with profuse suppurative discharge, and then recovery.

Mortification may extirpate a cancer, although rarely. The whole tumour is thrown off by sloughing. Cases are recorded by Garneri,† Cruveilhier,‡ Sir E. Home, Cline,§ Steidele,‖ Dupuytren,¶ Richerand,** and C. T. Jackson.††

Cicatrization—and permanently established, after destruction by ulceration—has been witnessed in some cases. Nicod‡‡ supports this statement. Bayle saw several such recoveries. Svendsen§§ relates one, that of a man with an encephaloid cancer in his right thigh. This tumour ulcerated, bled frequently, and subsequently diminished in such manner that the patient left the hospital as cured.

Hitherto, I have illustrated the curative power of Nature by

---

* Thèses de Paris, 1807, p. 27.
† Bulletin de la Soc. Méd. d'Emulation, Dec. 1810, Sept. 1811.
‡ Essai sur l'Anat. Pathol., 1816, t. i., p. 127.
§ Lancet, vol. ii., p. 401. ‖ Journ. de Médecine, tom. lxxxii.
¶ Journ. Hebdom., 1829, t. iv., p. 38; Gaz. des Hôp., Nov. 24 and 31, 1831.
** Nosographie Chirurgicale, t. i., p. 381.
†† Mason Warren, Amer. edit., art. Cancer, p. 147.
‡‡ Bulletin de la Soc. Méd. d'Emulation, 1810, No. 1.
§§ Acta Soc. Reg. Med. Havniensis, 1829, vol. vii., p. 100.

the course and tendency, or career, of various kinds of injury, and by that of various diseases of nutrition. The operation of an innate restorative power, or property, of the living tissues, is equally conspicuous in the career of *Blood-diseases*. All these diseases run their appointed courses, and tend to this or that issue—regulated, possibly, but not controlled or overcome, by human interference. Art is powerless, otherwise than to *conduct* the disease to a favourable issue. For example, "you may," as Pitcairn remarked, "guide a fever; you cannot cure it. What would you think of a pilot who attempted to quell a storm? Either proposition is equally absurd. In the storm, you steer the ship as well as you can; and in a fever, you can only employ patience and judicious measures to meet the difficulties of the case."

Blood-diseases are eminently "self-limited;" but, moreover, their natural course and tendency are invariably to, or towards, recovery. There is in each case ample evidence of a restorative effort. So far, fatal as may be the issue of these diseases, they are manifestations of Restorative Power, and appropriate illustrations, therefore, of the Principle I am advocating.

Reviewing the whole class of Blood-diseases, their curative course and tendency are perhaps most conspicuous in the exanthematous fevers. The effort to spontaneous cure is clearly shown by the character of all the *constitutional* symptoms which idiopathic fever represents. During the "brooding-time" of any such fever, no restorative power is apparent; but the supervention of fever implies that 'reactionary disturbance' by which, if victorious, the blood-poison is expelled from the body. Such, at least, is always the *effort* of Nature. Reaction denotes acceleration of the circulation, as opposed to 'febrile oppression' of the nervous system; and it is responded to, in some respects, by the excreting organs seeking to expel the blood-poison; but, more especially, by the function of nutrition, in decomposing and destroying the poison by combining it with the natural tissues. This crowning act of all is evinced by a characteristic 'skin-eruption,' and its

maturation, after which the fever subsides. The blood-poison is expelled, and health eventually restored.

Such is, briefly, this natural process of recovery—self-directed, self-completed. Hazardous in all blood-diseases, perilous indeed in some, must this process of expulsion be; for, until completed, the whole body is deprived of that which is essential to life, and subsequently the blood is reinstated only by little and little instalments. The convalescence from any blood-disease is always protracted, and even precarious.

These general reflections on the self-curative course and tendency of blood-diseases are sufficient, without my further illustrating their generic character in this respect, by the career of each particular species.

Analogous restorative power is manifested by *reaction* responding to the 'shock of injury.' The nervous system is not, indeed, oppressed by a blood-poison, but overcome and prostrated, either by the sudden expenditure of nerve force, or by its suspended generation; and to meet the contingent demand, the heart possesses the power of itself reviving after a time, and reproducing the nervous force, by virtue of an accelerated circulation of blood. The heart responds to the duty suddenly imposed on it, and the phenomena of reaction are those of restoration. So strong, indeed, is this reserve power of the heart, and so eager is this organ to do its duty, that reaction is apt to pass beyond and overshoot the standard of healthy circulation—subsiding only, by temporary fits of reaction, down to the more even calm of health.

This natural reserve power is competent to establish reaction, without any artificial assistance, in the young and vigorous.

"A young man of unbroken and vigorous constitution was thrown into a dungeon at Hayti. Thumb-screws were put upon him, and a jug of water placed by his side,—to which, however, he could not apply his mouth, owing to the confinement and acute pain in his hands. The gaoler, who came to him occasionally, lifted the pitcher to his mouth, at which times he drank freely; in the intervals, his mouth was parched; he was restless, but dozed a little. In this state he remained upwards of forty-eight

hours, during which period he never felt the least inclination to void either urine or fæces. Some hours after the screws had been removed, he passed a moderate quantity of very high-coloured urine, after which the secretion gradually returned to its ordinary state, and he suffered no subsequent inconvenience."*

Similar instances might be cited in evidence of what I would term 'cardiac reserve force.' This force comes into operation whenever necessary to restore the balance of the blood's circulation, and to recover, chiefly, the functions of animal life. The efficacy, and occasionally the sufficiency, of this reserve force is attested by its overcoming febrile oppression of the nervous system and the shock of injury.

On the other hand, the nervous system is itself the source of certain occasional muscular actions of a restorative character, and all of which indicate what may be termed 'nervous reserve force.' This force is manifested by the involuntary muscular actions connected chiefly with the functions of Respiration and Digestion, by which each of these functions, being capable of adaptation to various evil contingencies, assumes a self-restorative character. They are expressions of restorative power emanating therefrom.

The function of Respiration is essentially that of aerating the blood—thus preserving its vitality, and eliminating certain excrementitious matters. This end is attained by the alternate movements of inspiration and expiration, through the action of those muscles which enlarge and diminish the capacity of the thorax; and these muscular actions are under the presidency of, and regulated by, a portion of the nervous system. But the acts of inspiration and expiration must be performed regularly and sufficiently, otherwise the essential purpose—aeration of the blood—is not duly accomplished. Whenever, therefore, this fails to be effected, the reflex nervous reserve force is displayed, and any deficient supply of air immediately compensated by extra and deep inspirations, as many as may be required to meet the demand. Respiration is thereby adapted to any such contingency, and the

* Constitutional Irritation. B. Travers.

function restored. By virtue of the reflex nervous reserve force, a long-drawn, involuntary inspiration, now and then, compensates the want of breath experienced by any one oppressed with care. 'Sighing' is the act of respiratory restoration, and it bespeaks some expenditure of that reflex reserve force which, operating through the muscles of deep inspiration, then comes to our relief. 'Yawning' is a still deeper inspiration, invoked possibly by the want of breath consequent on idleness.

Not only is a deficient supply of air thus remedied; but respiration is capable of meeting other contingencies—through other adaptations, depending also on the same reserve force, and whereby this function assumes a self-restorative character. It is enabled to maintain a due supply of air, by speedily removing any obstacle from the passages through which air is admitted and expelled. The bronchial tubes, trachea, and larynx are kept free by 'coughing,' the nares by 'sneezing;' and both these actions are accomplished by some expenditure of reflex nerve force *in store* for such occasions. How is it, then, brought into operation? The larynx is endowed with so high a degree of sensibility, that it immediately takes notice of any irritant accidentally present in the glottis. The smallest foreign body, or even a drop of water, is intolerable. Forthwith, an inspiration, as long and deep as possible, takes place involuntarily; the *rima glottidis* is then closed by the spasmodic action of its own muscles, and immediately a violent expiration ensues, with an expulsive blast of air, generally sufficient to carry any obstacle before it. The foreign body is thus dislodged and coughed up. So jealously watchful is the glottis of anything intruding, that even cold air may be rejected by this door-keeper of the lungs. The mucous membrane of the trachea and bronchial tubes is far less sensitive in health. Nevertheless it may become so, when subjected to continued irritation, or under the influence of nervous excitement. Bathed with their naturally bland mucus, these tubes are easy; let inflammation occur, and their preservative sensibility is soon discovered. The mucus of bronchitis is ejected, or coughed up, again and again;

while blood, pus, and tuberculous matter, are at once recognised as foreign bodies, and peremptorily ejected. Pneumonia and phthisis thus find vent by expectoration. In nervous asthma the mildest air is regarded as intrusive, and rejected accordingly. All this implies some expenditure of the stock of reflex nervous force, *reserved* for such occasions, and expended through the action of those muscles by which deep expiration is accomplished. The act of 'sneezing' is of similar character. Invoked by any foreign particles in the nasal passages, it is accomplished by a sudden and violent expiration,—the aperture between the mouth and larynx being more or less completely closed by the *velum palati;* and this expiration—analogous to cough—is a natural restorative provision, and a manifestation of reflex nervous reserve force, ever ready to be drawn upon as occasion may require.

The function of Digestion takes care of itself, in a measure, by certain *involuntary* muscular actions. 'Vomiting' and the act of 'diarrhœa' are protective provisions, by which indigestible food and poisonous matters are ejected; and both these actions are manifestations of the same reserve force. They are analogous to coughing. In both actions a deep inspiration is followed by the spasmodic closure of the glottis, and then an expulsive expiration, together with the contents of the stomach or of the rectum. The offending matters are thus expelled from the system. But these restorative actions are aided, in one case, by the muscular contractility of the stomach; in the other, by that of the rectum co-operating with the levatores ani muscles, and by the peristaltic action of the intestines previous to the act of defecation. In either way, the ingesta, solid and fluid, are ejected, if indigestible; and poisonous matters, swallowed accidentally or by design, are providentially expelled. Irritating secretions also—as bile, inflammatory mucus, and serum—are disposed of in like manner. The whole gastro-intestinal canal is thus kept clear of offensive matters, whether introduced as food or poison, or secreted from the blood. Blood-poisons are apparently excreted, in part at least, by the same means; for although chemical analysis has hitherto

failed to detect any such poison in matters vomited or voided from the bowels; yet the diarrhœa, persistent and peculiar in some blood-diseases, is significant. In typhoid fever, not only is the diarrhœa characteristic, but the inflammatory enlargement, &c. of Peyer's *glands* is perhaps *post-mortem* evidence of its having been the effort to excrete the typhoid blood-poison.

This *excretory* function of the intestinal mucous membrane, and the subsequent expulsion of poison thereby eliminated from the blood, form another phase of the restorative power vested in the intestinal canal, coupled with the action of those muscles which more especially expel its contents. By vomiting, and ordinary diarrhœa, the gastro-intestinal canal is itself cleared or cleansed; but that diarrhœa which carries off any blood-poison purges the blood, and *this* is restorative to the whole body. The intestinal canal, considered as an excreting organ, takes rank with the lungs, kidneys, and skin.

Associated with the function of Digestion, are two sensations —*hunger* and *thirst;* both of which, by their peculiar *inclinations*, in various diseases, exhibit the curative efforts of Nature. Hunger and thirst are sensations referred respectively to the stomach and fauces; but they indicate the wants of the system, in respect of food and drink. These sensations are indeed, in health, the surest guides to the *organic requirement* of alimentary matter,—not only in point of quantity, but that also of *kind*, although less apparently the monitors whereby the selection of articles of food is instinctively regulated. "What would you like for dinner?" is a question, the answer to which is at once suggested by a self-regulating choice of one or more particular kinds of food. And in *health* this natural choice is always the surest guide respecting the particular wants of the system then pressing. So likewise in *disease*, the quantity of food required is measured by instinctive desire. Loathing of all food in fever,—ravenous desire, in convalescence, to restore the consequent waste of the body. And the *kind* of food required is equally suggested by instinctive choice. "What food may he take? shall I give him a little of this or that?"

—"Let him have what he *fancies most*," is the answer sanctioned by the results of experience.

The instinctive longing for some particular *kind* of food is a wise provision of Nature in the cure of disease. Often unerring, always suggestive, this instinctive choice should ever be consulted, and its dictates fulfilled, or at least regarded. How often do we hear of some desperate and almost hopeless case in which, life being reduced to its lowest ebb, the food-fancies of the dying were indulged, just because such indulgence could do no harm. "Let him take whatever he likes best." But this last, and apparently hopeless, resource is not in vain. Ever-provident Nature, appealed to at even the eleventh hour, has not turned a deaf ear to our faith in her restorative power. From the moment *instinct* has been allowed to select the *kind* of food, life has rallied. This is even more wonderful than the instinctive measurement of the quantity required, which is suggested only as the sensation of hunger subsides during ingestion; whereas the right choice of food is an altogether *predetermining* instinctive suggestion. And the marvellously admirable accuracy of this restorative instinct of choice would be yet more conspicuous, should chemical research ever demonstrate that the longing for some particular chemically constituted kind of food in any particular disease is invariably connected with the inward want of that special kind of food consequent on that disease. The disease having created the want, would then suggest its own remedy. What conclusive evidence would this be of curative provision, and prompting to spontaneous cure; what an illustration of the curative power of Nature.

If, in convalescence from disease, the kind of food required be not taken, it is usually rejected by the stomach. Natural instinct selects that kind of food which the stomach will retain, and which supplies the particular and critical wants of the system.

This selective instinct and the efficacy of its promptings are further witnessed in the particular articles of food chosen by patients—apart from the teaching of experience—in diseases of the stomach. Food specially suited to the impaired digestive

powers of this organ is instinctively chosen. A remarkable instance of self-regulating choice of food, in a case apparently of chronic gastritis, occurred to Dr. William Hunter.* The story is told, in his own graphic language, to inculcate the efficacy of that curative treatment which avoids offending a very weak stomach either by the quantity or quality of food administered; but the course of this case and the directions given show the curative power of instinctive choice. The patient was ordered to begin with one spoonful of milk for a meal, then to proceed very gradually to a greater quantity, and to other fluid food, especially to whatever "his own fancy" might invite him. Guided thereby, the child—who had been reduced to the condition of a living skeleton by frequent vomitings—speedily recovered his wonted flesh, strength, colour, and spirits, and became "very importunate to have more substantial food," whereby he grew eventually into "a healthy and very strong young man."

There is, then, evidence that patients having disease of the stomach, or disease unconnected therewith, may safely trust the suggestions of their instinctive choice of food. There is also evidence of blood-restorative power vested in the gastro-intestinal canal,—by virtue of its function in excreting blood-poisons, and of the self-restorative or protective power of this canal by virtue of the cleansing operations, vomiting and purging. These operations are, however, chiefly the result of muscular actions depending on the reflex reserve force of a certain portion of the nervous system, and each implies some expenditure of such force.

Analogous restorative operations are effected by the respiratory apparatus. Deep inspirations—as sighing and yawning, whenever necessary, to compensate for deficient aeration of the blood; deep expirations—coughing and sneezing, whenever necessary, to clear the air-passages. The former operations have reference to the blood; the latter are self-restorative,—or protective of the working condition of the pulmonary organs; and both

* Medical Observations and Inquiries, vol. vi.

these kinds of operation—deep inspiration and expiration—are the result of muscular actions contingent on the issue of the same reserve force.

The Lungs are not merely excreting organs; but certain other organs are *solely* excreting, and are specially devoted to the purification of the blood. They severally eliminate its effete constituents; they mutually combine to maintain the balance of its composition. So also in disease these same organs eliminate from the blood matters morbid by quantity or quality, and they restore the balance of its constituents. Nay, more; in disease, excreting organs mutually help each other by vicarious or compensatory excretion; and when two such organs perform the same function in health, either supplies the functional quotum of its companion when disabled, and does so by double work. Double organs are thus mutually accommodating. Four adaptations therefore of excreting function are provided:—extra excretion; readjusting excretion; compensatory excretion; and supplementary excretion; and by either of these functional adaptations Nature may endeavour to recover the condition of health. Viewing the Restorative Power in its entirety, they are four additional manifestations of this Power.

Familiar to clinical observers as are these adaptations of excretory function, it is unnecessary to adduce more than a few examples. Others will be readily suggested.

The Kidneys excrete lithates or an *extra* quantity of lithic acid during the subsidence of gout. Such is the natural termination of a fit of gout. The blood is thereby purged of these noxious matters. Then, again, the kidneys excrete an excess of phosphates or of oxalates, whenever the acid of either of these salts respectively superabounds in the blood. *Readjusting* excretion, by means of the kidneys, is witnessed in the course of bilious cholera. Evacuations of bile leave a proportionate excess of urinary constituents in the blood, and the kidneys endeavour to readjust its composition by excreting a corresponding proportion, when the fever accompanying this disorder at once subsides.

Compensatory excretion by the same organs is of daily occurrence. Whenever the skin is less active than usual in the performance of its duty, the kidneys endeavour to compensate its deficiency by a more copious excretion of urine. This diuresis is most obvious if the perspiratory excretion be suddenly checked. Checked perspiration from exposure to cold soon finds vent by diuresis, the kidneys immediately undertaking their restorative function by a vicarious discharge. *Double* work on the part of either kidney is a more rare mode of functional adaptation. It implies the total loss of excretory power by one kidney, and the gradual hypertrophy of the remaining healthy one to double its former size. *During* this accommodating growth the work done cannot be double, and therefore a certain, although decreasing portion, of urinary constituents must remain, and perhaps accumulate in the blood. But when once double hypertrophy of the single organ is attained, double work begins and continues. The hypertrophied kidney, faithfully representing its disabled fellow, does the work of both, and the urine excreted is also of a perfectly healthy character. Supplemental excretion differs thus from vicarious discharge. The latter is only compensatory, and not identical with the healthy excretion it represents, because not the product of an organ identical in point of structure and function.

Other double organs besides the kidneys are, in like manner, mutually accommodating. Either lung being suddenly disabled, its companion forthwith undertakes to supply its function, and ultimately, it may be, does accomplish *double* work. The Skin is apparently less competent to undertake analogous adaptations of function. An *extra* quantity of sweat is excreted in some diseases, as a critical discharge to relieve the blood of some poisonous matter. For example, drenching acid perspiration is the natural termination of rheumatic fever. *Readjusting* excretion probably occurs from time to time. The skin as well as the kidneys may help to restore the blood's composition after excessive evacuations of bile; but this adaptation of function has not

yet been proved by clinical observation. *Compensatory* or vicarious excretion by the skin is often witnessed in cases of suppression of urine. The perspiration acquires a strong urinous smell. Fatal may be the termination of this disorder, yet Nature thus endeavours to compensate for the loss of restorative power by the kidneys. In some cases of suppression, compensation is apparently undertaken by the intestinal canal. Evacuations of fluid resembling urine are not uncommon; and in advanced Bright's disease of the kidneys, urea has, I believe, been actually detected in the vicarious intestinal discharges.

Observing these and other instances of compensatory excretion, it appears that vicarious discharge, of whatever kind, is effected either by some mucous membrane or glandular structure connected therewith, or by the skin. From one or other of these surfaces vicarious discharge may issue.

The organs of Reproduction in the female, quiescent as they are for a considerable period of life, undertake duties from time to time, during the active period of their career, which may be here appropriately considered.

The nature of *menstruation*, and the physiological purpose or purposes of its periodic return, have not yet been positively determined. This function is accompanied, though not invariably, with a periodic maturation and extrusion of ova; and it would appear that the menstrual flow of blood is destined for the production of a decidual membrane. Apparently therefore, at each menstrual period, Nature especially offers the opportunity for conception. Assuredly sexual desire by the female is experienced more especially about the time of menstruation, and conception is then most apt to follow sexual intercourse. If this be the physiological purpose of the menstrual discharge, then indeed it cannot be an excretion; otherwise than, failing to form a decidual membrane, it becomes an excrementitious discharge, or rather a superfluous overflow. But this decidual explanation of the physiological purpose of menstruation is, I conceive, not compatible with certain pathological facts. One is, the periodic recurrence of

menstruation in many cases of pregnancy, and even during the whole period of utero-gestation, *after* a decidual membrane has been formed. Then again, suppression of the periodic menstrual discharge—amenorrhœa—induces symptoms of *blood-poisoning*. Its retention in the blood destroys the red corpuscles, or prevents their development, possibly both; and is attended with that pallidity and general decline of the health known as chlorosis. Menstruation therefore appears to serve some *excrementitious* purpose, although possibly such is not its only purpose. This view is supported by other pathological evidence. Suppression of the natural discharge is followed in some cases by a *vicarious* discharge. The menstrual fluid is carried off through some other channel, usually by some glandular structure; and this relief is attended with amelioration of the constitutional disorder consequent on habitual suppression. It is apparently a compensatory excretion.

Instances of vicarious menstruation are not unfrequent. Some portion of mucous membrane, or of the skin, undertakes this compensatory office. The discharge may come from the lungs, stomach, intestinal canal, or bladder; and it is alleged that from the nostrils, eyes, ears, or gums, a similar discharge may issue. The skin, and certain portions more especially, as in the neighbourhood of joints, occasionally fulfils the same purpose. The mammæ also in some instances. Unlike the proper menstrual fluid, this vicarious discharge is less like blood; nevertheless it resembles the menstrual fluid, more or less, in point of colour and odour. The vicarious discharge occurs at the regular periods, or sometimes intermediately.

Granting the function of menstruation to be excretory; then certain disorders of this function may possibly assume a restorative character in relation to the blood. Menorrhagia may in some cases be an effort of Nature to relieve the system, by excretion of an *extra* quantity of menstrual discharge; in other cases this apparently excessive discharge may be contingent on some other flux, and serve only to restore the balance of the blood's composition.

Menorrhagia would then be a *readjusting* adaptation of the function of menstruation.

Should the excretory function of menstruation eventually prove to be one, if not the only, physiological purpose of this periodic discharge, it will be impossible to deny the value of Pathology, as an instrument, so to speak, in Physiological investigation. If, as I have elsewhere shown in this work, the method of investigation by experiments on animals affords only *data* for *inferential* conclusions respecting human physiology, pathology supplies the *data* for *positive* conclusions. Indeed, respecting the function of menstruation, pathology contributes almost the only *data* available.

In concluding the series of illustrations I have now adduced to exhibit the manifold operations and resources of the Restorative Power, there yet remains one aspect of this Power; to which, however, I can only briefly advert :—

The *readjusting* power of Nature, during *convalescence ;*—a provision which extends to the readjustment alike of structures and functions, and thus the general re-establishment of health. It is, in fact, the final effort of the Restorative Power, completing recovery.

Examples are coextensive with the whole range of Pathology; and any preferential selection is scarcely possible. I may as well take an instance of such readjustment, after a condition which cannot be called one of disease, and where the process of convalescence is simply readjusting. I mean convalescence after natural labour.

The parturient female is in a state of shock or collapse; and the uterus, although relieved of its burden, has, internally, a raw surface, like that of a large open wound; whilst the vagina and vulval opening have undergone considerable distension. From this state of constitutional disturbance, and local injury, a gradual *readjustment* of structure and function succeeds. Collapse, involving failure of the heart's action, with feeble pulse, and suspension of the nervous force, with exhaustion,—these disturbances of function are speedily readjusted. Reaction succeeds in the course

of a few hours. The pulse regains its force and frequency; but overshoots the average standard of the individual, at least in respect of frequency. The pulse may rise to one hundred beats and upwards per minute. After a few hours more have elapsed, this reaction partially subsides, then returns with the first secretion of milk, and afterwards again subsides to nearly the average standard. Cotemporaneously with reaction, the cerebral obscurity of collapse clears off, and exhaustion is exchanged for some returning strength. The respiration also, the frequency of which has hitherto undulated with the alternate rise and fall of the pulse during reaction, now resumes its average, even rate of frequency. The skin and kidneys gradually resume their average functional degree of activity. The digestive organs become less capricious, and the bowels more regular —neither relaxed with diarrhœa, nor bound with constipation, as during pregnancy.

Adverting to the state of the uterus and vagina; immediately after the act of parturition, the uterus contracts, and can be felt in the abdomen—now pendulous—to have contracted to about the size of an infant's head. The uterine vessels are thus squeezed and emptied, and hemorrhage prevented or arrested. Contraction having lasted for a short time, is followed by relaxation; then another contraction, and so on alternately, each successive contraction gaining ground on the preceding one. These repeated contractions gradually reduce the size of the uterus so much, that after about a week or ten days it begins to descend into the pelvis. Subsequently, the *fundus uteri* can usually be felt at the brim of the pelvis; but in the course of another week it recedes altogether beyond the reach of touch. Uterine contraction is induced whenever the infant sucks, and its repetition is denoted by "after-pains." Not however by contraction alone is the womb readjusted to its former size, locality, and pelvic relations. Its organic structure simultaneously undergoes the process of "fatty degeneration" (or disintegration, as I would call it) and absorption; followed by the development of a new tissue, in lieu of a portion of that which has been thereby removed. In the course of less

than a week after parturition, fatty transformation begins; the uterine substance, losing its firmness of texture and reddish hue, becomes brownish-yellow and friable. Absorption ensues, and about the fourth week the organ has entirely regained its original size. Then fibro-cellular tissue apparently is developed to partly supply the place of the muscular texture which had been temporarily provided to convert the uterus into a contractile organ. By the end of the second month the womb has recovered its former character in point of structure, as well as having previously regained its former size, locality, and pelvic relations.

I have said that the inner surface of the uterus, after parturition, resembles a large open wound. Its cavity is, however, very soon considerably reduced in size, by contraction of the uterine substance. That portion of the interior to which the placenta was attached is elevated and ragged, and the whole surface has assumed an ash-grey colour, smeared over with a slate-coloured discharge. The cervix and os uteri are apparently ecchymosed; the vagina also exhibits some evidence of recent distension, but this speedily disappears, and the passage, with its vulval orifice, soon resumes its former dimensions.

Hemorrhage having been arrested by the uterine contractions, a drain still continues, as "the lochial discharge," or "the cleansings," from the interior of the uterus. At first reddish and thin, unlike blood, it changes to a greenish, yellowish, and finally brownish discharge, subsiding in three or four weeks. This discharge, issuing from the uterine vessels which were in apposition with the placenta, ceases as the raw surface left by its separation gradually heals. The wound, so to speak, which was inflicted by the dislodgment of the placenta, being repaired, the uterus is refitted to undertake its periodic menstrual function.

Lastly, but not so in the order of occurrence, the mammæ secrete milk. About twenty-four hours after parturition, this provision of sustenance for the infant is supplied, and it continues as long as may be required. At first a thin serous fluid, it soon acquires the character of true milk. Its secretion is induced by

a persistent determination of blood, verging on inflammation, and accompanied by heat, swelling, tension, and pain, with general feverishness; which symptoms are, however, relieved entirely by free secretion, disposed of by the act of suckling from time to time. During lactation, the mammæ are adapted to their functional requirements; the supply is equal to, and regulated by, the demand; but this demand is only temporary, and when the proper time arrives, secretion ceases, without inconvenience to the infant or any discomfort to the mother. The breasts quietly resume their ordinary condition of inactivity. In short, these organs, no less than the uterus and constitutional powers, gradually readjust themselves after parturition, and regain their *former* state of health.

Such, then, is briefly the process of convalescence after natural labour. Nothing can more beautifully illustrate the Power vested in the body—in each of its several parts and functions—to recover, without any artificial assistance, the *balance* of health; a twofold state of structural and functional adjustment and co-operation, evidently ordained, by virtue of this self-restorative Power, to be the usual and natural condition; disease the exception. Such, obviously, is the intention of Nature, and it becomes more apparent by the *frequency* of self-restoration from disease and injury in the course of each individual existence.

Nay, more than bodily restoration is provided for. The *mind* is endowed with a similar Power of self-recovery from the bitter trials and vicissitudes to which it is subjected during this world's pilgrimage. Early struggles with poverty; the shock of disappointed hopes, and the blast of withered expectations; pecuniary losses and reverses; the venomous wounds of calumny; the faithlessness of false friends; the death of a true one; the wrenching apart of natural affections by the rough grasp of Death;—these and a thousand other wounds are successively filmed over as time elapses, and eventually soundly cicatrized. The mind recovers—not indeed the elastic buoyancy of youthful spirits, but its wonted energy and unconquerable will. Regulated only, but not subdued,

the intellectual powers remain unimpaired; with the affections and passions chastened by this cruel-kind ordeal. "Covered with wounds, yet without a scar," was the expression made to me, and the experience of one in approaching the haven of death after his long and rough passage through life.

The Principle inculcated in this chapter has, I conceive, been amply illustrated and firmly established. The existence, operation, and resources of an innate Restorative Power, self-*sufficient* in *some* disorders to ensure recovery; a natural curative effort and *tendency* in *all* diseases and injuries—is, I affirm, a Principle which, upon the evidence I have adduced, cannot fail to be recognised and acknowledged.

This Principle is the *immediate* foundation of (Conservative) Therapeutics—Medical and Operative. While, therefore, the *earliest* and *most exact* Diagnosis is essentially conducive thereto, and the value of a similar standard of Etiology, as regards the operation of (persistent) internal causes, must be equally conceded; yet the earliest and most exact Prognosis or foreknowledge of the *natural* course and tendency of disease or injury is that aspect of Clinical Medicine and Surgery which will immediately guide and regulate the (Conservative) Practitioner.

Unhappily, 'the natural history of disease' has hitherto been watched but little, and recorded less. Experience, so called, is too often an indefinite impression, rather than the accurate knowledge to be acquired only by recorded clinical observation; and such observation is itself too often misled by the operation of medicinal agents, and by the whole influence of therapeutic interference. The *natural* course and tendency of disease and injury is, comparatively speaking, unknown. Consequently our foreknowledge is limited, and in but few instances has it attained, or even *approached*, that standard of *earliest* and *most exact* foreknowledge which is necessary to meet the full requirements of (Conservative) Practice.

In so far, however, as this standard has been attained, such

foreknowledge of the natural course and tendency of any morbid condition towards an unfavourable issue announces the *earliest occasion* for interference, and indicates the unfavourable conditions, or it may be the complications, to be removed if possible; while an equal foreknowledge of the natural course and tendency towards recovery—representing, as they do, the degree of Restorative Power in operation—indicates the *least* amount, as well as the *kind*, of *assistance* solicited by Nature during the case, to conduct it to a favourable issue.

Such Prognosis is, therefore, the *immediate* source of guidance and regulation in the Practice of (Conservative) Medicine and Surgery.

THE PRINCIPLES OF THERAPEUTICS,

MEDICAL AND OPERATIVE.

The Guiding Principle of Therapeutics is established by considering all the preceding chapters on Prognosis—or foreknowledge of the natural course and tendency of injuries and diseases, individually to, or towards, a favourable or an unfavourable issue.

Pathology alone determines the *earliest* occasion for, and the *least* amount, no less than the kind, of interference and assistance—medical, operative, or both—necessary to aid and complete the operation and resources of the innate Restorative Power.

The eminently Conservative character of this Principle is obvious, and converts its Medical aspect into the Conservative Practice of Medicine, and its Operative into Conservative Operative Surgery.

CHAPTER XVI.

PATHOLOGY, THE GUIDE IN SURGICAL OPERATIONS AND MANIPULATIONS.

The twofold condition of 'diseased' or 'injured' and 'living' textures and parts, is *alone* our guide as to the *kind* and *least* amount of instrumental and manipulative proceedings in Surgical Operations.

This Principle is also our guide in Surgical Manipulations, apart from Operations, and in the application of Surgical Apparatus.

The Conservative value of this general Principle illustrated by the chief Operations of Surgery.

THE important relation of Anatomy to Operative Surgery has long since been urged, but the exact relation of Pathology thereto would not appear to be so generally acknowledged, or indeed understood.

One feature of its application is, to determine the necessity for *any* interference whatever with the operation of the Restorative Power; and if so, to appoint the earliest occasion and least amount, no less than the kind of assistance, according to the operative results of that Power, towards the reparation of disease or injury.

Another aspect of its application is, to guide and regulate the *subsequent* management of a Surgical operation, and to estimate its Therapeutic value by the results of its assistance combined with the operation of the Restorative Power.

This twofold influence of Pathology on our conduct *before* and *after* Surgical operations, is, however, only in conformity with the general relation of this science to Therapeutics, the Principle of which I have already endeavoured to establish.

But the guidance of Pathology *during* (the performance of) any surgical operation is its direct relation to Operative Surgery, and the Principle thereof yet remains to be established.

On reviewing the history of Operative Surgery in this respect, we discover ample evidence of its past reliance on Anatomy *alone*, whilst the superaddition of *Pathological* Anatomy has been either wholly overlooked or only partially recognised; and with the view of fairly showing *how far* Surgical Operations and the teaching of the Schools have alike been guided by the Principle I am about to establish, I shall make extracts from certain works on Operative Surgery, which have been successively acknowledged and received as standard authorities down to the present day. I have selected that epoch which comprises the history of modern Surgery, when, as an art, it began to assume a definite position in this country and in continental Europe.

Read the melancholy reflections of John Bell in the year 1801, and gather the fruits of his profound experience. "Anatomy," said he, "is not made interesting to the pupil as the basis of our reasoning on disease—the rule of our prognostications, the sole guide in operating; he is taught to know the parts and remember their names, and then is dismissed from the School; and of this School we all are pupils, abandoned early in our career,

left to learn, in the hurry of practice, the very Principles of the Science.

"The Anatomy of Surgery differs widely from that taught in the Schools: it were better the young surgeon had no conception of the forms of parts, than such as must be corrected by sad experience; for the parts of the human body are presented on the table of the anatomist, not only in circumstances but in forms in which they can never at any after period appear to the surgeon. If the bones are demonstrated, they are not displayed clothed with their muscles, and connected by their periosteum with the surrounding parts; they are not represented as parts of a living system, nourished by vessels, and subject to changes and diseases analogous to those of the living body: these are conceptions which the surgeon attains by slow and painful experience, for his teacher aims only at making him know and remember processes and grooves and holes, which to know is of no importance, and to remember impossible. The practical surgeon, indeed, learns by experience the changes of which those apparently inanimate parts are susceptible; but while he is observing how tumours rise and vanish, are produced by disease, or cured by remedies; while he is learning to discuss the tumours, to cure the ulcers, to destroy the dead and to support the living parts of a bone; while he is acquiring by experience all that he should have been taught, all that makes Anatomy useful, he believes that he is forgetting anatomy because he is forgetting a Gothic and barbarous system of names, remembers no longer holes and hollows of no import, and is exchanging the pedantic lessons of the schools for a higher kind of knowledge."*

Even Italy, the oppressed and desolate, responded to this spirit of progress; for thus wrote Scarpa in 1809:—"There are a certain number of surgical operations for the prompt and safe execution of which mere anatomical knowledge will suffice; but in many others the surgeon cannot promise himself

* Principles of Surgery, 1801, vol. iv., p. 4.

success, even though he be well acquainted with Anatomy, unless he has particularly studied the numerous changes of position and alterations of texture, of which the parts upon which he is about to operate are susceptible. If he has not the requisite information upon all these points, false appearances may deceive his judgment, and make him commit mistakes, sometimes of a very serious and irreparable kind.

"In order to have a convincing proof of this truth, it will be sufficient to take a view of the different species of herniæ and their numerous complications. Assuredly, no anatomist would believe that the cæcum, naturally fixed in the right iliac fossa, and the urinary bladder, situated at the bottom of the pelvis, could undergo, without being torn, so considerable a displacement as to protrude through the abdominal ring and descend even into the scrotum; that the same intestine—the cæcum, could pass from the right iliac region to the umbilicus, protrude at this opening, and form an umbilical hernia; that the right colon could have been found protruded from the abdomen at the left abdominal ring, and the left colon through the right one; that the liver, spleen, and ovary could sometimes form the contents of umbilical, inguinal, and femoral herniæ; that the cæcum could engage itself within the colon, and even protrude at the anus; that the stomach could be forced through the diaphragm, and form a hernia within the chest; that the omentum or intestine, or both these parts together, could sometimes escape from the belly through the foramen ovale or sacro-ischiatic notch of the pelvis."*

In this country the guidance of Pathology *during* Surgical Operations was long altogether overlooked. For instance, early in the present century, 1807, or upwards of fifty years ago, there appeared a purely Anatomical Operative Surgery,† by Sir Charles Bell. More recently, after the lapse of forty years, reference was made by Mr. Liston (1846) to the influence of Pathological Anatomy on the "abridgement" of Operative Surgery; but in

* Sull'Ernie, 1809.

† System of Operative Surgery founded on the Basis of Anatomy, 1807.

the same edition of the work referred to,* and in similar works by other eminent surgeons, due notice is taken of instruments and their uses, with the methods of performing Surgical Operations, guided by the knowledge of Anatomy *alone*.

Before the resources of Pathological Anatomy had influenced the teaching, or perchance the practice, of Surgery in this country, we can readily trace their application thereto in France. The more early pursuit of pathological anatomy as a science, and the recognition of its special influence on the performance of surgical operations, are alike due to our Gallic brethren, who, in these respects, have certainly anticipated us. For the same year in which first appeared the "Practical Surgery" of Robert Liston, produced also an analogous work† by M. Vidal (de Cassis), wherein, however, the following remarks occur:—"If all operations were performed on sound parts, the normal state being known, one could lay down principles which should apply to all operative proceedings, and which would only then necessitate modifications depending on situation—modifications, moreover, which Anatomy could foresee. But it is not always so. There are even numberless cases where the operator is thrown upon his own resources by anomalies of pathological effects, which have changed the form, disturbed the relations of parts, and altered their consistence and colour."

At length British surgeons slowly recognised the influence of Pathological Anatomy. Mr. Fergusson alluded to this position in the third edition of his Treatise.‡ The nature of "Surgical Anatomy," so called, is thus defined:—"From what has been stated, it will appear that I include a *certain* amount of knowledge of what is termed Pathological Anatomy in my definition of Surgical Anatomy." And again:—"Having these views with reference to surgical anatomy, I shall, in the following pages, where I deem the subject of *much* importance, not confine my descriptions to the healthy parts alone, but shall *occasionally*

* Practical Surgery, 1846.

† Traité de Pathologie Externe et de Médecine Opératoire, 1846.

‡ Practical Surgery, 1852.

digress where it may appear advantageous to do so." The italics are my own. Here, then, the *occasional* application of pathological anatomy to the performance of surgical operations is acknowledged. But more absolute are the acknowledgments of M. Sedillot, in the following year (1853); for says he,—"The study of pathological anatomy *completing* normal and surgical anatomy, comes to add to the certainty and to the resources of operative medicine; and the methods of reducing dislocations, fractures, the proceedings in most amputations, dans la contiguité des membres, the ligature of arteries, bony resections, removal of the anterior ramus of the lower jaw, the preservation of the corpora cavernosa protected from cancerous degeneration by their fibrous sheath, are so many proofs of the immense assistance that this science derives each day from pathological anatomy."*

The foregoing statements tend to elucidate the general nature of the question under consideration; but the *universal*—or unexceptional guidance of Pathological Anatomy in the Operations of Surgery is not recognised in practice, and still less in precept. Pathological anatomy is not regarded as the polar star of Surgical Operations; and if so recognised, we yet find the *purely* Anatomical element prevailing in the Descriptions in books; the delineations themselves relapse into mere Anatomical Dissections; and I am not aware of any one work on Operative Surgery in which the Surgical Anatomy, so called, is modified throughout by constant association with Pathological conditions.

Yet it is precisely under these circumstances that the Operating Surgeon interferes. He is never called on to touch the body in its healthy anatomical condition.

True it is, that certain operations are *apparent* exceptions to this otherwise unexceptional law. It may be that the seats of operation and of disease or injury are not identical;—that our operation is somewhat removed from the diseased or injured locality. Such are amputations, and the ligature of arteries for

---

* Traité de Médecine Opératoire.

aneurism. But even under these circumstances, pathological anatomy can alone determine whether or not we operate clear of the disease and amid healthy tissues;—whether, for example, the bone and soft parts left after amputation are healthy, and whether we cast our ligature around a healthy portion of artery. This *negative* application of pathological anatomy is obviously of the highest importance in respect to the successful results of surgical operations.

On all other occasions, the physical properties and relations of parts disclosed during an operation are then so changed by disease as sometimes scarcely to admit of recognition; and thus it is that anatomy *plus* certain pathological alterations, or the *pathologico*-anatomical conditions of the body, are those with which the operating surgeon is concerned. All the surgical value which has hitherto been universally conceded to Anatomy should, therefore, be transferred to this compound—*Pathological* Anatomy.

Guided by this *à priori* principle, we can predicate those pathological conditions which from their nature must chiefly influence the performance of surgical operations;—alterations of certain physical properties, more especially of colour, consistence, and elasticity; also modifications of shape and size, with those affecting the situation, position, and relation of parts;—such peculiarities altogether change the scene with which the mere anatomist is familiar.

Nor are these the only circumstances that overshadow the appearances to which he is accustomed. Whoever has observed the arm of a dead subject as it lay extended over the side of a dissecting-table, must have been struck with the well-marked bicipital depression, especially visible on a thin subject. If injected, the brachial artery can almost be distinguished as a prominent line throughout its course. Apparently, a single incision would bring one down upon the vessel, and so it does. The skin, gluey and adhesive, hangs upon the knife; nor does the incision gape; the artery is soon exposed, not being overlaid by the contracted biceps, and only, perhaps, obscured by the turgid vein on its inner

side. Contrast all this with the same proceeding during *life*. We observe no such well-defined groove to guide our incision; the skin yields before the knife with a crimp and elastic resistance; the wound gapes; the swollen and vibratile belly of the biceps muscle, especially if amply developed, overlays the artery, while the vein, perhaps not so turgid, immediately conceals it. More or less hemorrhage will also further obscure the vessel, which can only be recognised by its beautiful fawn-colour and its pulsating under the finger. I have purposely excluded the brachial plexus of nerves from this sketch, in order more clearly to contrast the *dead* with the *living*.

This illustration will apply, *mutatis mutandis*, to operations for the ligature of other arteries; and the experience of every practical surgeon will supply him with the more extended application of the same principle during all other surgical operations.

But if pathological anatomy has not hitherto been sufficiently regarded as the foundation of operative surgery, still less have the peculiar appearances and conditions of living tissues been recognised. We must, however, acknowledge the guidance of *living* (not merely functional) conditions during the performance of surgical operations. The condition of life modifies certain physical appearances, and chiefly those affected by *pathological* anatomy. Thus, the colour, consistence, elasticity, and even the size and shape of the various parts of the body,—their situation, position, and relation to contiguous parts,—are presented to the surgeon, when modified by the twofold conditions of *Disease* and *Life* combined.

These together may be termed Pathology, and not Pathological Anatomy, which represents only *dead* structural disease. Pathology is, therefore, our guide during surgical operations. In proportion as we are familiar with pathological conditions, by so much are we enabled to foresee, and to provide for, the peculiar appearances and conditions which the knife discloses, and to recognise them as they are successively presented in surgical operations.

Guided by this anticipatory knowledge, our operations are no longer discoveries made by dissection, but *planned* and *methodical*

proceedings, conducted on known principles—in fact, an ART, based on the science of Pathology.

When a pupil of the late Robert Liston, I frequently witnessed, and for many years observed with care, the operations of that illustrious surgeon; and I venture to affirm, that if one circumstance more particularly contributed to the ease and elegance of his operations, it was the *suitable* (artistic) *method* on which he planned and conducted them. If I might use the expression, there was a kind of *slow-quickness* about all he did,—the former quality indicating the method he pursued, the latter resulting from its sufficiency and skilful application.

The twofold Principle I have advanced will be further elucidated by tracing the *combined* influence of disease and life on the scenes of the chief Operations of Surgery.

And here the results of *inflammation,* meeting us so constantly, first invite attention. The swelling concurrent with an effusion of serum or pus (diffused) deepens and displaces those tissues amid which it infiltrates. Their physical properties are soon changed. Cellular tissue, so abundant, assumes the appearance of wet tow; muscle becomes soddened and discoloured; the larger blood-vessels perhaps alone escape, appearing dissected out and isolated from their fellow tissues. Shortly, the fibrin effused solidifies, and its more or less complete organization ensues. If the latter change,—then induration and subsequent contraction of the tissues affected, together present pathological conditions which contrast with those arising from the mechanical agency of fluid. In due time the indurated tissues contract and disturb the relative position of adjacent parts. If *gangrene* supervene, then we observe the gradual loss of all the physical properties peculiar to each living tissue, as severally they are consigned to the uncontrolled dominion of chemical forces. Observe the skin darkened or black, and also the intermediate tints of tissues between it and bone, whitened and rough. Moreover, the tissues resign their consistence and elasticity as they imperceptibly lose their organization and return to a liquid form. I

would not locate these changes, because with unimportant differences they may occur in every region with which the operative surgeon is concerned.

If we review the history of other products not due to inflammation, we observe them also disturbing the relation of parts surrounding their locality, and by pressure gradually obstructing and even obliterating hollow organs in their vicinity. The clinical history of all tumours exemplifies these mechanical results; but *growths* more especially, by their gradual and unlimited enlargement, produce such displacements. This circumstance chiefly determines the necessity for extirpating tumours which are otherwise harmless in their local and constitutional consequences. I would not allow such benign growths, as fatty or fibrous tumours, to remain too long imbedded; for, although the general health may continue uninfluenced thereby, they may yet by their gradual enlargement so far encroach on adjacent and important parts as eventually to render their own removal difficult, if not impracticable. This indication more especially refers to tumours situated in regions where vital structures are crowded together. Such are the face and neck, where the presence of large vessels, nerves, &c. complicates the removal of tumours. Under these circumstances I have lately* removed more than one tumour, which, although of innocent tendency, would soon have passed beyond the ramus of the jaw posteriorly, and had already encroached on the cavity of the mouth. This condition would suggest the early extirpation of tumours (benign in themselves) when lodged in the sides of other cavities; as, for example, the abdomen and joints. Thus placed, they might by pressure and inward progress eventually endanger life, if their removal were attempted at a later period.

Next, inspect certain *regions* of the body, in which the relation of their pathology to surgical operations being more special is even more conspicuous.

Observe *aneurisms*,—say one of the axillary artery. The mere

---

* Lancet, December 6, 1856.

anatomist would suppose that the subclavian artery in the outer third of its course would offer no special obstacles to its easy deligation. On the dead subject but few exist. The operator observes the depression over the vessel, in the situation supposed, above the clavicle. He makes his incision along that bone; he very soon recognises the subclavian vein below, branches of the brachial plexus above; and the shoulder being now depressed, he at once passes a ligature around the artery. But with the shoulder *elevated* by an axillary aneurism, and the artery above having a corresponding depth,—the vein turgid from obstructed return of blood (more so under chloroform),—the sterno-mastoid and omo-hyoid muscles swollen and vibratile, with perhaps also a portion of the trapezius;—these modifying conditions of disease and life combined, all conspire to render this operation perhaps the most difficult in surgery. They tried the skill of a Liston and a Dupuytren; the former of whom at first ligatured the lower cord of the plexus instead of the artery; the latter, at the end of one hour and forty-eight minutes, only succeeded in casting a ligature around the fourth cervical nerve! Need I say more for the influence of aneurisms on adjacent parts during life?

Turning to *hernial tumours,* as I would term them for my present purpose, how significant is the relation of Pathology to Operative Surgery! The mere anatomical knowledge of the so-called 'coverings' of herniæ will avail but little when tested by an operation on the living body. I can understand the coverings of a piece of intestine when thrust in the direction of a hernia through the crural or inguinal apertures in the dead subject. We might then dissect and display the superimposed layers of tissue thus artificially made tense. We might successively remove the skin, superficial fascia, intercolumnar and cremasteric fibres, with perhaps also some cellular tissue and the fascia transversalis from off the sac of peritoneum, successively covering a portion of intestine thus protruded through the internal inguinal aperture. But I cannot understand how all this applies to a recent oblique inguinal *hernia,* and still less to one of former date; for then the

external and internal (or rather deeper), inguinal apertures are found so approximated, and the 'coverings' enumerated so matted together by constant pressure, as to have altogether effaced their mere anatomical characters and relations. In like manner, what are the 'coverings' of femoral *hernia?* One stroke of the knife might bring us down upon the sac, through skin and fasciæ—superficial, cribriform, and transversalis: where is the septum crurale? It therefore appears that Anatomy can be used only as a *diagram* for the study of herniæ, and that, like other pathological conditions, we must rely on dissections of herniæ themselves to direct us during operations for their cure. In short, we can no longer put our trust in Anatomy *alone*, but may rely with confidence on Pathology as our guide.

Looking around the *urinary bladder* and *neighbouring organs*, we again discover the intimate relations of pathology to surgical operations. The thin scrotal skin and tunica vaginalis may easily be transfixed with a common needle, and one might therefore suppose that the fluid of a distended hydrocele would always be readily detected and evacuated. Apart from pathological experience, no one could have predicated from mere anatomical knowledge that the tunica vaginalis may itself become so thick and dense as not only to disguise the fluctuation and translucency of hydrocele, but to yield before the point of a trocar with the resistance of thick pasteboard.

Should the *testicle* require removal, how much are our incisions modified and the adjacent parts endangered by the enormous bulk to which this organ and its coverings sometimes attain? The urethra and fellow testis might be wounded by a bare turn of the knife. Anatomy *per se* can only furnish our landmarks under these circumstances, and we must trust to our actual experience of such tumours during their removal from the living body.

Again, the every-day operation of *catheterism* cannot be learnt on the dead subject. The parts are then so peculiarly flabby, and the urethral passage so adhesive to even a well-oiled catheter, as to communicate no sensation of that grasp of the instrument which an operator experiences during life. If, indeed, the urethra

be indurated and contracted with *stricture*, then certainly we can learn nothing but the direction of the passage by practising on the dead body.

Approaching the *bladder*, and inspecting the scene of other operations designed to gain an entrance thereto, we find, no matter where we seek admission, that our operations are guided by anatomy, modified by the twofold conditions of disease and life combined. Is it our intention to remove a calculus by the lateral or Cheselden operation? Then what availeth it us to know that our incision through the skin may also divide the superficial perineal branches of artery and nerve, and will inevitably sever the inferior hæmorrhoidal? No anatomical knowledge, however exact, could foretel how far distant the artery of the bulb may be from the margin of the triangular ligament which is not seen; nor can we discern the transverse artery and muscle, and still less perceptible are a few fibres of the levator ani. Yet such are the misleading details of Anatomico-Surgical works respecting "the parts cut in lithotomy," or "the structures divided in this operation." All we know is, that as our first incision was directed by anatomical considerations, so also that a trifling stroke or two of the knife is continued until, aided by the point of our left forefinger, we touch the groove of the staff just in front of the prostate; the knife being then carried through it into the bladder, is followed by the finger; this, at once a protector (of the rectum), blunt gorget and guide, is required to discover the *pathological* conditions of the prostate and bladder, as it dilates the former and explores the latter.

Should *retention of urine* demand relief, the same *pathological* knowledge is needed. The variable size, shape, and consistence of the prostate, and even its relative position to the neck of the bladder, as altered by disease and examined during life, can alone determine our pathway when guided no longer by anatomy and lost to sight.

The foregoing illustrations are sufficient evidence of what Pathology alone can do for the performance of Surgical Operations.

And here I shall conclude—not that the thread of illustration has snapped in our hands, but that much more being suggested to the minds of experienced surgeons, will fully corroborate and further elucidate the twofold Principle I have endeavoured to establish.

Need I say more for Pathology—for Anatomy attired in the garb of disease and life?—assuredly the only ground of confidence and comfort to the Operating Surgeon, of safety to the Patient, and of the most enduring Professional reputation. The tribute of respect, therefore, hitherto paid to Anatomy, in relation to Practical Surgery, must henceforth be transferred to Pathology; for the latter includes the former, and much more—this, too, of immediate practical consequence.

Without *such* knowledge, I would say—"the operator is seen agitated, miserable, trembling—hesitating in the midst of difficulties, turning round to his friends for that support which should come from within, feeling in the wound for things which he does not understand; holding consultations amid the cries of the patient, or even retiring to consult about his case, while he lies bleeding, in great pain and awful expectation."

But the old carpenter style of Operative Surgery, derived merely from the dissecting-room, is fast passing away; it belonged to a period when Surgery ranked with the mechanical arts, and shared their honours. Pathology will henceforth be recognised as the polar star of Operative Surgery; and the brief review I have presented of this Science in relation to the chief Operations of Surgery, establishes its guiding Principle—that the twofold condition of diseased or injured and living textures and parts can alone guide our instrumental and manipulative proceedings in Surgical Operations.

This guidance also fulfils the cardinal Principle of Therapeutics—viz., the *least* amount, no less than the kind, of interference and assistance which may be necessary to complete the operation of the Restorative (or Reparative) Power of Nature.

Pathology is therefore the foundation of 'Conservative' Operative Surgery.

CHAPTER XVII.

THE PRINCIPLES OF SURGERY

(AND OF MEDICINE CONCURRENTLY),

ARRANGED SYNTHETICALLY.

The Practical value of Pathological Principles of Therapeutics should be tested and estimated by STATISTICAL RESULTS of the unaided Restorative Power, of Medicinal agents administered, and of Surgical Operations and manipulations conducted, in conformity therewith.

The Statistical method of investigation.

General Conclusion.

WHOEVER has read with care, and reflected on, the foregoing pages, will be prepared to subscribe to the following as the guiding Principles of Surgery—Clinical, Medical, and Operative; and also their derivation *ultimately* from Clinical Pathological Anatomy, or the application and guidance of Pathological Anatomy during life, in each such aspect of Surgery.

As Principles, they are concise Propositions; and few, it may be said, in number; but the growth of knowledge is unlike that of other things,—for the more it increases, its bulk diminishes. "All of them may be called Principles when compared with a thousand other judgments, which are formed under the regulation of these *primary* propositions."

PATHOLOGICAL SURGERY.

THE PRINCIPLES OF CLINICAL SURGERY,

OR,

THOSE OF DIAGNOSIS, ETIOLOGY, AND PROGNOSIS.

THE PRINCIPLES OF DIAGNOSIS.

Negative Principles.

I.—Anatomy and Physiology are respectively incompetent guides to an early and exact Diagnosis.

II.—Pathology—Functional disturbances—constitute an insufficient guide to an early and exact Diagnosis.

Functional disturbance does not invariably accompany, and does not at the earliest period accompany, injury or local disease; and the same disturbance of Function is no measure of the Structural lesion existing, may also accompany the same injury or disease simultaneously in different parts of the body, or may accompany very different kinds of injury or disease. Functional conditions, therefore, are only Symptoms, *i.e.* casual coincidences, not exact Signs of morbid conditions.

Coroll.—Hence the insufficiency of purely Clinical observation in this Department of Surgery.

Positive Principles.

I.—Clinical Pathological Anatomy, or Pathological Anatomy applied during life, supplies the earliest and most exact Diagnosis.

Sub-Principles.

a. By Physical signs—alone—insufficient. Physical properties are most readily recognised during life at the earliest period of disease or injury, and therefore at a time when the morbid condition itself is least complicated and most remediable by the simplest measures; but such properties are, nevertheless, the most equivocal or inexact signs. Unaided Physical Diagnosis, therefore, fails in the Practice of Surgery.

b. By Structural characters—alone. Changes of minute structure can be detected during life at the earliest period by puncturing Growths and Deposits situated near the surface of the body, and examining with the microscope these and all other morbid products found in discharges and secretions issuing from internal parts and organs. Moreover, these alterations of structure, thus detected, are more constant in any given case than those of physical properties, and are therefore more exact signs of the particular morbid condition in question; and being also discoverable at the earliest period, before the supervention of complications, supply the chief method of Diagnosis, whenever such minute structural alterations are available.

c. By Chemical conditions—alone. Alterations of Chemical composition are, indeed, the most essential condition of disease, but also the least readily detected and identified. Chemical Analysis, therefore, fails in Diagnosis.

THE PRINCIPLES OF ETIOLOGY.

I.—Clinical Pathological Anatomy is the guide to the earliest and most exact detection of 'internal causes' during life.

Thence the Principle of Prevention in the Practice of Medicine and Surgery.

Pathological Anatomy can be applied during life to most early and exactly detect and discriminate the structural condition, situation, and extent of diseases and injuries regarded as internal causes, in order to the Prevention of their conseqences,—by the extension of such local morbid conditions to textures and organs (continuous and) adjoining, and the sympathetic affections of organs and textures remote (including Constitutional affections); also the disorganizing results in the texture or organ first affected. This Principle can be extended to the Prevention of the local consequences of Constitutional disease.

II.—Pathology shows the Operation of 'internal causes' by their Functional manifestations.

Coroll.—Hence the accessory guidance of purely Clinical observation in this Department of Surgery.

Local may proceed from Constitutional morbid conditions; as, of the blood, nervous system, or both co-operating.

Constitutional may proceed from Local morbid conditions: injuries of mechanism; diseases of nutrition, including inflammation; from perversions of the digestive process, of excretion, or of respiration; or lastly, from contagion.

Local may proceed from Local morbid conditions of textures or parts—continuous, contiguous, or remotely situated in the body.

Constitutional may proceed from Constitutional morbid conditions, reciprocally those of the nervous and vascular systems.

Internal causes may be associated, either by co-operation or succession, in the animal economy.

THE PRINCIPLES OF PROGNOSIS.

I.—Clinical Pathological Anatomy is the guide to the earliest and most exact foreknowledge of the course and tendency of diseases and injuries individually, to or towards a favourable or an unfavourable issue, by representing the essential conditions on which their career individually depends.

Sub-Principles.

a. The persistence or not of the immediate cause or causes, and therefore the question of an internal cause, or more, being in operation, is the *immediate* basis of the earliest and most exact Prognosis, unfavourable or favourable respectively.

b. The earliest and most exact Prognosis of a disease or an injury is *regulated*

by the kind and extent of *structural* alteration that the organ or texture has undergone; and moreover, by the period during which such alteration of structure has been in operation as an (internal) cause of functional disturbances of the system. Nevertheless, an acute disease or a recent injury is, *cæteris paribus*, more unfavourable than a chronic lesion, to which the system has become habituated.

II.—Pathology declares the course, tendency, and issue of morbid conditions, whether disease or injury, by their continued Functional manifestations.

Coroll.—Hence the conclusive guidance of purely Clinical observation in this Department of Surgery.

Sub-Principle.—The comparative Functional importance, and influence, of the organ or texture *determines* our Prognosis, unfavourable or favourable, as to the course and tendency of any morbid condition of structure it may have undergone.

Organs and Textures which fulfil Functions by virtue of their vital endowments (and chemical composition) suggest an unfavourable Prognosis.

Organs and Textures which fulfil mechanical functions suggest a more favourable Prognosis.

Local disease or injury, *per se*, suggests a favourable Prognosis.

Local disease or injury sustaining, or sustained by, any Constitutional disorder suggests an unfavourable Prognosis.

Constitutional diseases, implying each some morbid condition of a texture or textures, of general distribution, as well as of predominant Functional influence throughout the system, suggest an unfavourable Prognosis.

The Restorative Power,—its existence, operation, and resources, manifested by the natural course and tendency of injuries and diseases, individually, to, or towards, recovery.

THE PRINCIPLES OF THERAPEUTICS,

MEDICAL AND OPERATIVE.

Pathology alone determines the *earliest* occasion for, and the *least* amount, no less than the kind, of interference and assistance—Medical, Operative, or both—necessary to aid and complete the operation and resources of the innate Restorative Power.

The eminently Conservative character of this Principle is obvious, and converts its medical aspect into the Conservative Practice of Medicine, and its operative into Conservative Operative Surgery.

In Surgical Operations and Manipulations.

The twofold condition of 'diseased' or 'injured' and 'living' textures and parts, is *alone* our Guide as to the *kind* and *least* amount of instrumental and manipulative proceedings.

This Principle is also our Guide in Surgical manipulations, apart from Operations, and in the application of Surgical Apparatus.

Such, then, are the fundamental Principles or Institutes of Surgery, and of Medicine, unitedly. More may in time be added, by the progress of Pathology, applied thereto. Unlike the abstract and conclusive reasoning of mathematics, the scene of all Physical Science is ever-changing and progressive. Viewing the onward moving diorama which it presents, a point distant in the horizon to-day, becomes our starting-post to-morrow; but, established as these Principles are upon so extensive an analysis of Pathology, they cannot, I trust, pass away with time; and those which may yet be discovered thereby, will prove to be subordinate and illustrative generalizations.

Pathology may determine the question of any remedial interference, and furthermore suggest the kind or purpose, as well as the least amount of such assistance as may be necessary—whether medical, operative, or both; but the *data* of Pathology cannot enable us to *predicate* the results of any Therapeutic measures. These results must be determined by *independent observation.*

What knowledge of the effects and uses of remedial measures could be deduced from our most exact knowledge of the earliest alterations of structure, the constitutional causes, the course and tendency; together constituting the pathology of scrofulous caries? We could not thereby have foretold the beneficial effects of iron, bark, and cod-liver oil in this disease. Pathology might indeed indirectly suggest the (rational) medical treatment of scrofulous caries, by virtue of its known analogy to alterations of structure, &c., which occur in other parts of the body; but such knowledge must itself have been acquired by *prior* observation of the results of therapeutic measures, and is therefore not due to any *à priori* reasoning from the nature of the disease itself. In like manner, Pathology might suggest the surgical operation of removing the articular ends of the tibia and femur—forming the knee-joint—in the case of advanced "ulceration" of their cartilaginous coverings; and this surgical proceeding may fulfil the kind, no less than the least amount, of operative inter-

ference which may be necessary under these circumstances; but the results of such assistance can only be determined by observations made *independently* of Pathology, and not by the most exact knowledge of the nature of the disease or injury in question.

We may therefore conclude, that the value, absolute and comparative, of Therapeutic measures—whether medical or operative—cannot be predicated by the most exact knowledge of Pathology, but can alone be determined and estimated by independent observations of their results.

But why by *Statistical* results? For three reasons. The accuracy of the results (of observation) in question presupposes *accuracy* of the *observations* themselves, and the *identity* of the diseases or injuries severally, respecting which the efficacy of different modes of Therapeutic treatment is compared; and furthermore, the identity also of medicines, or of surgical operations, employed.

Statistics, or the Numerical Method of counting our data, one by one, and weighing their similarity (or difference), necessitates due consideration of the accuracy of each observation, and of the fitness, by identity, of each, as an item of that aggregate wherefrom we draw a general conclusion. The results of this more exact method therefore, rather than those derived from the impressions only, which constitute 'experience,' are proportionately more trustworthy.

Then again, the *number* of observations is a very important consideration. Assuming the accuracy and identity of the observations themselves, their value rises in proportion to their number. This third *desideratum* is specially supplied by Statistics, as compared with the vague impressions of experience; and it is commonly recognised as the essential principle of Statistics.

Thus writes Professor Simpson of Edinburgh:—"The principle upon which the usefulness and stability of the whole doctrine of Medical Statistics rests, is a very simple one. It amounts to this: among facts, data, or unities of variable chance, such as the probabilities of death within a given time, or the probabilities of

attacks of particular diseases within a given time, or the probabilities of averting death in particular diseases by particular methods of treatment, or operation,—there is ever a mighty uncertainty as to the results, if we consider only single cases, or a small and limited number of instances; but our results approach more and more to certainty, in proportion as we deduce them from a greater and more extended number of instances,—from a larger and multiplied series of facts. There is always great uncertainty and instability in regard to the results of single or isolated cases; but a proper aggregation and conjunction of cases affords results which are comparatively certain and stable."*

Dr. Laycock alludes to the other characteristics of Statistics, which I have specified. "Experience and the empirical knowledge flowing from it, are alike due to observation of multitudinous facts; but the facts from which the principles are deduced are not specifically stated in detail. They are therefore not estimated numerically, nor formally collated in their various bearings on each other. But the Numerical Method is that by which facts and observations are thus formally estimated and collated, or compared, with a view to more definite and accurate conclusions. Experience tells us that a certain event is generally to be expected to occur under certain circumstances; the numerical method tells us how often it is to be expected. Experience tells us that a certain event will be generally followed by another; the numerical method shows how often the sequence takes place. Induction from the facts of experience indicates (and, it cannot be denied, fallaciously, in many instances) the causal relations of things; the numerical method examines and collates more precisely the facts and observations upon which the induction is founded, and states the results numerically. The Numerical Method is, in short, none other than that method by which experience and induction are rendered as accurate as possible.

* Edin. Monthly Journal of Medical Science, November, 1847.

When, therefore, we speak of the numerical method in reference to Medical Science, we only speak, in truth, of a more strict and more systematic method of observation and induction than the method of common experience."*

Dr. Guy† defines Statistics to be the science which prescribes rules for the bringing together of scattered observations, arranging them in classes, testing their sufficiency in point of number, and deducing from them, when so arranged, average and extreme results, fitted by their very condensation to be standards of comparison and data for reasoning. In further explanation, this author subsequently adds, that as averages founded upon large numbers of facts are numerical expressions of true probabilities; so extreme values are expressions, in the same precise language, of possibilities.

It becomes necessary to clearly understand the real significance of 'Arithmetic mean values,' and as compared with other mean values. This, the foundation of Statistics, and the various bearings of Arithmetic Means, are so ably and intelligibly explained by Professor Radicke, of Bonn, in a recent essay,‡ that I shall largely avail myself of its substance. The process of investigation *usually* employed in Therapeutic and Hygienic inquiries—*e. g.*, respecting the influence of any given agency, upon the metamorphosis of tissue —is preliminarily adverted to.

An individual is subjected, during a certain number of days continuously, to the influence of the agency in question, with the observation of a manner of life in other respects as uniform as possible; and the daily excretions—more especially, and often exceptionally the urine—are determined both with reference to their aggregate amount and to the quantities of their most ordinary chemical elements. The *data* obtained are then compared with

---

* Lectures on the Principles and Methods of Medical Observation and Research, 1856, Lect. vi.

† Cyclop. Anat. and Physiology, 1852, art. Statistics.

‡ Wunderlich's Archiv für Physiologische Heilkunde. New Series, vol. ii. part 2, 1858.—Importance and Value of Arithmetic Means, &c. Trans. for New Syd. Soc., 1861, by F. T. Bond, M.D.

those of a second series of days, which may precede or follow the first, and in which the same mode of life is observed, excepting the *absence* of the agency under investigation. The comparison is, however, usually limited to placing the Arithmetic Means obtained from the numbers of the first series of days, by the side of those deduced from the second series, and to concluding, in accordance with the preponderance of the first Means over the second, or *vice versâ*, that the agency in question increases or diminishes the excreta under observation. The urinary quantities and the chemical elements are generally determined and expressed by decimals, and a difference in the last one or two figures of these is considered amply sufficient for the basis of any conclusion.

The defect of this method of proceeding lies in the fact, that those who employ it, not clearly comprehending the real significance and value of Arithmetic Means, have attributed to them a value which in such applications as these they do not actually possess. It is therefore necessary to understand the *different* acceptations in which the term Arithmetic Mean may be received, according to the *variety* of case to which it may be applied.

Of Mean Values in general.—The term Mean Value is generally understood to signify the Arithmetic Mean exclusively; but in mathematics this term is employed in a wider sense. *That* one of any series of numbers is called their mean value, which, lying between the greatest and least of those numbers, stands in a definite dependence upon the whole series. We may, therefore, employ several kinds as means, accordingly as we find it desirable to exhibit this dependence in one way or another; and the special nature of each case must determine which kind of mean shall be employed. Those most frequently met with are the following :—

The Arithmetic Mean.—The relation of this mean to its series of numbers is such, that the sum of the differences of those numbers which are greater, is exactly equal to the sum of those which are less than it. The Arithmetic Mean of the series 3, 6, 9, 4, 13, for instance, is 7 ; the numbers greater than 7 (*i.e.*, 9 and 13) differ from it respectively by 2 and 6 ; those less than

7 (*i.e.*, 3, 6, and 4), on the other hand, respectively differ from it by 4, 1, and 3; and the sums of the two series of differences—viz., 2+6, and 4+1+3—are equal in value. If we assume that the differences of the greater numbers are negative, then the algebraic sum of *all* the differences (in this case $-2-6+4+1+3)=0$.

The Geometric Mean.—If n represents the number of figures in the series, the Geometric Mean represents the n^{th} root of the product of these figures. The Geometric Mean, for instance, of 3 and 12, $=\sqrt{3\times12}=\sqrt{36}=6$; whilst the Arithmetic Mean of the same numbers is $7\frac{1}{2}$. The Geometric Mean also of 3, 9, and 8, $=\sqrt[3]{3\times9\times8}=\sqrt[3]{216}=6$; whilst the Arithmetic Mean of those numbers is $6\frac{2}{3}$. The reason why the result always actually turns out a mean number, and why this number also differs but slightly from the Arithmetic Mean, is, that the extraction of the root is an operation which is antagonistic to repeated multiplication, and therefore the alteration produced by multiplication is to a certain extent neutralized by it. The second of the above Means, for instance, $\sqrt[3]{3\times9\times8}$, is obviously greater than $\sqrt[3]{3\times3\times3}$ (*i.e.*, greater than 3), but is less than $\sqrt[3]{9\times9\times9}$ (*i.e.*, less than 9), and consequently lies between 3 and 9 as extremes. If we substitute 9 for the third of these numbers,—viz., 8,—we get $\sqrt[3]{3\times9\times9}$, and therefore a number which is nearer to $\sqrt[3]{9\times9\times9}$ than to $\sqrt[3]{3\times3\times3}$ (*i.e.*, nearer to 9 than to 3). And if we substitute for 8 the numbers which descend successively from 9 to 3, we get numbers (viz., $\sqrt[3]{3\times9\times9}$, $\sqrt[3]{3\times9\times8}$, $\sqrt[3]{3\times9\times7}$, &c., &c.) which diverge gradually more and more, as in the case of the Arithmetic Mean, from 9, and approach nearer and nearer to 3; so that the Geometric can never differ greatly from the Arithmetic Mean.

The Harmonic Mean.—In order to define this Mean as clearly and succinctly as possible, it is necessary to remember that the *reciprocal value* of any number represents that figure which is obtained when we make the number in question the divisor of unity. Consequently, as an illustration, the reciprocal value of

$9=\frac{1}{9}$, of $\frac{4}{5}=\frac{1}{\frac{5}{4}}=\frac{5}{4}$; hence the reciprocal value of a fraction is that fraction inverted. With this explanation, the Harmonic Mean may be defined as the *reciprocal value of the Arithmetic Mean of the reciprocal values of the given numbers.*

According to this definition, we obtain the Harmonic Mean of 3 and 12, for instance, if we take the Arithmetic Mean of $\frac{1}{3}$ and $\frac{1}{12}$ $\left(\text{viz.}, \frac{\frac{1}{3}+\frac{1}{12}}{2}=\frac{5}{24}\right)$, and then find its reciprocal value (viz., $\frac{24}{5}=4\frac{4}{5}$). The Harmonic Mean of 3 and 12, therefore, is $4\frac{4}{5}$. In the same way, to obtain the Harmonic Mean of 3, 9, and 8, we take the Arithmetic Mean of $\frac{1}{3}$, $\frac{1}{9}$, and $\frac{1}{8}$ $\left(\frac{=\frac{1}{3}+\frac{1}{9}+\frac{1}{8}}{3}\right.$ $\left.=\frac{41}{216}\right)$ and find its reciprocal value—viz., $\left(\frac{3}{\frac{1}{3}+\frac{1}{9}+\frac{1}{8}}=\frac{216}{41}=5\,\frac{1}{4}\,\frac{1}{1}\right)$. This process always arrives at a mean number, for the *reciprocal value of the reciprocal value* of any number is the number itself again; thus the reciprocal value of 8 is $\frac{1}{8}$, of which the reciprocal value is again $\frac{8}{1}$ or 8. The changes which the numbers operated on suffer in obtaining their Harmonic Mean, through the process of taking their reciprocal value, are again to a certain extent neutralized by the repetition of the same process; or,—to express it more definitively,—when we take the Arithmetic Mean of the reciprocal values of the given numbers, we obtain the reciprocal value of a mean number; and when we again take the reciprocal value of this latter, we obtain the mean number itself. Thus, if we employ the second of the above illustrations as an example, it is evident that $\frac{1}{3}+\frac{1}{9}+\frac{1}{8}$ is intermediate to $\frac{1}{3}+\frac{1}{3}+\frac{1}{3}$ and $\frac{1}{9}+\frac{1}{9}+\frac{1}{9}$, and consequently that the Harmonic Mean $\frac{3}{\frac{1}{3}+\frac{1}{9}+\frac{1}{8}}$ must fall between $\frac{3}{\frac{1}{3}+\frac{1}{3}+\frac{1}{3}}=\frac{1}{\frac{1}{3}}$, and $\frac{3}{\frac{1}{9}+\frac{1}{9}+\frac{1}{9}}=\frac{1}{\frac{1}{9}}$, *i.e.*, between 3 and 9.

Moreover, it will be evident that when (as was done previously in explaining the Geometric Mean) we leave the first two of the three numbers, 3, 9, and 8, unaltered, whilst we substitute for the

last a value which descends gradually from 9 to 3, the result must also approximate by degrees from 9 to 3, so that the Harmonic Mean must pass through the same course as the Arithmetic Mean does, when treated in a similar manner.

The Quadratic Mean—so named by Radicke—is equivalent to *the square root of the Arithmetic Mean of the squares of the given numbers.* Thus, for instance, the Quadratic Mean of 3 and 12 =the square root of the Arithmetic Mean of 3^2 and 12^2, *i.e.*, of $\frac{9+144}{2}$, or of $\frac{153}{2}$, or of 76.5, and its value is consequently $\sqrt{76.5}=8.75$. In the same way, the Quadratic Mean of 3, 9, and 8, $=\sqrt{\frac{9+81+64}{3}}=\sqrt{51.33}=7.16$. The result is on each occasion a mean number, because the alterations which the given numbers suffer at the commencement, in the process of squaring, are to a certain extent compensated in the extraction of the square root which follows: or, to speak more exactly, the Arithmetic Mean of the squares of the given numbers is obviously equal to the square of a mean number, and the square root of this is consequently the mean number itself. For example, the Quadratic Mean of 3, 9, and 8, viz., $\frac{\sqrt{3^2+9^2+8^2}}{3}$, is obviously less than $\frac{\sqrt{9^2+9^2+9^2}}{3}$, and greater than $\frac{\sqrt{3^2+3^2+3^2}}{3}$, and consequently is intermediate to the extremes of the numbers in question, (since $\frac{\sqrt{9^2+9^2+9^2}}{3}=\sqrt{9^2}=9$, and $\frac{\sqrt{3^2+3^2+3^2}}{3}=\sqrt{3^2}=3$). Moreover, it must be remarked, as in the case of the Geometric Mean, that if the two extreme numbers are left unaltered, the result will be nearer to 9 or 3, in proportion to the greater or lesser amount of the third number.

It can be exactly and mathematically demonstrated that the Quadratic always somewhat exceeds the Arithmetic Mean, and that, to an amount proportionate to the inequality of the given numbers.

The more equal the numbers, the nearer does the Quadratic approach to the Arithmetic Mean; and when they are quite

equal, it exactly coincides with it. This character, however, is not peculiar to the Quadratic Mean, but possessed also by the Geometric and Harmonic Means, and it occurs, as a necessary sequence of the general idea of a Mean, with all kinds of Means. The Mean of the numbers 9, 9, 9, &c., is 9, of whatever kind it may be.

The Arithmetic Mean may represent either (1) a pure average, or (2) the probable value of a definite and *fixed* quantity, or (3) the probable value of a *variable* quantity estimated in its mean condition.

The Arithmetic Mean as a pure Average.—A pure average is found when the numbers from which it is deduced are regarded as exact, and are either independent of one another, or their dependence, if such exist, is not taken into consideration.

This is illustrated by the quantities of urine found in two series of researches by Boecker,* on the action of Sarsaparilla. He gave to an individual, whose diet was in other respects exactly estimated, a certain quantity of decoction of Sarsaparilla daily, and found that the quantities of urine passed were, in cubic centimètres, as follows :—

1467	1744	1665	1220	1161	1369
1675	2129	887	1643	934	2093

During a second series of twelve days the individual experimented on took, with the same dietary, instead of the decoction, an equal quantity of distilled water, when the quantities of urine were as follows :—

1263	1740	1538	1526	1387	1422
1754	1320	1809	2139	1574	1114

It is assumed that these numbers accurately represent the quantities of urine excreted, so that the Means of the two series may be taken as representing simple averages. Are we, then, to conclude from these numbers that the Sarsaparilla diminished the quantity of urine? Obviously not, for it will be readily seen that

* Reil's Journal für Pharmacodynamik und Toxicologie, vol. ii., parts 1 and 2.

if we were to stop at the eighth day in both series, the Means would then be respectively 1554 and 1494, and we might therefore, with equal accuracy, conclude that the urine had been increased by the Sarsaparilla. The difficulty involved by so doing is not removed by assuming that the data of twelve observations are more certain than those of eight, for it might easily happen that the thirteenth altered the relations of the two once more. In fact, if the thirteenth day in both series were to exhibit the same quantities as the twelfth—which would certainly be not at all impossible—the Means would then be respectively 1545 and 1515, and would, therefore, once more indicate an augmentation of the urine. Where, then, are we to seek for the error of such an inference?

If the quantity of urine excreted depended solely upon the nutritive or medicinal agents ingested, the series of observations would, on the supposition that the ingested matters were daily equal, necessarily give uniform numbers, with the exception of unimportant differences that might be attributed to imperfections of measurement. Practically, however, this is never the case, even in investigations where the greatest trouble has been taken to ensure equality in the measurements; and so far as the case under consideration is concerned, the irregular and excessive inequalities in the numbers obtained stand in no sort of relation to those of the quantities of food consumed. Other influences, therefore, must operate upon the amount of urinary excretion. Amongst those affecting the metamorphosis of tissue, we may enumerate, with more or less probability—atmospheric agencies (the temperature, moisture, and pressure of the air), bodily or mental occupations, idiosyncracies, the frequent and sometimes rapid fluctuations in the weight of the body, &c. Consequently, when the first eight numbers of series *a* give a greater Mean, and the whole twelve numbers a smaller one, than the corresponding number of series *b*, the fair inference is, that the sum of all the influences which affected the metamorphosis of tissue during the first eight days of the series *a* (amongst which, in addition to those

referred to, there were probably many others, and possibly that of the Sarsaparilla), *exceeded* the sum of those which affected it in the corresponding eight days of the series *b*. On the other hand, the sum of the influences which operated during the whole of the twelve days in series *a* was *less* than that of those acting during the corresponding days of series *b*. It is *possible* that the Sarsaparilla may have exercised some influence amongst the other agencies at work; but whether it really did or not, and whether in the way of augmentation or diminution, the numbers do not enable us to *determine*. They show only that its influence, if present at all, was greatly exceeded and concealed by that of agencies which were extrinsic to the dietary.

Therefore, in drawing a conclusion of a *medicinal* character from the numbers in question, its terms would be somewhat as follows :—Sarsaparilla may, or may not, affect the quantity of the urine; but so far as any *medicinal* value it may have is concerned, it cannot be from its influence on the urine, since that is so much inferior to the influence of other agencies to which we are every day exposed. This, in fact, agrees pretty well with the interpretation which Boecker himself has put upon his investigations.

The Arithmetic Mean as the probable value of a definite fixed quantity.—When we wish to find, either directly or indirectly, by measurements, a *single* quantity of *definite* value, and the different measurements which have been taken by the same or by different methods give—in consequence of the imperfection of the instruments or methods employed—different results; the arithmetic mean value of the numbers obtained is, under the circumstances, the probable value of the quantity of which we are in search; or in other words, it (the Arithmetic Mean) has, under the circumstances, a greater claim to be regarded as the *real* value than any of the numbers obtained.

The proof of this proposition is based upon the generally-received law,—that in the frequent occurrence of any two conditions, with reference to which there is no reason why the one

should occur more frequently than the other, the frequency of occurrence of the one becomes more and more equal to that of the other, in proportion as the number of instances to be compared increases.

More correctly;—the conjectural reliability of the mean value depends upon *two* conditions: (1) that the number of measurements is itself sufficiently great; and (2) that in each of the different measurements, the error in one direction does not probably exceed that in the other.

The measurements of *quantitative* chemical analysis are singularly liable to inaccuracy. For the quantitative determination of any one element of a substance, in addition to repeated weighings, numerous operations are often necessary, from none of which can special and unavoidable errors be excluded; so that the inaccuracy of a single determination, representing the sum of various individual errors, sometimes reaches a high percentage. Therefore, in comparing the chemical composition of two different substances, it is requisite that our measurements should be *repeated* in proportion as the difference in composition of the substances in question approaches the limits of possible error.

Estimation of the accuracy of the Mean as indicating the probable value of a fixed quantity.—If the two conditions above mentioned—under which the Arithmetic Means exhibit the probable value of the quantities to be determined by them—were always fulfilled, the numbers they give might then be treated as absolutely accurate. But as this is never completely the case, a standard is requisite, especially when we compare several Means with one another, by reference to which their reliability may be estimated. Professor Radicke here proceeds to indicate some of the methods whereby this standard may be discovered:—by the "Method of Successive Means," "Estimation of the accuracy of observations by the aid of the *Mean Error*," and by that of the *Probable Error*.

The Arithmetic Mean as the probable value of a variable quantity determined under Mean conditions.—For example;—where the

mean is taken from a daily series of observations of the amount of urine excreted by an individual; or of the quantities of different substances which the urine may contain.

In order that the Arithmetic Mean may represent the probable value, certain conditions already specified must be fulfilled —*i.e.*, the individual quantities of each day must be obtained as the Means of observations—*repeated* as often as is practicable—indeed, of observations repeated with a frequency proportionate to the *inaccuracy* of the *methods of measurement*. The chemical analysis especially should be performed over and over again. In the determination of the urinary quantities, a repetition of the measurements is as inapplicable (unless, in addition to the volumetrical determinations, the check of determinations by weight be employed, as was done by Boecker) as unnecessary, since the errors of measurement, when contrasted with the other irregularities, are altogether unimportant. Those arising from the greater or less completeness with which the bladder may be emptied, must be placed to the score of varying influences, and of the accompanying physical conditions on each occasion, so that their equalization may find account in the eventual increase of the day's observation.

Comparison of the Means of several complete series of observations of variable quantities.—Suppose that two series of observations, of a nature analogous to those described under the last head, are instituted on the same individual, during one of which he is exposed to a given agency, whose influence on the excretions we wish to ascertain. The question to be determined is, if, and to what extent, the Arithmetic Means of the two series, which exhibit an approximate value to the averages, can give any conclusion on this point.

In order to allow us, in the case under consideration, to come to the conclusion that the excretions have been increased, the difference between the Means must be greater than the double of the largest possible error of observation, *i.e.* greater than the sum of the uncertainties of these Means.

The determinations hitherto considered have been by *complete* series of observations, *i.e.* when the observations extend far enough to allow the inequalities in the Arithmetic Means to compensate one another, which may be recognised by the limits within which the Successive Means fluctuate, commencing notably to approach one another. In practice, however, as a rule, this condition is not fulfilled. Therefore, how to deal with *incomplete* series of observations becomes an important question, and is fully considered by Professor Radicke:—1. In cases where the disturbing agency operates all at once. 2. Where the disturbing agencies are periodic, a single one only being in operation, or a second one intervening. 3. The case, of most common occurrence, where, in addition to periodically-disturbing agencies, irregular disturbing causes also come into operation.

In reviewing the conclusions drawn from the preceding Observations, and framing Rules for the institution and conduct of new Investigations; the question, how long series of observations should be, in order to justify our drawing an independent conclusion from them, cannot at present be well answered; indeed, *an absolute answer is impossible,* since the degree of uniformity of the manner of life, the concurrence of certain unavoidable causes of disturbance, the physical condition of the individual subjected to examination, and in an inferior degree the accuracy of the observations, will cause differences. In fact, there is not a single investigation of sufficiently protracted duration to determine even for a single case the point where the compensation of the aggregate fluctuations has reached an evident stage. The more complete series have hitherto seldom embraced more than twelve to fifteen days, and must in every case be regarded still as incomplete.

It is, therefore, necessary to know how to calculate from incomplete series of observations, so as to avoid as much as possible the contingency of errors. But the rules for computation, as adapted to different circumstances, can be understood only by reference to the whole of Professor Radicke's Essay. In like

manner, the rules for the employment of Quadratics to determine Mean Fluctuations.

I shall conclude this general exposition of the Statistical or Numerical Method of Investigation, and its application to Therapeutics, by enumerating some of the *general results* or principles already established by Statistics, in relation to Medicine; and certain *objects* of *inquiry* also, yet to be attained only by this method of investigation. They are respectively embodied in a series of propositions by Professor Simpson.*

1. The absolute number of deaths from all causes, in a given time, in a given population, is always nearly the same.

2. The absolute number of deaths from individual diseases and specific causes, in a given time, in a given population, is always nearly the same.

3. The absolute number of those that recover should, *cæteris paribus*, be as fixed as the number of those that die from individual diseases, in a given time, in a given population.

4. Statistics enable us to prove that the general mortality, the mortality in particular departments of practice, and the mortality from individual diseases, are capable of being altered by altering the attendant circumstances.

5. Statistical evidence alone enables us to ascertain correctly the effects of various minor conditions upon the Fatality of Surgical Operations—such as the influence of the age, sex, &c., of the patient; the special success of different operators, &c.

6. Statistics supply in general the only true and ultimate "measure of value" of any proposed alternative operation, or of any new practice in Surgery.

Guarded by Statistics against sources of fallacy which would otherwise endanger our conclusions, we may obtain either special or comparative results. We may thereby determine the value of certain drugs and surgical operations in certain diseases or injuries;

---

* Edin. Monthly Journal of Medical Science, November, 1847.

or we may bring into competition different Systems of Therapeutical treatment, or even different Principles—that of doing, perchance, too much Therapeutically; and of doing nothing at all, but trusting to Nature. I need not specially extend these suggestions to the Practice of Surgery, nor dwell on the value of new remedial measures, medical and operative, which may thus be discovered and established.

But the more we repose in the Restorative Power, and explore the field of its operation, the more gratefully we rely on the resources of this Power, when discovered, and the less we then meddle with its operation; the more early and exactly we measure the least amount, no less than determine the kind of our interference and assistance, when needed; the more vigilantly we then watch the effects of different medicines, surgical operations, or both, on the same disease or injury, and the more severely we test their comparative therapeutic value by independent and repeated observation of their Results, estimated by Statistics,—the more nearly shall we fulfil the Therapeutic Principles of Rational Medicine and Surgery.

THE END.

WELLCOME LIBRARY INSTITUTE

BY THE SAME AUTHOR.

Fully illustrated by Coloured Lithographs and Wood Engravings.

EVIL RESULTS OF OVER-FEEDING CATTLE—A new Pathological Inquiry, demonstrating the fallacy of the Principle of the English Prize Cattle System, and its relation to Public Hygiene.

CRITICAL REVIEWS AND LEADING ARTICLES, BRITISH AND FOREIGN.

The British and Foreign Medico-Chirurgical Review, July, 1858.

"Anxious to demonstrate that the system which is at present pursued by persons ambitious of obtaining prizes at cattle-shows for their farm produce is a vicious one, Mr. Gant has applied the test of Pathological Anatomy." . . . . . "We recommend the supporters of the Smithfield Club Cattle Shows to pay attention to the facts ably and clearly put before them by Mr. Gant, and a little reflection will show them that there is an error in pursuing the system of fattening animals intended for human food to the extent to which it is now carried; they are perpetuating an error alike disadvantageous to the producer as to the consumer."

The Sanitary Review and Journal of Public Health, July, 1858.

"Mr. Gant shows that the English system of feeding cattle is based on a vicious principle; that the present system of overfeeding leads to destruction of the muscular tissue, destroys the healthy action of the heart, and induces pathological changes, the recital of which would be sufficient to disgust the least delicate and fiercest appetite."

The Glasgow Medical Journal, July, 1858.

"We trust that Mr. Gant's valuable work will be the means of awakening the attention of the general public, and that the pernicious system which it has so ably exposed will be for ever put an end to."

Cosmos.

"M. Gant, aide-chirurgien de Royal Free Hospital à Londres, pathologiste déjà célèbre, a fait de nombreuses observations sur les animaux, bœufs, moutons, porcs engraissés et vendus dans le bazar d'exposition de Baker-street. Il a disséqué, et examiné avec le plus grand soin, au microscope, les cœurs, les poumons, les foies, et les autres viscères de ceux de ces animaux que leur excès de graisse faisait remarquer entre nous ceux qui avaient remporté les prix. Il est arrivé ainsi à des conclusions d'une importance extrême que nous analysons fidèlement."

The North American Medico-Chirurgical Review, September, 1858.

"We are glad to see that the Surgeon and Pathological Anatomist to the Royal Free Hospital has taken this matter into consideration, and we regret that he has not pursued his inquiries further, but yet must thank him for his praiseworthy zeal in awakening attention to the subject." . . . . . "Why should not our health officers supervise our meat-markets, as well as direct their attention to quarantine duties? or why should not a competent individual, well versed in pathology, be appointed as meat inspector instead of the mere apologies for men who now hold that office? We would recommend this book to the attentive perusal of the scientific farmer and breeder."

The Lancet, Leading Article, May 1, 1858.

"To Mr. Gant much praise is due for his exertions, and his essay is well worthy of perusal by the pathologist, as also by the scientific agriculturist and breeder of cattle."

The British Medical Journal, Leading Article.

"Now that benevolence and the wasteful grease pot are reinforced by the last word of science, we trust to see prize beasts 'grow small and beautifully less;' and if the exertions of Mr. Gant should lead to this result, he will deserve the thanks of the country, for it is of the utmost importance that healthy meat be supplied to a nation like ourselves, which depends for its strength upon its proper supply of animal food."

The Medical Times and Gazette, Leading Article.

"Mr. Gant has done good service to the public by exposing some of the evils of the modern system of fattening cattle."

Dublin Medical Press.

"Virtue, as the fat Falstaff says, 'is of little regard in these costermonger times.' So we fear Mr. Gant's reward will be the sense of having performed a public duty, and performed it scientifically and well."

THE IRRITABLE BLADDER: ITS CAUSES AND Curative Treatment.

LONDON:
JOHN CHURCHILL AND SONS, NEW BURLINGTON STREET.

LONDON:
SAVILL AND EDWARDS, PRINTERS, CHANDOS STREET,
COVENT GARDEN.