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A MANUAL

OF

PATHOLOGICAL ANATOMY.

BY

CARL ROKITANSKY, M.D.,

CURATOR OF THE IMPERIAL PATHOLOGICAL MUSEUM, AND PROFESSOR AT THE
UNIVERSITY OF VIENNA, ETC.

———

TRANSLATED FROM THE LAST GERMAN EDITION

BY

WILLIAM EDWARD SWAINE, M.D., CHARLES HEWITT MOORE,
EDWARD SIEVEKING, M.D., GEORGE E. DAY, M.D., F.R.S.

———

FOUR VOLUMES IN TWO.

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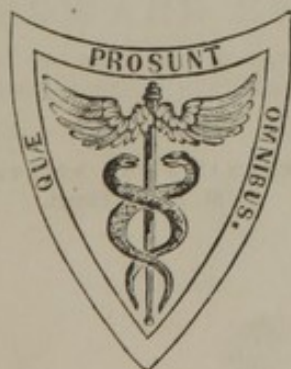
A MANUAL

OF

PATHOLOGICAL ANATOMY.

BY

CARL ROKITANSKY, M.D.,

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UNIVERSITY OF VIENNA, ETC.

VOLUME I.

GENERAL PATHOLOGICAL ANATOMY.

TRANSLATED FROM THE GERMAN,

BY

WILLIAM EDWARD SWAINE, M.D.,

FELLOW OF THE ROYAL COLLEGE OF PHYSICIANS;
PHYSICIAN EXTRAORDINARY TO H. R. H. THE DUCHESS OF KENT.

AMERICAN PUBLISHER'S NOTICE.

THE numerous unsuccessful attempts which have been made to present the following work in an English translation, sufficiently attest the very general estimation in which it is held, as well as the difficulty of the undertaking. The task having at last been executed by the united labors of four gentlemen, each well qualified for the portion intrusted to him, the American publishers take much pleasure in presenting to the profession of the United States, this great store-house of pathological knowledge, in a convenient and accessible form. The world-wide reputation of the author and of his work render eulogy superfluous, while the appearance of the translation under the auspices of the Sydenham Society is a guarantee of its fidelity. Under these circumstances, and as subsequent papers and researches of the author have been introduced in their appropriate places by the translators, it has not appeared to the publishers that additions were necessary or desirable to such a work or to such an author, and they have consequently endeavored simply to secure an accurate reprint. For greater convenience of reading and reference, and to lessen the cost, the four volumes have been bound in two, the paging, titles, &c., rendering each complete in itself.

The volumes of the English Edition were not published in their regular sequence, Volume II. being issued first, and Volume I. last. The reader is therefore referred to Dr. Sieveking's Preface to Volume II., as well as to Dr. Swaine's Preface to Volume I., for some explanation of the work, and of the manner and auspices under which it has appeared.

PHILADELPHIA, August, 1855.

EDITOR'S PREFACE

TO VOL. I.

In issuing this portion of Rokitansky's " Pathological Anatomy," it is necessary to offer, on behalf of the Council of the Sydenham Society, some apology for the delay which has attended the completion of this important and voluminous work. In his interesting preface to the second volume, Dr. Sieveking has recorded *one* reason for the order in which the volumes have been published; but he has not adverted to the main consideration by which the Council was influenced, namely, the apparently well-founded hope that they might be enabled to present the Association with the histological portion of the work *in a new and revised edition.* Encouraged from time to time in this hope by the author himself, the Council did not hesitate to defer, from year to year, the publication of the first volume, until they felt that it would be improper to tax the patience of the members any further. The new edition is still promised, but with no surer pledge for its early completion than heretofore! The editor has, however, availed himself to a considerable extent of certain papers read by the author before the Imperial Academy of Sciences at Vienna; namely, On the Structure and Growth of Cyst and of Cancers, &c. He has even found it not at all incompatible with the general unity and concordance of the work to substitute, almost bodily, the author's more recent essay on " Cyst and Alveolus," for the comparatively brief and imperfect article on the same subject in the original. These papers, there is reason to believe, contain the principal results of the author's more recent investigations, and therefore, in all probability, the most important of the additions that might be anticipated in a new edition. The Council has also sanctioned the introduction of two plates in illustration of the newly added matter.

At the conclusion of the work will be found a copious Index to the four volumes collectively. To this each editor has contributed his respective share, thus offering to the English reader facilities altogether wanting in the original work.

On the other hand the editor has felt the necessity of abridging somewhat the author's general introduction, partly because, totally unlike the general tendency of the work, it is of too "transcendental" a character either to suit the English language or to harmonize with English ideas; but more particularly because it is interwoven with a train of speculative reasoning upon the relation between power and matter, which might, in this country, very possibly give rise to misinterpretation and rebuke.

What Dr. Sieveking justly alleges of the general peculiarities of Rokitansky's style, and of the difficulty of rendering his writings intelligible in English, is, by all who are conversant with the original, admitted to apply with especial force to the first volume. Upon this ground the editor ventures to urge his claim for a fair measure of indulgence on the reader's part.

In conclusion, the editor, having been disappointed of a promised autobiographical sketch, takes leave to subjoin a few extracts from a short account of the career of this great pathologist, copied by a friendly hand from the last edition [1854] of Brockhaus's "Conversations Lexicon."

"Charles Rokitansky, the founder of the German [it should rather have been called Austrian] medico-anatomical school, was born at Königsgraetz, in Bohemia, was educated at the Gymnasium of Leitneritz, and graduated, at Vienna, in 1828. Shortly afterwards he was appointed Assistant in the pathologico-anatomical department of the University, and, in 1834, Professor of Pathological Anatomy. At the same time he was instituted Prosector at the General [united Civil and Military] Hospital at Vienna, and also sole medico-legal Anatomist for the examination of all doubtful cases of death throughout that metropolis.

"The immense fund of materials thus placed at his disposal [the number of corpses dissected by him is summed up at 30,000] was almost entirely reserved for the elaboration of that grand work on pathological anatomy, which, in the consciousness of having thoroughly mastered the subject, he gave to the world between the years 1842 and 1846; which has passed, *unaltered*, through three reimpressions; and which, under the auspices of the Sydenham Society, has been translated into the English language."

"In 1849, Rokitansky was appointed Dean of the Medical Faculty, and, in 1850, Rector of the University, of Vienna."

AUTHOR'S PREFACE.

THE appearance of this first volume brings the publication of my "Pathological Anatomy" to a close. As was the case with the earlier volumes, the completion of this one has been delayed by lack of leisure, and especially by long and repeated attacks of illness.

Whilst engaged in working out the design of this Pathological Anatomy, I have throughout endeavored to act the part of a clinical teacher; and I believe that, in so doing, I have apprehended the requirements of our day, and usefully disposed of the colossal materials within my reach.

The same self-reliance that characterized the commencement of my pathologico-anatomical studies, has stood by me whilst engaged in observing and interpreting the facts of which the said materials are composed: for, each individual discovery encouraged me more and more to pin my faith upon Nature alone. Still I have never failed to watch and to appreciate the achievements of other men.

The present work will at any rate tend to show, how thorough is my conviction that Pathological Anatomy must constitute the groundwork, not alone of all medical knowledge, but also of all medical treatment; nay, that it embraces all that medicine has to offer of positive knowledge, or at least of what is fundamental to it. Its domain will here, however, be found more extended, and more nearly approximated to the confines of Pathological Chemistry than has generally been the case in pathologico-anatomical writings.

Upon individual sections of the work I must confess to have exercised a certain favoritism; and I have striven to cultivate and to carry out some important general views, with a well-tested conviction

of their truth. Amongst these views I may here single out for exemplification the doctrine of a primitive diversity in blastemata, as the only tenable basis for a humoral pathology.

From a comparison of the antecedently published volumes on special pathological anatomy with the present one, it will be seen that the former furnish the groundwork of the views here propounded, and that my convictions, upon the whole, remain unchanged.

<div align="right">THE AUTHOR.</div>

VIENNA, July, 1846.

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PLATES I. AND II.

Figs. 1 and 4 represent proliferous cyst-formations from the cortical substance of the kidney, as a sequel to Bright's disease. The two figures, 1 and 4, illustrate well Rokitansky's history of proliferous cyst-development, and at the same time what he understands by the often-occurring expression, "alveolar type or arrangement."

In fig. 1 we have the cyst in all its phases. *a* is a simple cyst, arising out of the expansion of the elementary granule, first into the nucleus, from this into the cell, and progressively into the cyst. But it has remained barren, and contains only a diaphanous, viscid serum within a simple cyst-membrane. *b* represents a parent-cyst, the *early* history of which accords with that of the barren cyst; within it, however, new granules have formed, and gradually become developed into vesicles or cysts containing other nuclei, until the parent-cyst has become replete with them, and from being spherical, they are rendered polyhedrical by mutual compression. In an adjoining parent-cyst, many of the filial cysts have remained barren, others contain nuclei in the act of splitting. *c, c, c, c,* represent another form of development of the parent-cyst. Here, again, the parent-cyst has gone through the same phases, from the elementary granule upwards. But, as the cell dilates into the cyst, a granule forms centrally to the latter and expands into a filial cyst, centrally to which a third granule opens out in the same manner; and so on. These intra-cystic cysts in their dilatation ultimately close upon the parent-cyst, forming secondary, tertiary, and ulterior layers, to which an external, fibrous layer is generally added out of the surrounding blastema. Or this fibrous coat accrues in the *alveolar* shape. Fig. 1 affords several examples of this. It is, however, better seen in

Fig. 4.—*a* is 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 what the author designates as the "alveolar type or arrangement." It is, however, still better defined in

Fig. 2, which represents cyst-formation in a medullary carcinoma. From the carcinomatous framework a bulb-like excrescence is thrown out, within the extremity of which a parent-cyst forms and becomes replete with filial cysts, each containing a central nucleus. This parent-cyst surrounds itself with a broad marginal area of blastema, within which elongated, caudate nuclei course round the cyst in several tolerably regular circles or series—the rudiments of a dense fibrous envelope. Such is the "alveolar type," which applies to the fibrous fabric of follicle walls as well as to those of cyst-formations. (See "Cyst and Alveolus.")

Fig. 3 represents a transverse section of a colloid cancer. *a* is an older portion of densely fibrillated fibro-membranous structure. *c* is a transverse section of a more recent fibro-membranous stroma; *b*, a transverse section of the colloid warp which intertwines with the said fibro-membranous stroma. (See p. 220.)

Fig. 8 represents the multilocular, fibro-membranous stroma of colloid cancer deprived of its colloid contents. (See p. 221.)

Figs. 5, 6, and 7, represent so many stages of the development of medullary carcinoma. They are severally described in the same order in which they are here numbered, at pp. 220 and 221.

Figs. 1, 2, and 4 are magnified by 90 diameters, the five remaining figures by 400 diameters.

Several of the figures here given are embodied from Rokitansky's " Essays," in Mr. Paget's admirable " Lectures on Surgical Pathology," vol. ii.

Figs. 1, 2, and 4, are derived from Rokitansky's Essay on " Cyst and Alveolus," read before the Imperial Academy of Sciences, at Vienna, in 1849; figs. 3 and 8 from his Essay on " Colloid Cancer," published in 1852; figs. 5, 6, and 7, from a thesis of his on " Cancer-stromata," also published in 1852.
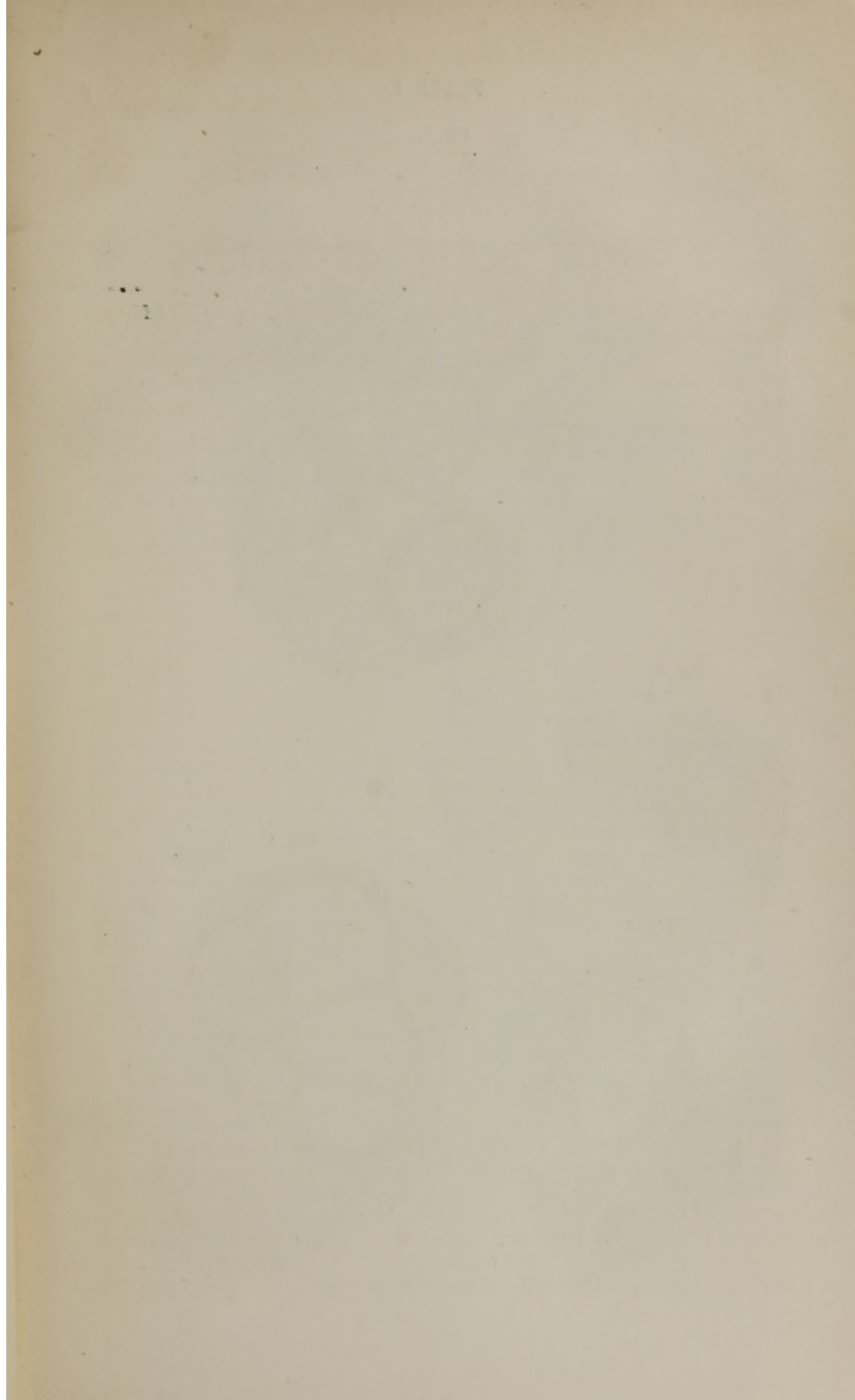
PLATE I.

Fig. 1.
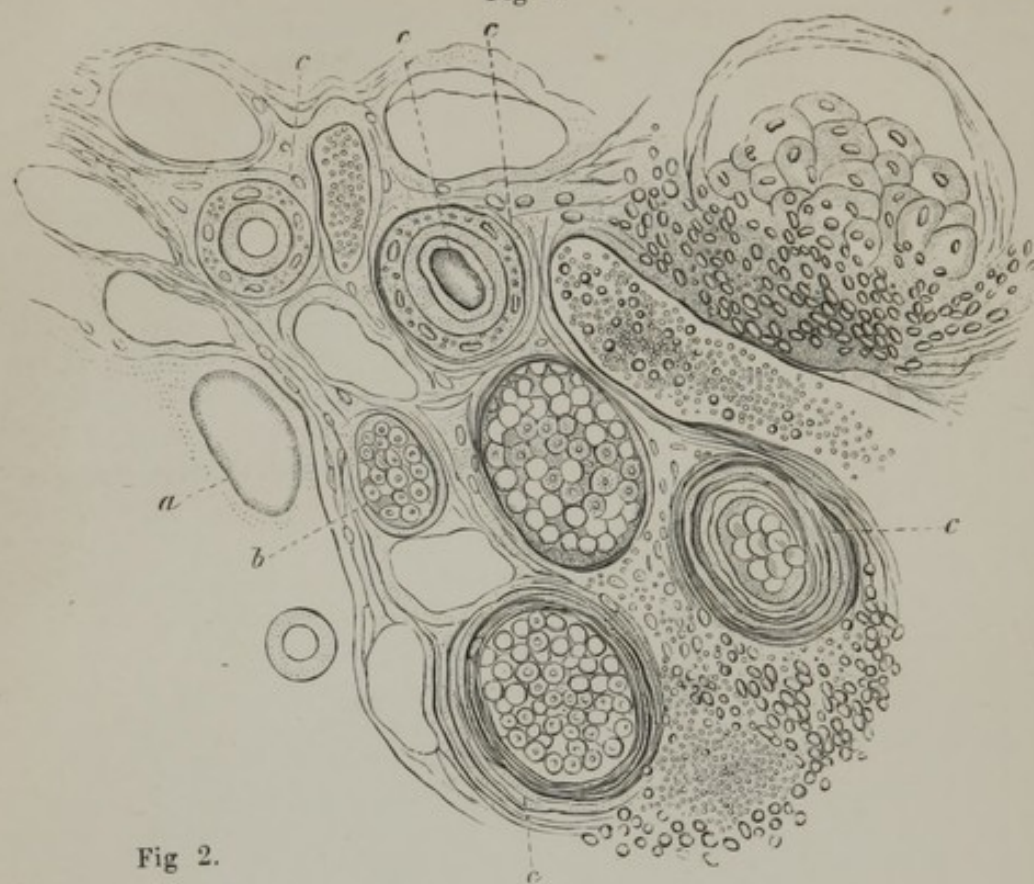
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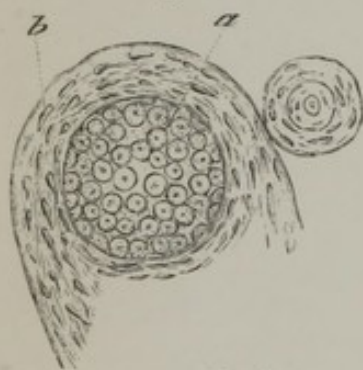
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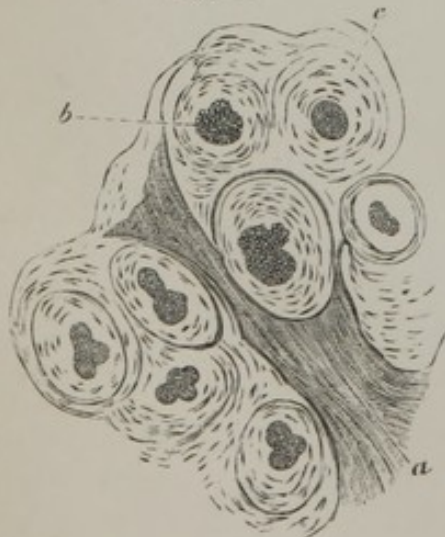
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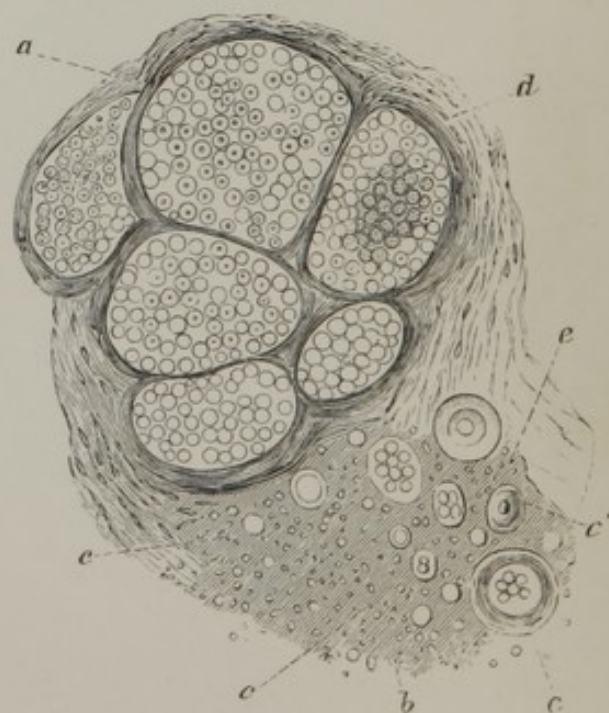
PLATE II.

Fig. 5.
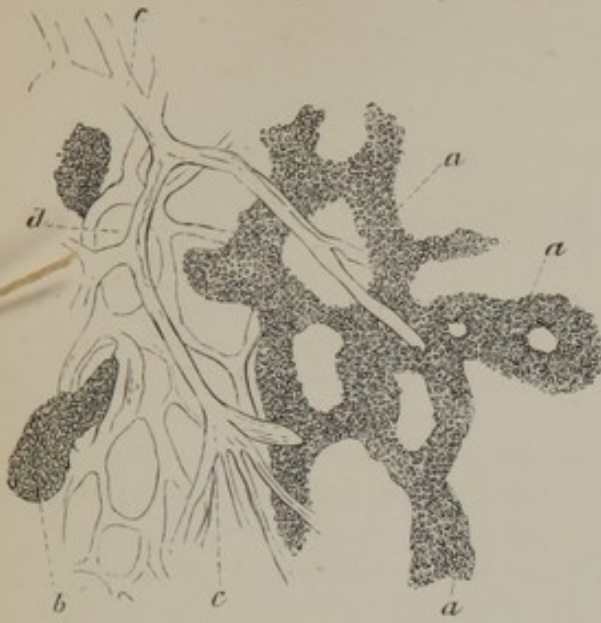
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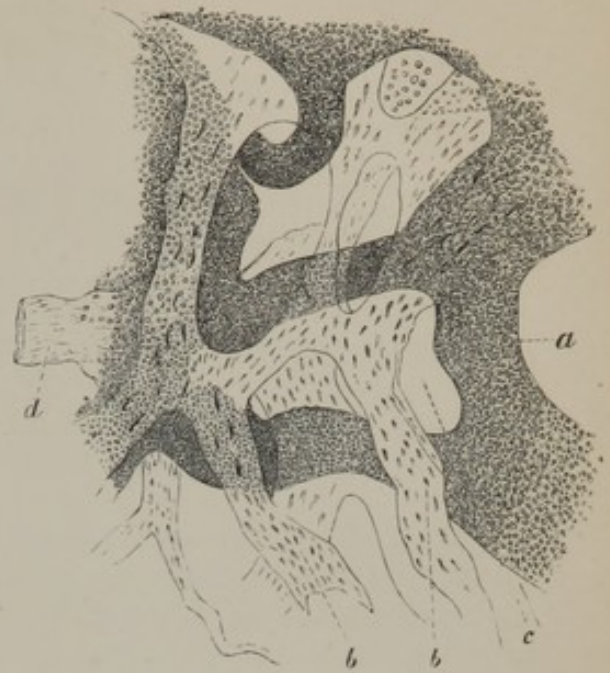
Fig. 7.

Fig. 8.
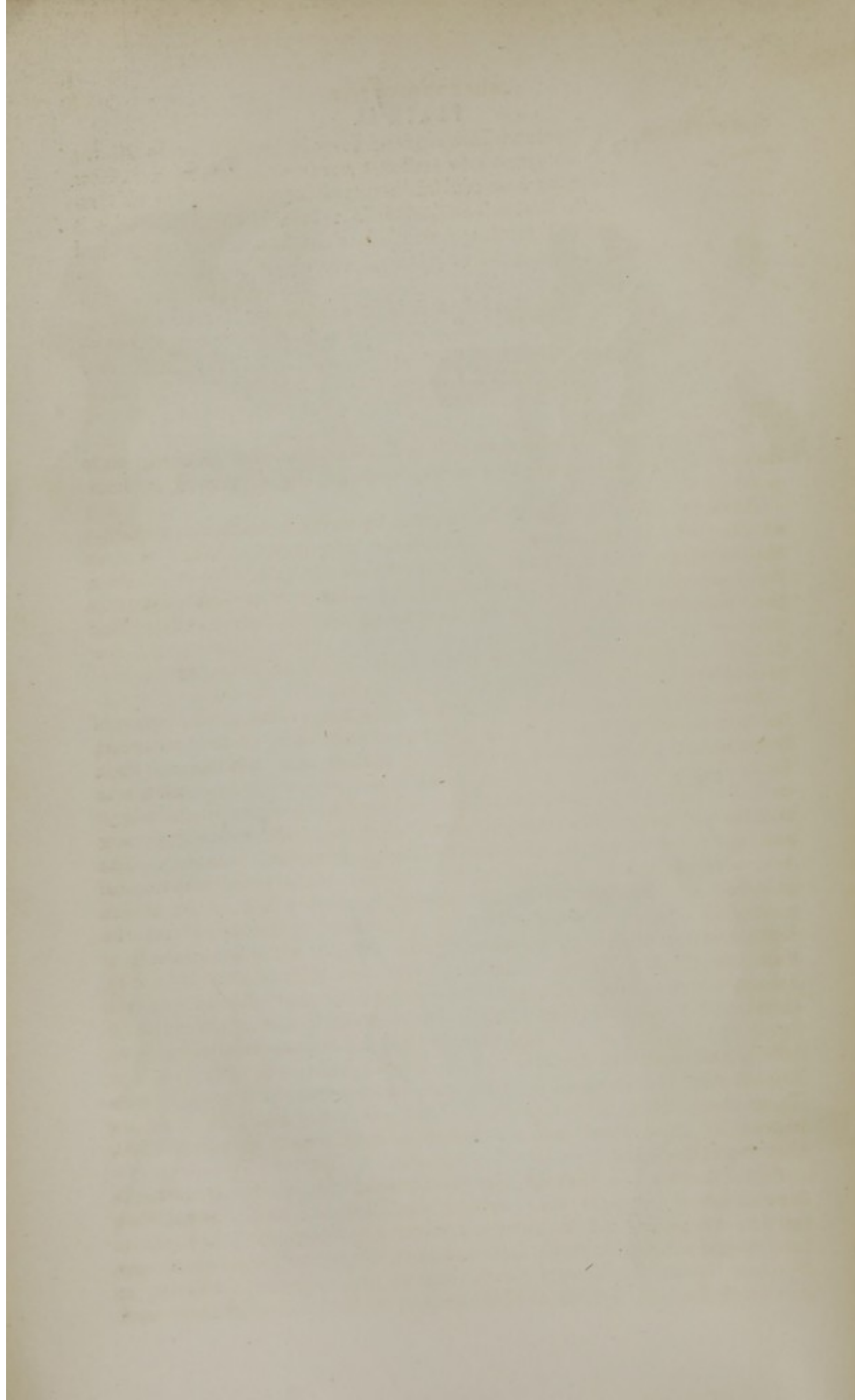
INTRODUCTION.

PATHOLOGICAL ANATOMY may be said to be a modern science. It is indeed only of late years that it has assumed the dignity of an independent science at all.

Although, according to Pliny, dead bodies were examined in Egypt at the time of the Pharaohs, that is to say, many centuries before Galen, with a view to detect the seats of disease; the result of those researches has remained unrevealed to us. Even upon Greek medicine the pathologico-anatomical observations made by its founders and scholars have been without material influence. They were indeed gradually lost sight of in the medical schools, which arose out of the successive systems of philosophy of a later period.

Not until the commencement of the sixteenth century—the period of the regeneration of anatomy—does the epoch begin of an occasional, fragmentary, indeterminate study of pathological anatomy. Still, Eustachius, the rival of Vesalius, must have been deeply impressed with its importance; for, towards the close of his life he expresses his regret that he had not rather bestowed upon pathological anatomy that time and attention which he had devoted to physiological anatomy. The first who dedicated himself in an especial manner to pathological anatomy was Antony Benivieni, who wrote, at Florence, "De abditis morborum causis" (1507). He was followed by Mathieu-Reald Columbus, the protector of Vesalius (1590), Volcher Coiter, a disciple of Fallopius (1573), Salius Diversus (1584), Marcellus Donatus (1588). Johannes Schenkins collected the observations made up to his time (1584). Johannes Wierus (1569), Felix Plater (1614), Fabricius Hildanus (1606), Tulpius (1672), Vesling (1664), Thomas Bartholin (1654–1675), Stalpaart van der Wiel (1677), Daniel Sennert (1676), Friedrich Ruysch (1691), cultivated pathological anatomy after their own fashion. Their observations, although partially of great interest, often bear the impress of superstition, and are disfigured by the fanciful way in which they are interpreted.

Since the time of Harvey, the discoverer of the circulation, who, in denominating our particular science, *medical anatomy*, showed how fully he comprehended its import, various physicians have worked out sundry branches of pathology anatomically. Amongst them are Thomas Willis (1677) and J. J. Wepfer (1658–1727). Others, as Fernel (1679), F. Sylvius (1734), Baillou (1735), have, in their com-

pendia of pathology, adopted pathological anatomy for their ground-work. Bonnet was, however, the first who compiled an ample repertory on this subject ("Sepulchretum," 1679); and even this work unites to the imperfections of earlier observations the lack of a standard physiological principle, and of a definite practical tendency. The same applies equally, if not more forcibly, to Blankaard's "Anatomia practica" (1688).

Above both these—above all that had been previously accomplished —stands pre-eminent, Morgagni and his work, "De sedibus et causis morborum" (1767). Notwithstanding its defects, this book remains a model of industry and perseverance, of method and arrangement, of breadth and perspicuity, and, lastly, of originality, for all time.

In the same century, special investigations, not unworthy of record, were made by J. Moritz Hofman, Walter, Albinus, Vater, Levret, W. Hunter, Senac, Meckel, Böhmer, Van Doeweren, Camper, Bleuland, and others.

In a work containing a vast number of facts ("Historia anatomico-medica," 1768), the purpose attained by Morgagni, failed in Lieutaud's hands, through lack of detail, of analysis, of a practical generalization of facts. On the other hand, Sandifort ("Observ. anat. path., 1777) merits, for the richness and solidity of his writings, to be classed along with Morgagni.

The compendia published in 1785, by C. T. Ludwig, and in 1796, by Conradi, and even the greater work of Voigtl (1804), so marked by literary industry and so serviceable withal, have not advanced science, either by aptness of discrimination, by a judicious selection of matter, nor yet by any remarkable progress in the method of anatomical research.

Mathew Baillie's anatomy of morbid structures (translated into German by Soemmering, in 1794) is distinguished by greater depth of research into the fabric of organs, and both by its generalizing tendency and its physiological character. These latter qualities are, however, still more decidedly impressed upon the aphorisms from pathological anatomy published at Vienna, by Velter, in 1805.

The most decided impulse was given to a right conception and application of pathological anatomy by Bichat in his general anatomy. Bichat founded upon the latter an especial physiology, or rather, blended the two. Pathologists, imitating this, endeavored to reconstruct their science upon an anatomical basis.

France was the country in which this attempt was made in the most effectual manner; not that it was exactly the cradle of pathological anatomy, but that it was the land of all others, in which men sought and found in it a solid foundation for medical knowledge. Such men were, amongst others, Bayle, Corvisart, Laennec, Dupuytren, Broussais, Cruveilhier, Rochoux, Lallemand, Riobé, Andral, Louis, Gendrin, Bouillaud, Billard, Rayer. It is true that one of these, namely, Broussais, disseminated an error from which his pupils cannot yet disentangle themselves, an error in which Brunonianism seemed once more to be trying its strength upon novel ground. On the other side, however, Laennec invented and carried out a method which insures to

him and to his work the acknowledgment and admiration of future ages.

In England many have, up to our own day, worked in a similar spirit. Amongst these, we may mention the names of Abernethy, Charles Bell, Astley Cooper, Hodgson, Farre, Wardrop, Howship, Baron, Hodgkin, Hope.

In Italy, on the contrary, and in Germany—if we except the impulse so decisively given in the same direction by the ingenious Reil—pathological anatomy has been upon the whole less cultivated, and has exercised less influence upon medicine. Accordingly, Germany and Italy have but few men to place in parallel with those of France; few to add to the names of Scarpa, Malacarne, Paletta—of J. F. Meckel, Otto, and (in industry and method, the essentially German) Lobstein.

It was reserved for Germany, at the present day, to establish a pathological anatomy and a method of working it out, partly independent, partly framed according to the best models of France. Under the auspices of German universality and analysis, this renovated science, emancipated alike from the systems of a bygone age and from a vain eclecticism, has begun to incorporate itself with pathology in a way that promises both durability and brilliant progress, more especially in its natural alliance with German physiology, and under a consistent and rational standard of pathological chemistry.

Classification.—Just as there is a general and a special anatomy, physiology, pathology, so there must in like manner be a general and a special pathological anatomy. The former treats of general anomalies of organization, the latter of the special anomalies of individual textures and organs.

All anomalies of organization involving any anatomical change manifest themselves as deviations in the quantity or quality of organic creation, or else as a mechanical separation of continuity. They are reducible to *irregular* number [deficient or excessive formation], irregular size, form, position, connection, color, consistence, continuity, texture, and contents. They relate to the physical properties of the animal body and of its organs. The chemical properties, although not strictly pertaining to the field of anatomy, are too intimately connected with the physical, to be suffered to remain in the background at the present day. The animal fluids bear a similar relation to anatomy. Their anomalies will be taken into account, so far as it may appear needful, under the appropriate heads. Those of the sanguineous fluid will, however, demand a separate chapter. This will come in at the conclusion of the general anatomy, in which a frequent reference to them will have previously demonstrated the indispensable nature of the inquiry, as a sort of connecting link between general and special anatomy. We shall thus have to discuss, in ten separate chapters, the anomalies of organization. There are, however, a few general points which require some previous explanation.

I. The said anomalies, being simple alterations of the normal being and of its parts, appear as abnormal conditions, excluding the idea of an independent parasitic organism of disease.

II. No formation is incapable of becoming diseased in one or more ways. Several anomalies coexisting in an organ commonly stand to each other in the relation of cause and effect. Thus, deviations in texture very frequently determine deviations in size, in form,—and these again deviations in position. Deviations in position give rise to anomalies of volume and of texture.

III. Pathological anatomy, proximately concerned with anomalies of individual organs and systems—with local anomalies—has often reserved for it the task of revealing by experiment and deduction the existence of *general disease*, as also of establishing the mutual relations which exist between the two. The seat of general diseases may now be referred, almost without exception, to the blood [the fluids]. They appear, therefore, as anomalies of admixture or crasis, either primary or secondary.

IV. This demonstration of general disease is indeed a step in advance for pathological anatomy. It threatens, however, to mislead us into the error of exclusive, transcendental, all-pervading humoralism—into the error of denying all local disease, by deducing the latter in every instance from a corresponding general affection,—not but that many diseases really are but the localization of a pre-existent general disease.

V. The existence of purely local—independent of general—disease, from the simplest inflammation—from blennorrhœa, to tubercle and cancer, we look upon as grounded—

(*a.*) In the self-vitality of organs, and their independent relations to the external world.

(*b.*) In the local influence of direct or reflected stimulation. Either directly, or through the medium of the nervous system, stimuli effect a local modification in the vital processes of absorption and secretion—in the interchange of matter,—an anomalous reciprocity between bloodvessels and their contents on the one side, and the parenchyma-engendering products, abnormal both in quantity and in kind, on the other.

Normal nutrition and secretion are no doubt mainly dependent upon a normal crasis; but they are also based upon the perfection of the specific vital action proper to individual parenchymata. Anomalous secretions often arise out of influences which modify the vital action of the parenchyma, and consequently its reciprocity with the unchanged gross material, the blood: as, for example, augmented or otherwise altered secretion of milk, produced by local irritation or by anomalous innervation, the effect of mental operations. In like manner, local diseases are but a consequence of qualitative and quantitative alienation of the textures and organs,—the formative material (the blood), notwithstanding its reciprocity with the latter, not becoming sensibly contaminated.

Influences, especially of a mechanical kind, are often so strictly local, that it would be far-fetched to derive all local disorder from a general causal disease. Even the latter would be but secondary,—a mere transfer of the alienation locally produced.

The existence of local diseases is further shown—

(c.) By direct evidence, where local disease is established, of the absence of any disease of the blood crasis.

(d.) In the curableness by topical remedies—extirpation, isolation, &c.—of local diseases, without their recurrence either on the same spot or elsewhere. The cure may even involve the simultaneous removal of a general disease consequent upon the local one, this having possibly acted as an anomalous instrument for the elimination of certain elements from the blood, exhausting it of certain essential constituents.

VI. Local disease extends beyond its original seat in various ways:

1. By contiguity. The affection spreads to the immediate vicinity of its original seat. This extension is favored—

(a.) By uniformity of structure.

(b.) By intensity of disease.

(c.) By the nature of the malady. Certain diseases, such as tuberculosis and cancer, in their extension, spare no texture, whilst the typhous process upon the intestinal mucous membrane always finds an arresting formation in the sub-mucous areolar tissue.

2. The disease extends to remote formations, both similar and dissimilar. This mode of diffusion does not imply concurrent general disease, but proceeds, according to tolerably constant laws of sympathy, through the mediation of the nerves. It is greatly promoted, however, by a concurrent general disease, kindred in character with the local, causing it to increase, and multiplying the seats of the disease, as, for instance, in inflammation.

VII. The originally mere local is enhanced into a general disease of the same nature, or gives rise to one of different attributes. The former contingency may arise from the alienation of the peripheric nerves, productive of the local anomaly, being transmitted to the nervous instruments presiding over the circulation,—in other words, to the nervous centres, and in particular to the spinal cord and the ganglia. Or else the conversion assumes the substantive form of infection, the noxious matter evolved by the original local disease, or its products, being received into the circulation. This last event occurs where the products of the local disease exhaust the blood of certain ingredients, for instance, of fibrine, of albumen, of serum, of salts. To this class belong, in like manner, the anomalies occasioned by mechanical disproportion, such as the venous diathesis, cyanosis dependent upon disease of the heart or lungs, &c.

VIII. The presence of general disease may be the more safely inferred:

1. The more widely extended is the local disease over several uniform or dissimilar formations, and the greater its intensity.

2. The less the products of the local process are conformable with the character of the normal structures;

3. The less the extent and nature of the local disease, or of the structures involved, however important in the organism, suffice to account for the general appearances during life and after death;

4. The more anomalous, compared with the alienation of the solids, are the secretions and excretions; and,

5. The more the totality of the organism, in the absence of actual anatomical disturbances, seems cachectic and impaired.

6. The more marked is some anomaly in the circulating fluid, with respect to the quantity or quality of its component parts.

IX. General disease engenders in the most various organs and textures, according to their innate general or individual tendencies, either spontaneously or by dint of some overpowering outward impulse, a local affection which reflects the general disease in the peculiarity of its products. The general disease becomes localized, and, so to speak, represented, in the topical affection.

X. A general disease not unfrequently finds in its localization a perpetual focus of derivation, with seeming integrity of the organism in other respects. Recovery may, after a lengthened process, eventually take place through the exhaustion of materials at the local vent. Forced extirpation, on the contrary, or insulation of the locality, generally aggravates to a high degree the general disease, multiplying its points of localization.

XI. The disease has, even anatomically speaking, its stages of incipiency, increment, acme, and decline.

XII. The terminations of disease are, in like manner, subjects for anatomical research.

1. The issue of local disease in health consists either in the perfect re-establishment of the normal condition, or else in partial recovery; more or fewer important residua and sequelæ of the disease, not incompatible with a tolerably fair state of health, remaining entailed. Thus the previously diseased organ may have lost substance, or more or less its natural texture; or it may have suffered changes in form or in position, or interruptions of continuity.

2. The issue of one general disease in another general disease [metaschematismus] is frequent. Anatomical research proves, and chemical analysis will still more clearly demonstrate, that it is far more frequent and varied than would appear from mere clinical observation. This is taught in an especial manner in the mutual exclusion of different morbid processes, which seem to succeed each other, when in full vigor, sometimes almost by a necessary sequence. Thus dropsy may succeed to the exhaustion of fibrine and the excretion of albumen, cancer to tubercle, &c.

3. Transition by so-called metastasis often becomes the subject of the scalpel. It comprises various conditions:

(*a.*) The localization of a general disease at an unusual spot. It has the character of a vicarious or supplementary crisis. Instances are afforded in skin eruptions, and especially in the secondary typhous processes.

(*b.*) Topical processes constituting the localization of a metaschematism, with which, as in the former instance, a general disease concurs. Such metastases occur more particularly in the sequel of typhus, and in the shape of inflammation, suppuration, gangrene, in both external and

internal organs. They represent the localization of a general disease consecutive to the original typhous process.

(c.) Local processes, with the development of which the general disease is essentially abated, or thoroughly exhausted and extinguished. They are frequent, and deserve alone to be designated as *metastases,—* metastases in a restricted sense. They are either just sufficient vents for the general disease, and are only cured when the latter is subdued; or they heal spontaneously, the dyscrasis having, through their agency, become exhausted.

(d.) When, owing to whatever cause, a local disease has been checked in its development, it subsides only to reappear in another part, often with augmented force, and with the supervention of a new general disease, or the aggravation of one already existent.

4. *Issue in death.* Diseases are mortal for the most part,

(a.) Through exhaustion of power and of organic matter, tabescence, loss of fluids.

(b.) Through the suspended function of organs essential to life; through palsy; through sudden and extensive displacements; through hypertrophy, atrophy, diseases of texture.

(c.) Through vitiation of the blood and palsy of the nervous centres arising out of the conflict between contaminated blood and nervous medulla.

XIII. Where several diseases coexist in an individual, they are in part *primary*, in part *secondary* and subordinate to, although homologous with, the former. Again, they are partly sequelæ and residua of antecedent disease, as in the case of atrophy of the brain consequent upon apoplexy, upon encephalitis.

XIV. Very dissimilar anomalies may coexist in one individual, as mere local affections or complications. *Combination* or *exclusion* result only in the case of heterogeneous diseases founded in or determining a dyscrasis: for example, cancer and tubercle, organic heart disease and tuberculosis. The study of these two relations opens a rich field of promise for the furtherance of accuracy in diagnosis.

XV. The import of a disease bears a direct ratio to the worth of the organs attacked: take, for example, hypertrophy in different muscular organs.

XVI. With reference to the period during which anomalies originate, we have to distinguish *congenital*, or such as have become established during intra-uterine life, and *acquired*, or such as have arisen during extra-uterine life. The former comprehend *primitive anomalies*.

XVII. Primitive anomalies comprise *malformations*. These are deviations of the organism, or of an organ, so intimately blended with its primary development, as to occur only at the earliest periods of embryonic life, or at any rate before that of mature fœtal existence.

Malformations, when inconsiderable and harmless to the individual, are termed *lusus naturæ, variation, defect of formation, malformation;* when more marked, *deformity;* when excessive, *misbirth, monstrosity, monster.*

Despite some progress made in this field of late years, the genesis of malformation is still veiled in much obscurity. The opinions of modern physiologists on this point may be collected under two heads. According to the one section, the malformations are referable to a primitive malformation of the germ. According to the other, to various influences affecting the germ in the progress of development.

The former opinion resolves itself into that of the *ovists* and that of the *spermatists*. Those believed in the foreshadowing of the malformation in the ovum; these regarded it as dependent upon the spermatozoa as embryones.

At this day both theories are rejected as inapplicable to a vast number of malformations which unquestionably do originate during the development of the germ. Still the malformation might be founded in the nature of the ovum and of the sperma, although neither of these constitutes the embryo. The frequent recurrence of the same malformations out of the same parents, and the hereditary character of these anomalies, render this not improbable. We might further advert to the nature of certain malformations—inversions, duplicate formations, for instance—in which the fusion of two germs, and the bisection of a single germ, during their development, are neither of them quite conceivable.

The second of the aforesaid propositions embraces several hypotheses.

(*a.*) The oldest and most popular attributes the malformation to a sudden and forcible impression wrought upon the mother (Verschen). The question whether mental emotions do influence the development of the embryo must be answered in the affirmative. Instances undoubtedly have occurred of such maternal impressions—fright more particularly—when violent, giving rise to malformations. Seeing that many malformations originate in an arrest of development, and how frequently the former bear a certain resemblance to various animals, it is just conceivable that the development of the embryo may be so arrested by maternal emotions as *accidentally* to occasion a likeness between the object that produced the impression and the resulting malformation.

(*b.*) A second doctrine derives malformation from external mechanical influences, such as a blow, a thrust, a fall, &c., suffered by the mother; mechanical obstacles to the passage of the ovum through the Fallopian tubes, and to its growth in the womb; excess or deficiency of liquor amnii; restriction of space for the foetus; the formation of false membranes within the cavity of the amnion, &c. Although F. Meckel dissents from this doctrine, we are not quite prepared to relinquish it ourselves.

(*c.*) A third opinion assigns, as the cause of malformations, disease of the foetus.

Disease arrests the development of foetal formation with respect to growth, shape, position, and texture of particular organs, indirectly embarrassing the expansion of neighboring organs, or, it may be, causing their destruction. A common disease having this tendency is dropsy, as preventive of union or closure, and productive of disjunction or fissure. Inflammation and its consequences may be mentioned under the same head.

The conditions of encephalocele, of hemicephalus, of anencephalus,

and of spina bifida, being obviously due to dropsy, are beyond the pale of arrested formation. Certain anomalies of the peritoneum and of its viscera, formerly reckoned as genuine instances of arrested formation, have been shown by Simpson in certain instances probably to result from fœtal inflammation. Some malformations of the heart, especially defects in its septa, we hold to be owing to fœtal endocarditis, and to consequent coarctation of the heart's orifices.

The number of malformations, however, to which this doctrine fully applies, is as yet very small. Amongst those which do not admit of such an explanation are *duplicate formations*, and the great majority of malformations designated by the term *malposition*.

(*d.*) The fourth proposition—which the countenance given to it by Wolff, by Tiedemann, and especially by J. F. Meckel, has caused to be the more generally received one—has formed the groundwork for an elaborate scientific inquiry into the subject, in especial connection with the history of development. This sets forth that *most malformations represent certain stages of the development of the embryo and of its organs, at which stages formation has stopped short, or from which ulterior development has ceased to follow the normal type.* The *malformation* is therefore essentially an *arrest of development*.

This theory of malformations is in a great measure correct. Still it does not attempt to explain the *cause* of the arrest, which may be one of those already enumerated, be it concerned with the germ, with sickening of the embryo, with mechanical influence, or with mental emotion.

A good classification of malformations is, owing to the difficulty of establishing a principle of division generally applicable, as yet wanting. If we attempt to classify them according to external form, we meet with a barrier in their multiplicity. Another obstacle consists in this, that where several malformations coexist in the same individual, they must needs all be classified according to the one most pronounced, and the designation be therefore partially incorrect. A classification founded upon the occasional causes is impracticable, since the same malformation may originate from various causes. If we take for the basis external form and cause conjointly, the classification becomes bereft of logical unity.

It is desirable that we should become acquainted with the principles upon which the more remarkable classifications hitherto propounded are built.

Passing over the older classifications, we should except that of Buffon, as it forms the groundwork upon which almost all the latter ones are modelled. Buffon divided malformations into three classes: 1, malformation with excess; 2, with deficiency; 3, with inversion or perverse site. To this classification we may subjoin that of Blumenbach, under the following four heads: 1, fabrica aliena; 2, situs mutatus; 3, monstra per excessum; 4, monstra per defectum. These were followed by Meckel in his division of malformations, as follows: 1, malformation from deficient plastic power; 2, from excess of plastic power; 3, from deviation of the organs in respect to their natural form; 4, malformations characterized by ambiguity of sex—hermaphroditism. This dis-

tinction of hermaphroditism from other malformations constitutes the great defect of Meckel's classification.

Breschet has, in his classification, broken up Buffon's first class into two, by separating duplicate formations from malformations per excessum. The four orders of his classification are: 1, ageneses, devious formations with diminution of plastic power; 2, hypergeneses, with augmentation of plastic power; 3, diplogeneses, devious formations with the fusion of germs—duplicate formations; 4, heterogeneses, with alien character of the product of generation. The further division is as follows:

The first order, ageneses, breaks up into four species.

(*a.*) Agenesie, absence—defective development. It is either partial, as in hemicephalie, aprosopie, acephalie, apleurie; or it is general, as in microsomatie (dwarfishness, cretinism).

(*b.*) Diastematie, cleft formation at the median line. It is subdivided according as it affects the head or the trunk, into diastemencephalie, &c., and diastematosternie, &c.

(*c.*) Atresie.

(*d.*) Symphysie, coalition fusion.

The second order, *hypergenese*, presents two species, according as individual parts or the entire body are concerned. To the former species belong macrocephalie, macroprosopie, &c.; the latter consists of macrosomatie (giant growth).

The third order, *diplogenese*, is divisible into *external*, through fusion or adhesion, as in diplocephalie, diplothoracie; and *internal*, through penetration (per penetrationem).

The fourth order has three species.

(*a.*) Deviation as to site, either of the entire organism [extra-uterine pregnancy] or of individual organs [ectopie].

(*b.*) Deviation as to number, polypædie—the coexistence in the uterus of several fœtuses.

(*c.*) Deviation as to color—leucopathie, cyanopathie, cirrhopathie.

In this arrangement, the distinction of dip-ogenesis from hypergenesis is based upon the unproved doctrine of the fusion of two germs constituting duplicate formation. To the order, heterogenesis, are referred deviations which ought not to be designated as malformations.

One of the best known classifications of late years is that of the two Geoffroy St. Hilaires, father and son, who handle malformations, according to the natural method, under the term teratology (from τερας, monstrum).

Malformations are simple and *complex—anomalies simples et complexes*.

The simple—hémitéries—are either so-called variations, lusus naturæ, where the anomaly is slight, causing neither disturbance of function nor deformity, or else defects of conformation, malformations in a restricted sense, where, however trifling the anatomical deviation, they impede or preclude the exercise of one or more functions, or occasion deformity.

They are divided into five classes, the anomaly being respectively concerned with—

1. *Volume*, as regards size, both of the body generally, and of its individual parts;
2. Form;
3. Structure and coloration;
4. Disposition;
5. Number and existence, that is absence or presence of parts.

These classes, according to extent and to degree—according to the kind of malformation, are divided into *orders*, and these again distinguished according to the regions, systems, and organs involved. Thus the first class comprises the four orders—general dwarfishness and general gigantism, partial gigantism and partial dwarfishness of proportions. The fourth class includes the five orders—displacement, preternatural union, preternatural connection, sept-formation, disjunction, &c.

Complicated anomalies are classed in three subdivisions.

1. Heterotaxies (ἕτερος and ταξις). Anomalies important in an anatomical sense, but neither visible externally nor obstructive of any function. In mankind they comprise but one order, namely, *lateral* inversion of viscera (inversion splanchnique).
2. Hermaphroditism.
3. Monstrosities. Anomalies very considerable in degree, and consisting in a faulty anatomical arrangement greatly deviating from the type of the species, externally visible, and obstructive of one or more functions.

These last are divided into three classes,—into simple, double, and triple. The next division into *orders* is arranged according to physiological characters; the subdivisions then following, according to *tribes*, *families*, and *species*. Thus, simple *monstrosities* resolve themselves into three *orders:*

1. *Autosites*, in which independent, progressive development is possible. They are capable of thriving for a shorter or longer period extra uterum.
2. *Omphalosites*, in which mere passive nutrition is effected through the placental circulation. They are altogether very imperfect, more especially in relation to symmetry of the two sides of the body.
3. *Parasites;* shapeless masses, deficient even in an umbilical cord, adherent to the sexual organs of the mother, and nourished at their cost.

The first order, *autosites*, is divided into four tribes: the first tribe into two families; *ectromeliens*, malformations with deficiency of the extremities, with the varieties—phocomèle, hemimèle, ectromèle; and *symeliens*, fusion of members, with the varieties—symèle, uromèle, sirenomèle. The second tribe has the single family *celosomiens*, prolapsus of viscera and imperfect anterior closure, anterior fissure, eventeration, with the varieties—aspalasome, agenosome, cyllosome, schistosome, pleurosome, celosome. The third tribe embraces the three families: *exencephaliens*, imperfect brain, extra cranium; *pseudoencephaliens*, slender rudiments of brain, with deficiency of a large proportion of the skull;

and *anencephaliens*, complete absence of the brain and skull, with their varieties. The fourth tribe resolves itself into families: *cyclocephaliens*, arrested formation and fusion of nose, eyes, and upper jaw; and *otocephaliens*, approximation and blending of the ears, with arrested development of the base of the skull and brain, and concurrent malformation of the apparatus of mastication—with their varieties.

The second order, *omphalosites*, has two tribes, with three families and their varieties. The first tribe comprises the two families, *paracephaliens*, rudimental head formation, asymmetria and absence of extremities and of many vegetative organs; and *acephaliens*, complete absence of head, with its varieties. The second tribe has the single family of *anidiens*, reduction of the entire organism to a membranaceous sac, enclosing various soft formations and sundry bloodvessel ramifications.

The third order, *parasites*, has the one family *zoomyliens*, rudimental embryo in the abdomen, in the genitals, &c., with a kind of zoomyle.

Double monstrosities are of two orders.

1. Double autositic monstrosities—fusion of two autosites.
2. Double parasitic monstrosities—union of an autosite with an omphalosite or parasite.

The first order (double autositic) embraces three tribes.

The first tribe subdivides into two families: *eusomphaliens*, the union of two nearly perfect organisms, each possessed of a normal umbilicus and umbilical cord, with several varieties; and *monomphaliens*, the union of two organisms having one umbilicus in common, also with several varieties.

The second tribe includes the two families, *sycéphaliens*, fusion of head and trunk; and *monocéphaliens*, two trunks, with one head; with their varieties.

The third tribe contains the two families, *sysomiens*, single trunk, with double head; and *monosomiens*, mere vestiges of duplicity about the head; with their varieties.

The second order (double parasitic) comprehends three tribes.

The first tribe has two families: *heterotypiens*, parasite and autosite united about the umbilical region, with varieties; and *heteraliens*, with the single species, epiconu, parasitic head upon the vertex (capitis) of the autosite.

The second tribe blends the two families of *polygnathiens*, imperfect head implanted in the maxillary apparatus of the individual; and *polymeliens* (μελος, membrum), the parasite consisting solely of extremities and adjuncts; with varieties.

The third tribe has but one family, the *endocymiens*, a parasite enclosed within the autosite.

The *triple monstrosities* admit of the same distinction as the *double*, namely, into *triple autositic* and *triple parasitic*.

The doctrine of the two Geoffroys, respecting malformation, frequently errs in their having neglected to adopt for its basis the natural laws of development. The system is even not devoid of logical inaccuracies, nor sufficiently compendious for practical use.

Another well-known German classification is that of Gurlt. He divides malformations, generally, into the three classes of malformation in one body, or simple monstrosities (monstra simplicia or unicorporea); double or twin monstrosities (m. duplicia or bigemina); and threefold or trigeminal monstrosities (m. triplicia or trigemina).

The first class is divided into six, and, including hermaphrodite forms, into seven orders.

1. Malformation from deficiency of parts.
2. From minuteness of parts.
3. From preternatural fissure.
4. From non-perforation and from fusion of parts (atresia and symphysis).
5. From preternatural form and site.
6. From extraordinary number of parts.
7. Hermaphrodites.

The second class has two subdivisions.

I. Double malformations from coalition.
II. Double malformations from implantation.

The first subdivision breaks up into four orders.

1. Coalition without separation at either end of the body.
2. Coalition with separation at the upper end.
3. Coalition with separation at the lower end.
4. Coalition with separation at both ends.

In this classification, as in those of Breschet, double formations are made distinct from malformations through excess of parts.

The most recent classification is that of Otto. It approximates to those of Buffon, Blumenbach, and Meckel. It arrays malformations in three classes.

First class.—Monstra deficientia, furnishing three orders.

1. M. perocephala, deficient in some one portion of the head, of which there are seven species.
2. M. perocorma, malformations with deficient vertebral column.
3. M. peromela, deficient development of the extremities.

Second class.—Monstra abundantia. These are divided into two orders.

1. M. ex duobus coalita.
2. M. luxuriantia.

Third class.—Monstra sensu strictiori deformia. It resolves itself into four orders.

1. M. fissione deformia.
2. M. coalitu singularum partium deformia.
3. M. atresiâ deformia.
4. M. morbis manifeste deformia.

To this system there is much to object: for example, that perverse site, that anomalies with respect to bloodvessels, and certain herma-

phrodite formations, have no place in it; that fissures and atresiæ are not admitted as monstra deficientia; that monstra abundantia are ascribed to coalition, &c.

Lastly, Bischoff begins by showing that for a classification of malformations the anatomical character alone can be made available, and that the physiological principle is here altogether inapplicable. Having then pointed out the proper method of determining the anatomical character —the diagnosis—of a malformation, he proceeds to build up the following system, which, generally approximating to those of Buffon and Blumenbach, frequently differs from both in detail, whilst by its rigid adherence to anatomical principles, it seems to take the only admissible ground.

First class.—Malformations deficient in some essential attribute of their kind.

Second class.—Malformations possessing more than pertains to the standard of their kind.

Third class.—Malformations, the organization of which does not conform with the standard of their kind, but without either the deficiency or the superfluity just referred to.

First class.—The causes to which the malformations, here under consideration, are due, may be very various. In many instances we are justified in regarding them as products of imperfect conception, whether the fault lie in imperfect formation of the ovum or in anomalous quality of the semen. At this day, however, so much in this assumption is still hypothetical that we are compelled to deal with it cautiously, addressing ourselves, where it is possible, to other causes, more especially to interrupted evolution of an organ out of its germ, or to its development being impeded through external influences, such as impressions wrought upon the mother; destruction of the organ, in the progress of its development, through disease, particularly through dropsical accumulation; finally, destruction of an organ through mechanical influence—for example, the amputation of a limb by means of the umbilical cord or a pseudomembranous formation within the ovum, &c.

This class comprises the following orders:

1. Deficiencies in a stricter sense.
2. Malformation from diminutiveness of parts.
3. Malformation from coalition (symphysis).
4. Atresiæ.
5. Cleft-formation.

Second class.—Here we meet with a regular progression, from the supernumerary bone or finger up to the development of two perfect individuals, united only at one part. The series of this progression is so graduated and so complete, that Bischoff regarded it as impracticable, even on anatomical grounds, to make any break in the respective formations, although Breschet and Gurlt have done this by distinguishing formations possessing single supernumerary parts with a single head and trunk, from those in which these latter are twofold, and which they denominate twin-malformations. This distinction is, however, based upon a physiological principle in itself objectionable. It is only to the

former species of malformation that we assign excess of plastic vigor as the source of the supernumerary parts; whilst true twin-formations are referred to the fusion or coalition of double primitive germs, imply-ing deficiency of plastic vigor, inasmuch as each germ individually must be imperfectly developed.

This class comprises the following orders:

1. Malformations from superfluity of single parts, with a single head and trunk.
2. Twin malformations with double head and trunk.
3. Double malformations from implantation.
4. Triple malformations.

Third class.—Its defect is that its characteristics are principally of a negative kind.

The objects comprised in this class being very numerous, their pro-bable sources are in a corresponding degree various. For several formations no other cause can be assigned than an anomaly of plastic activity originating in some primitive configuration of the germ; in a few other instances disease may be assumed as the cause; the majority, however, will be explicable on the ground of arrest of development.

This class contains the following orders:

1. Change in the position of organs.
2. Deviations in the form of organs.
3. Deviations in the origin and the disposition of the arteries and veins.
4. Hermaphrodites.

As, according to our plan, all malformations will be considered under heads corresponding with the classes and orders of the above system, and as we shall also follow Bischoff in our physiological notice of the species, an occasional reference to this system will, for the present, obviate the necessity for a further enumeration of the species belonging to each order.

On the other hand, we have here to notice, in a general way, the laws which nature observes in the production of malformations, so far as a general working out of this subject has revealed them to us.

1. The worst malformation is never so anomalous as not to bear the general characters of animal life, and the external semblance of the particular class of animals to which it belongs. Even an individual organ never departs from its normal character so completely that, amid even the greatest disfigurement, this character should not be cognizable.

2. Deviations from the normal are, then, confined within certain limits, and this applies in an especial manner to anomalies of position. Although that which should lie on the right may appear on the left, and the converse—the abdominal organs occupy the thorax, and the thoracic the abdomen—the brain has never yet been found in the chest or abdomen, nor the kidneys within the skull. The natural history of development reveals the cause,—different organs and systems being developed out of different layers of the germ; those pertaining to the same layer may indeed err as to their locality, but in no instance will

an organ pertaining to the animal, become evolved out of the vegetative layer of the germ, nor the converse. Fleischmann calls this the law of localities (lex topicorum).

3. To this we may add, that certain conjunctions between organs, for example, the aorta and the intestinal canal forming a single tube in common, never occur; but that, as a rule, homogeneous or kindred parts alone unite, a law termed by Fleischmann the law of individuality (lex proprietatis).

4. The excessive development of one part determines the imperfect, retarded development of another, and the converse. Meckel having laid it down as the next thing to a law, that a preponderance of one organ is associated with the retarded growth of another, Geoffroy St. Hilaire has invested this law—as the law of compensation (loi de balancement)—with the most ample significance and extension. The said law has in reality sundry facts for its foundation; it is alleged, in particular, that individuals having on one hand or one foot a supernumerary finger or toe, are often found wanting in a finger or toe on the other foot or hand. A fœtus described by Neumann had on the left foot only the great toe, but, on the right, eight toes, the eighth being cleft. Segala's fœtus had no thumb to the left hand—to the right, two; it had on one side eleven ribs only, but thirteen on the other. In cases where more or fewer important parts are wanting or imperfectly developed, we often find supernumerary fingers and toes; for example, in anencephalia, cyclopia, spina bifida, hare-lip, cleft abdominal parietes, &c. In the siren-malformation there is, according to Meckel, always an excessive number of vertebræ and of ribs. In acephali, deficient in heart and liver, the kidneys are asserted by Elben to be preternaturally developed. On the other hand, in the double formation of individual parts, others are frequently imperfect: thus, bitruncate malformations are frequently acephali, whilst the bicephalous have often spina bifida; and in either case sundry other organs besides have suffered an arrest of development, being deficient in abdominal parietes, the intestinal canal being imperfect, the urethra imperforate, or cloacal malformation present. Meckel has even made this law apply to different children of the same parents: one girl had on each hand a supernumerary finger, her sister had *two* fingers wanting to one hand.

Meckel, rightly, we think, rejects the assumption of a *law* of compensation, where compensation is so far from general, and admits only that malformations are often influenced by a law common to organized bodies.

5. Not every organ or part is in an equal measure obnoxious to malformation. According to Meckel it is far more rare in organs supplied by cerebro-spinal nerves (muscles, larynx, lungs), than in those supplied by the sympathetic (the digestive, urinary, generative). The vascular system is, however, most liable of all.

6. Certain malformations affect certain organs. Thus, it is an admitted fact that formations resulting from the vegetative and the vascular layer of the germ seldom multiply, compared with those which result from the animal layer. Instances of multiplied heart, lungs, in-

testinal canal, uropoietic and generative organs, are far more rare than of multiplied head, organs of sense, extremities, &c.

7. Whilst certain malformations are about equally frequent in both halves of the body, certain others affect by preference the one or the other side of the upper or the nether half of the body. Where the vertebral artery originates immediately from the aorta, "this," observes Meckel, "happens invariably on the left side." Cleft lip and cleft palate are commonly found on the right side. Malformations from superfluity are much more frequent in the upper than in the nether half of the body. Thus, bicephalous monsters with a single trunk are more frequent than monocephalous with double trunk; supernumerary fingers than supernumerary toes. In like manner, anomalous bloodvessels are more common in the superior extremities than in the inferior.

8. Female malformations are, by all accounts, much more frequent than male. A reason for this cannot at present be assigned.

Of the hereditary nature of malformations, and their repetition in children of the same parents, Meckel has collected numerous examples. The entail is transmitted equally through the male and through the female line. Meckel adduces an instance of a man with six fingers to each hand and six toes to each foot transmitting the same malformation to his eldest son, whose three sons again were born with precisely the same redundant organization.

Various and manifold as are the forms of monstrosity, some of them recur with such uniformity of type, as to constitute a regular series. This applies to every organ, each being especially liable to some particular kind of malformation. This circumstance is of great importance in summing up the causes of malformations. It indicates that, in the majority, not an extrinsic, accidental cause prevails, but an intrinsic one, inherent in the laws of germination and development.

With the aforesaid laws, derived more immediately from malformations, a mistaken attempt has been made to couple two other special laws:

1. The first being that of Serres, according to whom the development of an organ altogether depends upon the development of the bloodvessels, and especially of the arteries. Conformably herewith, imperfect development, or the absence, or again the excess of an organ or part, would be a consequence of the insufficiency, or the absence, or again of the preternatural development of the supplying artery. But even were the fact altogether true, the cause of the defective or excessive development of the artery would still remain to be accounted for. The relation, therefore, not of dependence, but merely of correspondence between the degree of development of the malformed organ and of its supplying vessels, would be proved, as the rule, and even this subject to occasional exceptions. Bischoff, however, regards as decisive the direct observation that, in their rudiments, organs are immediately evolved out of the germ, previously to their being furnished from bloodvessels; the constituting cells *subsequently* becoming metamorphosed in such wise, that, out of one portion bloodvessels and blood, out of another the other (secondary) elements of the organ are derived.

2. According to the second law, the nerves are substituted for the

bloodvessels, as the media of development. Tiedemann has shown that, with the absence of certain nerves is coupled the absence of their dependent organs; that in all monstrosities with excess, a corresponding relation is demonstrable in the nervous system; and, again, that in malformations with coalition of organs, the fashion of this union is exactly imitated by the supporting nerves. On the other hand, the natural history of development has shown that the central parts of the nervous system constitute the first vestiges of the embryo, being thrown out cognizably as such by the germ. Upon such grounds, the opinion has been formed that, like the normal, so also the anomalous development of the different organs of the embryo is dependent upon the normal or anomalous development of the nervous system. Against this view the objection hinted at in the last paragraph might again apply.

XVIII. The disposition to different diseases varies according to age, sex, climate, &c.

Thus, aneurism belongs chiefly to manhood and advanced age, rickets exclusively to childhood; the fœtus labors under anomalies proper to primary development alone—namely, malformations. In childhood tuberculosis attacks, preferably to all other parts, the lymphatic glands, the brain; at and beyond the age of puberty, the lungs. The female sex greatly favors the occurrence, in the sexual system, of cystoids, of cystosarcoma, of the majority of cancerous growths. Under certain climatic relations, tuberculosis is rare,—intermittent fever, hypertrophy of the spleen, frequent; under the tropics the ossification of arteries is said to be extremely rare. Again, particular regions and parts of the body manifest different dispositions with respect to the frequency of congenital or acquired anomalies. Thus, Portal pronounces apoplexy to be more frequent in the right corpus striatum than in the left,—pneumonia is more common in the right lung than in the left. The arteries of the inferior extremities are infinitely more obnoxious than those of the superior to ossification and to spontaneous aneurism; the veins of the lower half of the body are almost exclusively subject to varix. Malformation from excess appears more frequent in the upper half of the body, malformation from coalition more frequent in the lower half; variations in the course of bloodvessels are more rare in the inferior extremities than in the superior.

PATHOLOGICAL ANATOMY.

CHAPTER I.

ANOMALIES IN RESPECT OF THE NUMBER OF PARTS.

THESE consist in *diminution* or *augmentation* of the normal number of organic parts. It is not rare for both to be found united in one individual, one part presenting a deficiency, another, in virtue of the law of compensation, an excess of formation. Thus, monstrosities, in which otherwise deficiency predominates, will exhibit a superfluous finger or toe ; double twin malformations, on the contrary, deficiency in various parts.

Deficiency or absence of individual parts, or diminished number of plural organs, are frequent,—for example, the absence of entire extremities, of individual fingers and toes ; amongst the viscera, of one of the kidneys. It is either *congenital* or *acquired*. In the former case it includes malformations with *deficiency, in a stricter sense.* There is scarcely any part that has not been found wanting, without detriment to the entirety of the rest of the body. In this respect, however, certain relations or sympathy may not be overlooked, by virtue of which the absence of one part is paired with that of another part. Thus, in acephali the heart is almost always absent, very commonly, too, the entire thoracic viscera, together with the liver, the spleen, the pancreas, —an example of compliance with rule perfectly inexplicable ; for neither can development from an identical germ, nor functional dependence of the organs here be argued. Occasionally so many parts are absent at once, that nothing is born save a head, a single extremity, or a shapeless mass. The absence of parts is very often, though by no means invariably, due to arrest of development.

The principal kinds of malformation that pertain hither are the following (Geoffroy St. Hilaire, Gurlt, Bischoff) :

1. *Amorphus, anideus.* A shapeless mass consisting of cutis, areolar tissue, fat, and a few bones ; is never found but in association with a perfect twin. It probably results from an early destruction of the germ.

2. *Acephalus.* The head alone may be wanting, or with it more or less of the trunk, so that, in fact, nothing may be present save a pelvis with the inferior extremities, or with one of these only. It is for the most part associated with a twin. Even though the trunk be present,

the heart is usually absent; the respiratory organs probably always; liver, spleen, and pancreas commonly; stomach and intestinal canal are generally very defective; the uropoietic and generative organs are mostly present, though incomplete. It is in some instances perhaps deducible from injury to or destruction of the germ, or from the disturbance occasioned by a twin.

3. *Pseudacephalus, paracephalus.* Rudiment of head, with the rest of the body either entire or defective. These are sometimes twin cases, and are for the most part the consequence of hydrocephalus.

4. *Aprosopus.* The face, and especially the eyes, nose, and mouth, wanting. The skull is diminutive, and exhibits the ears coalescing, either in front or above. The brain is always very defective. The pharynx terminates, superiorly, in a cæcal sac. Here again, probably, destruction—splitting of the medullary tube, as also of the dorsal plates, at their anterior part, takes place at an early period. Hence the non-development of the anterior brain-cell with eyes and nose, and in like manner the superior arches of the cranial vertebræ, of the parietal and of the frontal vertebræ, and the consequent inclining towards each other of the temporal bones for the closure of the vertebræ. Hence, probably, also, the non-development of the anterior visceral arches, involving absence of the inferior maxilla and of the facial bones, with anterior junction of the external ears, which emanate from the second and third visceral arches.

5. *Microcephalus.* Diminutive, incomplete head; similar to the foregoing, except that the inferior jaw is present, proving the development of the first visceral arch.

6. *Anophthalmus.* Absence of both eyes or of one eye only. Eyelids and lachrymal organs are present, although often defective—coalescent. Perhaps for the most part dependent upon dropsical destruction of the eye-vesicles, rudiments of the optic nerves being commonly discoverable.

7. *Absence of the eyelids;* an arrest of formation, these organs being of later development.

8. *Absence of iris;* in like manner, an arrest of formation.

9. *Anotus.* Absence of the external ear,—deficient development of the external portion of the first visceral groove.

10. *Brachyrhynchus.* Shortness of nasal prominence, owing to deficiency of intermedial jawbones.

11. *Acormus.* One rudimental head with a regular twin, or with a triple birth. Most probably due to mechanical hindrance to development, and to destruction occasioned by the other fœtus or fœtuses.

12. *Oligospondylus.* Absence of vertebræ or of semi-vertebræ, is owing either to an anomaly of original germinal development, or else to the coalescing of two or more vertebræ or semi-vertebræ.

13. *Anaedœus.* Absence of the entire generative apparatus, or of the external organs of generation only; very rare as an independent malformation, where the individual is otherwise normally formed. It is an arrest of development, the said parts not being evolved out of the germ.

14. *Peromelus* and *micromelus.* The limbs are wanting or maimed.

It is frequently an arrest of development; it may, however, result from mechanical influence,—from the severing of members through strangulation.

15. *Phocomelus.* Monstrosity with phocal extremities, the hands issuing directly from the shoulders, the feet from the pelvis, whilst the intervening parts are either wanting or merely rudimental. It is an arrest of development often dependent upon hydrocephalus or spina bifida.

16. *Parosomus.* Various deformities caused by the absence of individual parts.

17. Absence of various individual organs of the thorax or abdomen, of a lung, the liver, the spleen, the stomach or its blind sac, a portion of intestine, &c. It is either an arrest of development or the result of morbid destruction.

A particular kind of diminution of number consists in the symphysis or fusion of kindred formations; fusion of two fingers, toes, ribs, vertebræ, of the inferior extremities, of the kidneys, obliteration of the uterus. A deficiency of various grades is included under *cleft-formations.*

The acquired absence of particular parts is the result of mechanical influence or of disorganization. To the former kind belongs maiming by accident or design; for example, amputation, extirpation, and the like, which sometimes greatly resemble certain congenital defects. To the latter kind is to be referred the wasting of various organs; for instance, from spontaneous, primitive atrophy, from that consequent upon inflammation, from destructive suppuration or gangrene.

Preternaturally augmented number of parts is very frequent, and commonly congenital. It occurs in every variety of grade, from the duplication of individual diminutive parts to that of the body almost in its totality. These various degrees constitute a series, the gradations of which are so regular, that it would appear forced to divide malformations of this class into such as with a single head and trunk present duplicates of individual parts, and into such as at the same time possess double or triple head and trunk. Nor does such a distinction derive much support from the assumption that the former are due to an excess of plastic activity, the latter to the primordial existence and the fusion of twofold germs.

Bischoff (with Winslow, Haller, Meckel) opposes the following weighty arguments to this distinction, as also to the assumption of twofold germs and their fusion (Lemery, Breschet, Gurlt, Chaussier, and Adelon).

1. The malformations of this class, from the supernumerary finger or toe up to the development of two perfect embryos united only at one point, constitute so complete and uninterrupted a series, that it would be in the highest degree forced to assign for the one and for the other opposite causes, namely, excess of formative activity, and again fusion with deficiency of plastic power. Still no one can hold a supernumerary finger or toe to be due to the fusion of two embryos.

2. In twin malformations, none but the same organs, systems, or parts, whether internal or external, ever coalesce. It is invariably thorax with thorax, abdomen with abdomen, head with head, breech with breech.

Again, brain is ever found coherent with brain, bloodvessels with blood-vessels, intestine with intestine, &c.; never trachea with œsophagus, nerves with bloodvessels, and the like. This is a fact, the constancy of which precludes its explanation on the ground of accidental fusion from external causes.

3. Twin malformations manifest, for the most part, a change not limited to the parts immediately united, but pervading the entire organism. Such a change could hardly be brought about by accidental fusion.

4. Twin malformations always recur with great uniformity of character. Are external causes likely always to combine after the same fashion ?

5. Again, twin malformations often occur out of the same mother, and the tendency to them is hereditary. There is neither proof nor probability of an abiding external cause,—for instance, in the maternal organs of generation.

6. At no period of development is a mechanical fusion of the ova and embryos probable; indeed, our present knowledge respecting the nature and earliest development of the ovum renders such fusion highly improbable. The pellucid zone or yelk-bag is in the highest degree unfitted for it; nor is it at all possible to press two ovula against each other in such wise as to occasion the fusion of their yelks, of their germinal vesicles, or of the zonæ pellucidæ of the latter. In the Fallopian tubes and the uterus, however contracted, the ovula undergo no such risk, even in multiparous animals, where they lie densely grouped together. After the embryos have become developed and shrouded within their amnia, their coalition ceases to be even conceivable. These membranes would have to undergo previous rupture; for the occurrence of twins within a single amnion is too rare and too inexplicable to be here taken into account. How slight is the disposition amongst various embryos to coalesce is shown by those cases of twins, in which, owing to confined space, the one is almost flattened by compression, without any coalition having taken place.

It results from the above that the aforesaid division of malformations with supernumerary parts, and the assumption of twofold germs and their coalition, are inadmissible. The task, therefore, still devolves upon us of explaining this class of malformations. Accordingly we have to observe that :

1. The cause might consist in an anomalous formation of the ovum in its unimpregnated state. Bischoff adduces in favor of this view the occurrence of double yelks, as also the aforesaid hereditary character of twin formations and their recurrence out of the same mother. This cause would, however, apply only to more perfect twin formations, it being difficult to imagine a mere supernumerary finger or even extremity to have a similar origin.

2. Some have long conceived the formative power within the germ to be endowed with unusual energy, causing the development of a greater number of parts than belong to the species. The facts observed by Wolff, Von Bär, and Reichert, certainly relate to twin formations of a very early stage of development. Still this stage is too far advanced

to be cited in direct support of an assumed augmentation of plastic power in the germ, as determining either an approach to the formation, out of the plastic materials of the yelk, of a double, or even of the partition of a single zona pellucida. On the other hand, these cases are of a period so early, and of characters so marked, as to render it almost self-evident that the cause of the malformation must have been a primordial one, or at least have dated from the earliest period. The multiplication of individual parts is possible at a later period, provided the germ remain for them unchanged.

3. One species of twin formations can at the present time only be satisfactorily explained by the assumption of an ovum in ovo,—one ovum being primitively enclosed within another. We refer to *twin formations from invagination or implantation*, so-termed conceptionlike germination (Meckel)—diplogénese par pénétration. One fœtus incloses, at some part, another imperfect fœtus—a fœtus in fœtu : or else one fœtus is at some one point, commonly at the skull or palate, united with another fœtus, through the medium of a more or less perfect umbilical cord. Meckel regarded the fœtus in fœtu as a product of conception, and sought to maintain this view by an appeal to analogy ; adducing, for example, the formation of hair and teeth independently of copulation, sexless multiplication and propagation, regeneration. At the present day monosexual conception is hardly—multiplication by cotyledons or offshoots, in nowise—admissible. Certain of the observations in point relate to cases of malformation in the early embryo, in the third and seventh month, for example, in which a conceptionlike product is simply impossible. The occurrence of ovum in ovo, in the instance of birds, at least, is proved ; the intussusception of one ovum into another during development is, on the other hand, not conceivable.

4. Finally, an augmented number of parts depends not unfrequently upon arrest of development, and the anatomical excess is reduced to one of no real physiological import ; for example, the true diverticulum of the intestine as the remnant of the omphalo-mesenteric duct, double frontal bones, and the like.

Malformations, with supernumerary parts, are divisible into several orders, which, with their principal species, are as follows :

1st order.—Malformations with individual parts supernumerary—head and trunk being single.

Dignathus. Malformation with supernumerary lower jaw.
Caudatus. Human fœtus with tail-like process at the os sacrum.
Polydactylus. Malformation with supernumerary fingers.
Notomeles. Monstrosity having supernumerary limbs at the back.
Pygomeles. Having supernumerary limbs at the os sacrum.
Gastromeles. With supernumerary limbs at the normal extremities.
To which are to be added :
　　1. Supernumerary skull bones.
　　2. 　　　" 　　　vertebræ.
　　3. 　　　" 　　　ribs.
　　4. 　　　" 　　　muscles.
　　5. 　　　" 　　　teeth.

6. Double tongue (always superimposed).
7. Double œsophagus.
8. True diverticulum of intestine.
9. Double cæcum and vermiform process.
10. Double pancreatic duct.
11. Double hepatic duct.
12. Manifold spleen.
13. Double heart.
14. Multiplicity of kidneys, probably due to arrest of development.
15. Double or triple ureters.
16. Double bladder.
17. Triple testicle (?).
18. Double penis and clitoris (?).
19. Double uterus (U. duplex, bicornis, bilocularis); to be regarded altogether as arrest of development.
20. Testicles and ovaries, seminal ducts, seminal vesicles, Fallopian tubes, uterus, &c., in the same individual.
21. Supernumerary mammæ.

2d order.—Twin monstrosities, with double head and trunk.
(a.) Double formation of the upper portions of the body.
Heteroprosopus.   Two countenances; the one perfect, the other imperfect.
Dicranus.   Double skull; countenance either single, or double and conjoined; lower jaw single.
Monocranus.   Single skull; countenance partially double; brain double, but unequally so; three or four eyes.
Diprosopus.   Double countenance; the faces and heads are completely separate, or the separation affects the faces to the zygomatic arches only; lower jaw invariably double.
Dicephalus.   Two entirely separate heads, with two (seldom three) upper, and two (seldom three) lower extremities.
Thoraco-gastrodidymus.   Two heads and necks, thorax and abdomen united into one; four upper and two or three lower extremities. (The Sardinian twin sisters.)
Gastrodidymus.   Twins united at the lower part of the belly; the four inferior extremities branch off from the sides in pairs, at right angles.
Pygodidymus (Gurlt), *Pygopages* (G. St. Hilaire).   Two completely distinct bodies, conjoined at their ossa sacra or coccygis. [The well-known Hungarian sisters, Helena and Judith, born in the year 1701, who survived their 22d year.]
(b.) Double formation of the nether parts of the body.
Dipygus or *Monocephalus* (Gurlt), *Thoradelphus* (G. St. Hilaire). Head, neck, and thorax single; abdomina and posterior parts separate; two or four upper, always four lower, extremities.
Heterodidymus (Gurlt), *Heteradelphus* (G. St. Hilaire); so-called parasite formation.   A large, regularly formed body, bearing, at the chest or belly, another, more or less incomplete.
Dihypogastricus; so-called Janus formation.   Double body, more or less coalescent above; separate from the umbilicus downwards.   Here,

either two heads are united with the two countenances (one of which is commonly defective), presenting in opposite directions; or else there is but a single (perhaps defective) countenance, with a double coalescent head. The trunk is double, united down to the umbilicus, and has four upper and four lower extremities.

Symphysocephalus (Barkow), *Cephalopages* (G. St. Hilaire). Twin monstrosity united at the head; the twins may be perfect, or of the one nothing may exist except the head.

(*c.*) Double formation, both above and below.

Diprosopus diœdoeus (Barkow), *Tetrascelus* (Gurlt). Two heads, united at the sides; thorax and abdomen coalescent; two or four upper extremities; urinary and generative organs, as also the inferior extremities, double.

Hemipages (G. St. Hil.) The heads superficially coherent at the sides; lower jaw in common; neck, thorax, and abdomen as far down as the umbilicus, coalescent; pelves separate; four upper and four lower extremities.

Thoracodidymus (Gurlt). Two distinct bodies united at the thorax.

Xyphopages. Two perfectly distinct bodies, united only in the vicinity of the ensiform process. (The well-known Siamese twin brothers.)

3d order.—Twin malformations through implantation.

Cryptodidymus (Gurlt); so-called fœtus in fœtu. The greater, perfect fœtus bears at some point beneath the skin, or within its natural cavities, a second, smaller, and imperfect fœtus.

Omphalo-cranodidymus. The umbilical cord, together with the rudiment of the one fœtus, rooted within the skull of the other.

Epignathus. An imperfect fœtus rooted, with its bloodvessels, at the palate of a more perfect fœtus.

4th order.—*Triple monstrosities.* Their existence is confirmed by modern researches.

Supernumerary parts may be normal, both in form and structure; in both respects, however, they are frequently in various degrees defective.

The frequency of duplicate forms varies in different portions of the body; for example, a multiplication of viscera, or of organs of sense, is far more rare than of extremities.

It will be seen from the above, that in twin monstrosities the connection between two individuals is either a mere superficial one, occurring through the medium of skin and of bone, or else one involving, at the point of union, the blending of cavities of the body, and the union, in various degrees, of the same organs in the two individuals.

Acquired preternatural increase of number consists, in man and in the higher animals, in a multiplication of the elementary constituent parts of a tissue,—of the essential or the secondary structural elements which enter into the composition of an organ. It is, therefore, the manifestation of increase of mass or density in an organ—never of the development of new, complex ones. Still, the arrest or alienation of tissues developed for the repair of injuries, or of destructive morbid processes, does sometimes determine the formation of supernumerary apparatuses foreign to the standard of the organism; for example, anomalous excretory ducts; accessory articulations.

CHAPTER II.

ANOMALIES OF SIZE.

ANOMALIES of volume manifest themselves as irregularities in magnitude, and as their opposite, diminutiveness, both being either congenital or acquired. They are often relative only, that is, applicable to one period of development or of life. Again, their significance and import may be limited to the proportions of the organ concerned, as in smallness of the brain; in enlargement of the heart. Finally, they refer, either uniformly or unequally, to the entire body or to individual organs.

ABNORMAL MAGNITUDE.

Congenital abnormal magnitude is sometimes general. In relation to the entire body it is termed gigantic growth—macrosomia. Some children are born inordinately large and powerful, and endowed with other marks of precocious development besides; for instance, closure of the sutures, unusual strength and length of hair, extrusion of one or more teeth. Others, impelled by innate predisposition, undergo preternatural growth during youth, and eventually arrive at dimensions exceeding the ordinary standard—in a word, grow up giants. Giant stature may depend upon the equal and proportioned lengthening of all the parts, or upon the predominant length of certain sections of the body, especially of the lower extremities. Giant stature does not needs imply corresponding development of the substance of organs and parts, certain of which may possibly have been checked in their growth; for example, the muscular system, the heart, the brain, the adipose tissue, the organs of generation.

Preternatural dimensions of individual organs of the body, both congenital and acquired, are of far more frequent occurrence. These originate in a primary anomaly or in excessive plastic activity of the germ, or, again, in hypertrophy, or in the dilatation of hollow organs, or, lastly, in a variety of diseases in which the textures of organs become involved at different periods of intra- and of extra-uterine life. These last consist for the most part, in hyperæmia, inflammation, and all kinds of heterologous formations. Congenital enlargement sometimes imports arrest of development, as, for example, a preternaturally large thymus gland.

Hypertrophy and the dilatation of hollow organs require to be considered somewhat more at large.

HYPERTROPHY.

Hypertrophy consists, as the term implies, in augmented nutrition, resulting in increase of mass, and generally also of volume. Long ere the term hypertrophy, and even the idea it conveys, were formally recognized in science, not only had the possibility of an increase of mass

and volume without material destruction of texture been speculated upon, but the fact itself actually observed in every variety of organ. Even up to the present time, however, the recognition of this species of anomaly has been characterized by a great want of clearness and precision. It is reserved for the discrimination of the present generation, by a searching comparative inquiry, based upon a more familiar acquaintance with the normal relations of the structure and admixture of organs, and aided by the physical appliances now at our command, to make an important progressive step over this wide and fertile field.

1. *Simple augmented nutrition*, the increment of mass and of volume, not dependent upon the accession of any element foreign to the organ concerned—TRUE HYPERTROPHY.

2. *Anomalous augmented nutrition.* The increase of mass and of volume is here founded upon the accession of matter alien to the organ concerned, be it formless blastema or determinate form-element. This anomalous matter, when uniformly incorporated with the texture of the organ, that is, received both betwixt and within the definite structural elements, manifests itself as *infiltration of the parenchyma*—FALSE HYPERTROPHY. It approximates closely to heterologous growth.

Such are the two sections into which, as a preliminary step, we would distinguish all the so-called hypertrophies. Each will, however, have to be specially considered in the sequel.

Widely as the *two* hypertrophies should appear to differ from each other, and little as, strictly speaking, the second series belongs hither, its consideration in this place will be found preferable as regards practical utility, and expedient for other weighty reasons.

(*a.*) From true hypertrophy to false there are insensible gradations, both qualitative and quantitative, and both forms may coexist in the same organ. Thus, augmentation of the fatty contents of the hepatic cells is, by the addition of free fat and by a change in the quality of the fat, exalted into a palpable heterologous process.

(*b.*) Between the two series there exists the common connecting link that both are based upon an anomaly of the crasis; that, provided no obvious local causes prevail, both are engendered by a peculiar, personal, more or less defined, morbid tendency of general nutrition.

Every organ is by nature susceptible of, and almost every one has with more or less of precision been described as actually found affected with, hypertrophy. This does not, however, now apply equally to both categories of hypertrophy—that of the areolar and of adipose tissues, and of the muscles, more especially the organic, commonly manifesting itself as *true*, that of the so-called parenchymatous organs still more commonly as *false hypertrophy*.

(*a.*) *True hypertrophy.*

True hypertrophy appears, *à priori*, incontestable, and numberless instances have been recorded of its occurrence in every part of the body. It is remarkable, however, that when tested by an analysis, with reference to elementary texture and development, the proof is attended with extraordinary difficulty as regards the most important organs and tissues.

When it is the question, not of an obvious augmentation of the less

important components of an organ—for example, of the areolar, the fibrous, the adipose tissues—but of a multiplication of the essential structural elements, the positive proof by elementary analysis often fails, although, both before and after death, the characters of the organ may seem quite sufficiently exaggerated to warrant the assumption of *true hypertrophy*.

Apart from a development of substance resulting from extraordinary succulence of the texture—that is, from its imbibing an excess of amorphous plasma more or less rich in nutrimental substances—hypertrophy can only depend either upon a multiplication of the essential textural elements by an accession of new ones, or else upon an enlargement of the original ones. Upon this point a generalization is not feasible at the present day, and it must suffice to set forth in due succession the results of researches touching special hypertrophies.

We may specify, as unquestionable, hypertrophy of the areolar, of the fibrous, and of the adipose tissues; of the common integuments, including, not alone the cutis and the papillary bodies, but also the sebaceous glands and the epidermidal formations; of the mucous membranes and their follicles; and lastly, of the bones.

Hypertrophy cannot indeed be demonstrated by a comparative enumeration of the form-elements; and the size of the latter varies considerably even in the physiological state. Where, however, the increase of mass is obvious, and there is no accession of heterogeneous elements, the sum of the primitive form-elements must needs have become multiplied, and hypertrophy exist. We find, too, in the involved textures—for example, in areolar tissue—an extraordinary number of the elements in their embryonic stages.

Hypertrophy of muscle, however simple it may seem, is in reality most difficult of proof. The increase of mass and volume in a hypertrophied muscle certainly seems due to augmentation of its amount of fleshy fibres; positive evidence, however, at least with respect to the striated muscles, has hitherto been wanting. An enumeration of the primitive fibres is not feasible, nor have elements obviously engaged in the embryonic phases of new muscle-formation been as yet detected. Still less has an enlargement of the primitive muscular fibres, through increase in the amount of their primitive fibrils, been made out. That the hypertrophy consists simply in the augmented growth of the myolemma is disproved by the saturated dye, the extraordinary resiliency, the functional energy, for example, of a hypertrophied biceps brachii. Least of all could it be explained on the ground of augmented fat formation—the effect of excessive development of fat, in whatever shape, being to repel the growth of muscle. The last two propositions are moreover refuted by the hypertrophy of organic muscles.

The examination of hypertrophied hearts, for which the opportunity is frequent, offers but little assistance towards the solution of the problem, more especially where the increase of mass is considerable. A new accession of muscular fibres is not manifest. On the contrary, in proportion to the diminished energy of the organ, their fibrils are found in the progress of reduction to a partially dark-colored molecule, and of gradual extinction. One thing alone is evidently adventitious, namely,

irregular aggregations of an amorphous fibro-laminated blastema, co-piously interspersed with nuclei in different grades of development into areolar tissue, and of areolar tissue itself, together with a large propor-tion of free fat and of adipose tissue.

In the hypertrophy of organic muscle the characters are more clearly defined. Here, along with nuclei, we meet with little, flat, elongated, and nucleated bodies, the rudiments of new fibres. A marked instance of hypertrophy of this nature is afforded, amongst others, by the preg-nant uterus, which, at the same time, exemplifies the disintegration of fibre, and the lingering of a multiplicity of nuclei, which are themselves eventually absorbed.

Even hypertrophy of the nervous system is little more than a problem. The development of fresh nervous filaments is unproved and even impro-bable. Nor is the enlargement of the nerve-tubules through increase of their contents more readily demonstrable. In the central organs, and particularly in the brain, the anomaly consists in an accumulation of the minute granular connecting mass interstitial to the nerve-tubules. At the circumference it can consist only of an augmentation of the neuri-lemma. In the ganglia the accession of new ganglion-cells, though not ascertained, is rendered probable by the regeneration of excised ganglia.

One of the hypertrophies most frequently discussed is that of glandu-lar bodies. We shall pass over, for the present, the false hypertrophies so frequent, particularly in the liver, the spleen, and the kidneys. That of other—for example, the mammary, the salivary—glands may consist in an augmentation either of some constituent of secondary importance —for example, areolar or adipose tissue—or of the parenchyma itself; and it is with evidence respecting this last form that we are here princi-pally concerned.

Examined with the naked eye, the parenchyma of the enlarged pro-state gland, as that best adapted for this experiment, certainly appears to have undergone an increase of mass. This might be brought about either by the creation of new acini (lobules), or by the enlargement of existing ones, through the apposition of fresh enchyma-cells; or, lastly, by the co-operation of both. The appearances in hypertrophy of the prostate gland render the new formation of lobules and of lobes highly probable. Henle's observations, however, of the existence of solitary enchyma-cells in the vicinity of the glandular lobules in the lachrymal gland of a calf, and still more, what is very readily witnessed, in hyper-trophied thyroid glands, render probable the new formation of such gland-cells, and, through the resorption of their partition walls, their blending with the lobules so as positively to enlarge these.

A peculiar kind of hypertrophy, concurrent with dilatation of the cavities of the acini, is a very frequent cause of the enlargement of glandular formations. This dilatation is due to an augmented secretion taking place within the follicles, determined by the same local or general causes as the hypertrophy itself; and this latter consists in an increase of mass in the investing fibres of the follicles. This condition is imme-diately followed by the expansion or degeneration of the follicle to a dilated cyst-like cell, with a stouter lamina of enveloping fibres. At the same time the secretion may become alienated both in quantity and in

quality, until a cyst is completed with contents altogether alien to the native secretion of the gland. This is witnessed in the follicles of the thyroid gland, in the Malpighian bodies of the kidneys, in the Graafian follicles, and in the acini of the salivary glands; in the mucous follicles, particularly those of the cervix uteri, where, even in the physiological state, they frequently dilate into capacious, thick-coated cysts, rupture, and discharge their contents.

Evidence of hypertrophy of the liver, of the spleen, of the lymphatic glands, is hardly obtainable.

The idea of a hypertrophy of the liver from the accessory formation of new hepatic cells would not indeed be discordant with our notions of the functional importance of that organ. All anatomical proof is, however, unattainable. That, on the other hand, upon which it more *obviously* depends, is turgescence of the hepatic cells from an increased proportion of fat and of bile, together with hyperæmia of the capillaries. This condition determines the more or less marked development of what is called the secreting substance of the liver,—one-sided hypertrophy of the liver, as it is termed, or nutmeg liver.

Hypertrophy of the spleen must be referred, first, indeed, to the reinforcement of its fibrous framework, but mainly to augmentation of the pulpy parenchyma of the spleen, that is, of the elements out of which it is constructed.

Nor can hypertrophy of the lymphatic glands be well traced to the adventitious development of new lymphatic vessels between their parenchyma, but rather to increase of the parenchyma between the lymphatic vessels. It is certain, at least, that in atrophy, the lymphatic vessels become deficient in parenchyma.

Hypertrophy of the lungs consists not in the addition of new cells, but in an augmentation of matter in the parietes of the existing ones. The ample, energetic (vicariating) function, moreover, of a hypertrophied lung seems to imply a multiplication of the capillary vessels by the creation of new ones. Thus, again, hypertrophy of the corpora cavernosa does not depend upon the addition of new cellular spaces, or their increase through the development of new septa, but upon increase of substance, thickening of the walls of the cellular spaces, with simultaneous dilatation of these latter.

(*b.*) *False hypertrophy.*

This has been already adverted to as a heterologous product. As such it occurs frequently in the form of infiltration. False hypertrophy is for the most part cognizable at a glance from the alienation which the general characters of the organ have undergone. Very marked hypertrophies of this kind are found to affect the liver, and with rather less frequency the spleen and even the kidneys; presenting, in the instance of the two former, what is commonly termed hypertrophy, physconia, engorgement, &c. These manifest themselves in the shape of fatty liver, waxy liver, of albuminous, lardaceous infiltration of that viscus, of the spleen, of the kidneys, and they will be reconsidered under the head of heterologous growths. They not rarely attain to a very high grade, are always distinguished as being palpably based in a constitutional dyscrasis,

and are, proportionately to the rapidity or slowness of their development, attended or unattended with pain.

To these hypertrophies, moreover, properly belongs the ultimate degeneration of hypertrophied and dilated glandular follicles into cysts.

Finally, we may here class all hypertrophies founded upon products of inflammation, so far as they consist in the adventitious development of a blastema foreign to the texture involved, and convertible into areolar and fibroid tissue.

Hypertrophy attacks one, or a few disconnected, but for the most part nearly kindred organs; or, again, an entire system—for example, the osseous, the lymphatic system. The general hypertrophy called polysarcia or corpulence, consists both in the excessive development of fat, and in extraordinary succulence of the soft tissues, more especially of the areolar.

Hypertrophied organs offer a variety of remarkable changes. The volume is usually augmented,—more obviously so in the case of false hypertrophies. Now and then the natural volume is retained, the failure of increase of volume being compensated for by augmented density of the organ, or some one anatomical constituent becoming hypertrophied at the expense of another one, which wastes in a corresponding degree. In hollow organs we distinguish between a *simple* hypertrophy with normal capacity, an *excentrical* with dilatation, and a *concentrical* with diminution of the cavity. In this last the volume of the organ may be augmented, or normal, or even diminished. Examples are afforded in hypertrophies of the heart, of the uterus, &c.

The weight of hypertrophied organs corresponds with their increase of volume and of density.

The shape always undergoes a change proportionate to the degree of the enlargement. Generally speaking, hypertrophied organs assume a certain roundness, losing their edges, their angles, and their flat surfaces. In the case of some organs, and of the liver in particular, the marked character of such disfigurement is not devoid of pathognomonic significance.

The *color* is, in *true* hypertrophy, the normal—only of deeper tint. Take, for example, the saturated red in true hypertrophy of muscular flesh, the saturated twofold coloration of nutmeg liver. In false hypertrophy, the coloration suffers various alterations.

The consistence of a hypertrophied organ is often unchanged, often increased, sometimes diminished. A remarkable degree of density and of resiliency characterizes hypertrophied muscle, more especially in the right ventricle of the heart; and, again, in hypertrophy of the spleen referrible to mechanical hyperæmia, and unattended by obvious enlargement. The same observation applies to false hypertrophies, in particular to brawn-like infiltration of the liver, the spleen, and the kidneys. *Fatty* degeneration of the liver is marked by a diminution of consistence.

The bloodvessels of hypertrophied organs sometimes present a dilated calibre and thickened (hypertrophied) coats. This is especially perceptible in congestion and hypertrophy of long standing; not so, or at least not in a marked degree, in other cases. Does the accessory formation

of new structural elements in the hypertrophied organ imply that of new bloodvessels likewise? Direct experience affords no information upon this point. In relation to the hypertrophy of vicariating organs, and of the lungs more particularly, it would be reasonable, where vicarious action really is in force, to take for granted the accessory formation of new capillaries. The nerves of hypertrophied organs are occasionally found considerably thicker than natural.

The causes of hypertrophy are:

1. Morbid increase of the quantity of blood in the capillaries of, and retarded circulation in, the affected organ; repeated and abiding hyperæmia. Examples are furnished in particular by the frequent hypertrophies of the abdominal viscera arising out of mechanical hyperæmia, of the mucous membranes in organic diseases of the heart, of the areolar tissue in the lower extremities in a varicose condition of their veins, and, lastly, by the hypertrophies of the mucous membranes brought about by the hyperæmia entailed by repeated inflammation.

2. Augmented, violent action induced by various direct or reflected stimuli. Examples present themselves in hypertrophy of the voluntary muscles, of the heart, of the organic fleshy tunics.

3. The groundwork of a lengthy series of hypertrophies consists in *a constitutional vice of nutrition and in an anomalous blood-crasis.* The hypertrophy is here the expression, the symptom, of general impairment. This applies to true, and with greater force to false hypertrophy. To this class belong hyperostosis, excessive development of fat, endemic goitre,—hypertrophy of the brain, and hypertrophy of the lymphatic glands in rhachitism,—the excessive development of fat with simultaneous impairment of its quality in alcohol-dyscrasis,—the conditions of fatty, of waxy liver, of brawny infiltration of this organ, of the spleen, of the kidneys in tuberculosis, rhachitisms, inveterate syphilis, &c.

4. *Inflammation;* the result of which is so-called *inflammatory hypertrophy,* to which we shall have to recur by-and-by. It engenders *true* hypertrophy in areolar and osseous textures alone; in all the rest, through the fresh deposition of areolar and of fibroid tissues, *false* hypertrophy.

Hypertrophies of both kinds are either congenital, or, what is far more frequent, acquired during extra-uterine life.

The *course* of hypertrophies is for the most part chronic. Nevertheless, they not rarely form within a surprisingly short period, or from time to time rapidly increase. They are then often painful affections, as, for example, the acutely developed fatty liver.

Hypertrophy, when it has attained a high degree, impairs the function of the affected organ, whilst the latter, by its increase of weight and of volume, obstructs the function of neighboring parts.

Of itself it commonly proves fatal through palsy, the result of the ultimate disproportion between the bulk of the hypertrophied organ and the powers of innervation. As examples may be cited hypertrophies of the heart, palsy of the hypertrophied intestine above a stricture, palsy of the hypertrophied urinary bladder, and the like.

A proper discrimination is requisite between increase of volume from hypertrophy and the dilatation of hollow organs, more especially if asso-

ciated with attenuation of the parietes. Dilatation is generally coupled with hypertrophy of the walls of the dilated organ,—termed *active dilatation*, co-significant with *excentrical hypertrophy*. *Simple dilatation*, in which the walls are of their natural thickness, is a kindred form. Dilatation may, however, be conjoined with attenuation of the walls; it is then denominated *passive dilatation*.

The causes of the dilatation of hollow organs are various.

1. *Mechanical impediments*, which obstruct the free passage and egression of the contents of the different canals and reservoirs. They occasion dilatation either beyond or behind their seat, and manifest themselves—

(*a*.) As local constriction of calibre, through pressure from without.

(*b*.) As coarctation consequent upon hypertrophy and change of texture in the walls of the organ. In instances rare, except in disease of the heart, as dilatation. Thus, whereas in the intestinal canal it is the accumulation of its contents, on the other hand, in dilatation of the orifices of the heart it is the increased diameter of the blood-column, in insufficiency of the heart-valves the regurgitation of the blood, that furnishes the mechanical impediment.

(*c*.) As obturation of canals with substances of various kinds, whether introduced from without or begotten within the organism, whether closing up by their bulk or obstructing by their aggregation,—in a word, as foreign bodies, secretions, &c.

Other local causes, however, besides the above-mentioned—accumulations of foreign bodies, of self-engendered deposits—are in like manner productive of dilatation.

2. Paralysis of the contractile elements in the walls of the organ, whether peripherous, and consecutive to mechanical, concussive violence, tension, &c., to disease of texture, especially inflammation; or determined by affection of the nervous centres.

3. Diseases of texture; for example, fatty degeneration, particularly of the heart.

4. *Inflammations*.

The different causes frequently act in unison in various sequences. Thus, coarctation begets accumulation of contents. This, together with a certain relative amount of existing dilatation, occasions paralysis of the organ. The paralysis causes dilatation, and thereby accumulation of the contents, which again, in turn, mechanically promotes the dilatation.

Dilatation destroys life through paralysis, either simply or with the concurrence of asthenic stasis, inflammation, and gangrene, towards the establishment of which the contact of retained contents in the progress of decomposition contributes its part. Take for example the intestinal canal, the urinary bladder, &c.

Sudden dilatation is wont to assume the passive character, a supervenient hypertrophy being more marked in the inverse ratio of the celerity with which the dilatation is brought about.

Increase of volume in one direction at the expense of the general bulk of the organ, the result of forcible tension, is distinct from hypertrophy.

ABNORMAL DIMINUTIVENESS.

Congenital abnormal diminutiveness affects the entire body, as *dwarf stature* (microsomia), the individuals being termed dwarfs, or pigmies. These are either born diminutive, or, owing to inherent predisposition, not developed after birth to the ordinary stature. Dwarf-growth manifests itself either in the corporal development remaining stationary at the stage of childhood, the not unpleasing outlines and proportions of which it then retains, or else it is founded in an arrest in the growth of the bones, especially those of the lower extremities, with simultaneous malformation of the osseous trunk. It is marked by a disproportion in the more important parts of the body—largeness and hydrocephalic shape of the skull; length of trunk coupled with shortness of extremities, especially of the inferior ones; deformity of bones, consisting in thickness, especially of the articular terminations. This latter dwarf-formation is always congenital, and the bone affection upon which it depends has been designated as congenital rhachitis; against which, however, we have as yet to urge that, however much its features resemble those of the rickets of childhood, the direct evidence of its identity with the latter is wanting.

Accordingly, dwarf-growths may depend, either upon a primitive vice of plasticity, or in an anomaly of development affecting specifically the osseous system. Growth may, moreover, become checked at an earlier or later period, subsequent to birth, by constitutional maladies of an exhausting kind, both congenital and acquired, and especially by such as affect the brain or spinal medulla.

Partial diminutiveness affects individual organs, systems, or sections of the body. It is founded sometimes in a primitive anomaly of, or in defective plasticity in, the germ; in pressure and in restriction of space within the uterus; or, again, in a hindrance to growth after birth, resulting from exhausting diseases, from paralysis; lastly, in atrophy. Where larger sections of the body are affected, such disproportions result as are observed in giant-growth, in dwarf-growth, and in numerous descriptions of monstrosity. Although it may affect every organ and system, it is nevertheless most conspicuous in the following: namely, the brain (and skull) [microcephalia]; the eyes [microphthalmus], the inferior maxilla [brachygnathus], the lungs (and thorax), the stomach and intestinal canal, the common integuments (shortness), the muscles, the skeleton, the heart and vascular system (especially the aortal), the generative apparatus.

It is necessary to observe, that—

(*a.*) In monstrosities, accessory, supernumerary parts are very frequently diminutive.

(*b.*) Formations checked in the development of their mass and volume, often exhibit an arrest in the development of their texture; for example, bones, muscles.

(*c.*) Preternaturally diminutive organs often display some other kind of deformity, referable to the same causal relations.

(*d.*) Next to diminutiveness is total absence, which, in reality, often

applies to individual formations entering into the composition of a complex part or system.

In hollow organs preternatural diminutiveness manifests itself as coarctation, and even as complete imperviousness, which, when affecting the external orifice of canals, is termed imperforatio, atresia.

As contrasting with hypertrophy, atrophy here demands a special consideration.

ATROPHY.

Atrophy, wasting [tabes], consists in the withdrawal from a formation, after it has reached a certain grade of maturity and bulk, of its constituent elements, without any compensating regeneration of these; the result being decrease of substance, usually coupled with diminution of volume.

General atrophy attacks simultaneously, or in rapid succession, many organs and systems, if not all. *Partial atrophy*, one organ exclusively, or at least preferably. To the latter we shall at once direct our attention.

As with hypertrophy, so every organ is liable to become affected with atrophy.

Atrophy may, in the first place, be essentially primary, that is, developed in an organ as its first and sole anomaly, through influences more or less palpable, but external to such organ. Or, again, it may be secondary, that is, the result of previous textural alteration in the organ. The first is akin and analogous to the periodical intra- and extra-uterine processes of involution of certain formations, as also to the senile atrophy or *marasmus* of organs.

Primary partial atrophy is often, indeed, purely local; in not a few instances, however, it is probably conditional upon a general derangement of nutrition, of which, in such case, it is but the manifestation or symptom.

Causes of partial atrophy are:

1. *Diminished supply of blood*—of alimentary fluid—owing to compression, obturation, coarctation, or obliteration of the afferent blood-vessels; for example, partial atrophy with lobulation of the liver from adhesive phlebitis of branches of the portal vein, atrophy of the cartilaginous investments of the joints from sclerosis of the spongy condyles, and the like. Thickening of the minute and capillary vessels from within may co-operate with ossification of the great arteries in producing atrophy, especially in the brain, by rendering the walls of such vessels impermeable to the plasma of the blood.

2. *Exhausting disease, or healing process;* for example, atrophy of the bones and of their adjacent soft parts as a consequence of caries, of destructive suppuration in the effort to repair injuries; atrophy of the uterus after childbed, and exhausting puerperal diseases, &c.

3. *Diminished innervation*, paralysis, or impeded action of an organ owing to mischief of a mechanical nature; for example, atrophy of muscles in anchylosis, in luxations.

4. *Pressure and distension.*—These occasion increased absorption, a species of atrophy designated by the term *detritus, usura.* Even the

most stubborn textures are not proof against it, the rigid osseous texture itself being in a high degree susceptible of it. It not unfrequently advances to the degree of a lesion of continuity.

5. *Anomalies affecting general nutrition, and the blood-crasis in particular.*—Upon such are based, for example, perhaps, the untimely decline (involution) of the generative organs in either, but especially in the male sex; but with more of certainty, several painful kinds of atrophy of the osseous system. Acute yellow atrophy of the liver is unquestionably founded upon an anomaly of the crasis, whilst the thyroid gland is atrophied by the fluids becoming impregnated with iodine.

6. *Consecutive atrophy* depends, as already stated, upon a previous alteration of texture, a breaking up thereof through hemorrhage (apoplexy), inflammation, and heterologous growths. Two contingencies may here arise; either the adventitious product and also the disabled textural elements of the organ may both undergo absorption, or else these latter may waste away alone, leaving in their place the new product, in the original or in subsequently diminished proportion and indefinite shape. Exemplifications occur in cell-infiltration of the medullary substance of the brain consequent upon encephalitis; in atrophy of the kidneys, resulting from inflammation or from Bright's granular disease; finally, in the merging of muscular fibre in the fatty degeneration of muscle and of normal textures in heterologous growths.

The morphological process connected with atrophy is not known in detail. To judge by a few facts—for example, the reduction of the uterus after delivery, the perishing of muscular fibre in fatty degeneration, the wasting of nerves, of lymphatic glands—the process essentially consists in the breaking down and liquefaction of the secondary elements (fibre), resulting from the metamorphosis of the cells and from the cells themselves. The nuclei at first remain, but subsequently undergo the same reduction and ultimate resorption. In the case of new growths, this blastema, arising out of the wreck of the said elementary bodies, may become subservient to the construction of anomalous textures.

With respect to the changes suffered by atrophied organs in their physical properties, we may offer the following general remarks.

The volume of the atrophied organ is indeed very commonly diminished, membranous formations having become thinner : this is, however, by no means invariably or immediately the case, at least not in any marked degree ; for example, in atrophy of the lungs or of the bones. In hollow organs the volume may, owing to a coexistent passive dilatation, even become augmented. In such organs atrophy is conjoined either with normal capacity, *simple atrophy;* or else with dilatation of the cavity, so-called *eccentrical atrophy*—for example, of the heart, of the uterus, &c. ; or, lastly, with coarctation, *concentrical atrophy*, in which the depth of the walls may be natural, or even greater than natural.

The weight of atrophied organs may be reduced, normal, or even increased : in the first case, proportionately to the simplicity of the atrophy; in the two latter cases the atrophy is consecutive, new growths supplanting the original textures.

The shape of atrophied organs embraces a variety of anomalies;

amongst which we may specify the deformity which attaches to the concentrical wasting of hollow organs and organs of cellular structure like the bones, the removal of the incisura interlobularis in atrophy of the lungs, the tuberous gland-like surface in secondary atrophy of the kidneys, &c.

The structure of atrophied organs involves various, and occasionally very marked changes. Thus, organs of cellular, of cavernous structure, by dint of the absorption which takes place at the parietes of their cells and canals, are rendered wide-celled—for example, in the lungs and in bones; and this structure eventually dwindles into a mere net or trellis-work. In consecutive atrophy, a new growth of a completely different texture occupies the place of the original structure; after inflammation, for instance, a honeycombed, meshy, or, on the contrary, a dense, callous, areolar tissue.

The consistence is in like manner subject to many changes. It is sometimes diminished, readily giving rise, upon slight occasion, to lesions of continuity; in the osseous system, for example. Sometimes it is increased. Secondary atrophy presents, in a marked degree, either contingency, according to the particular change of structure. Acute processes of the reduction of mass and volume determine, in certain organs, rather a decrease—chronic reduction rather an increase—of consistence.

Atrophied organs have a tendency to paleness of color. This, however, in some measure accords with certain changes affecting the native pigment of organs; for example, the decoloration of muscle, of the spleen to rust-brown, fawn-color, or yeast-color. The rule itself is, moreover, subject to sundry exceptions. Thus, the pure white of the medullary substance of the brain is exchanged for a whitish-brown. Organs that become atrophied without a proportionate thinning of their capillaries, sometimes, by dint of a *relatively* augmented supply of blood, assume a deeper color; for example, bones, kidneys. At the same time much depends upon the character of the atrophy; as in the cases of red and yellow atrophy of the liver.

The bloodvessels of atrophied organs become reduced in calibre, collapsed, and finally cut off from the atrophied organ, that is, the connection interrupted between its obliterated capillaries and the vascular trunk. This, however, has, in like manner, its exceptions, as in the case of dilatation of the trunk and ramifications of the pulmonary artery in atrophy and in emphysema of the lungs; in the case of dilatation of the bloodvessels of the brain, in atrophy of this organ.

The nerves of atrophied organs in all probability dwindle *pari passu* with the wasting of the diseased textures.

Atrophy is sometimes an acute, but more commonly a chronic process. In the former case it is frequently a painful affection.

The consequences of partial atrophy differ vastly in different organs. Either they are limited to a small range, to the locality itself, or they implicate more or less sensibly the entire organism. In this respect, atrophy of the central organs of the nervous system, and of the organs presiding over the preparation of the blood and over the grand secretory

functions, namely, of the lungs, liver, and kidneys, is of course foremost in importance.

General atrophy in the form of emaciation, consumption, affects, indeed, the entire body, but by no means all organs and systems simultaneously or in an equal degree. Next in order to the falling off in the amount of blood, is that of the adipose, the areolar, and kindred tissues; then follows that of the voluntary muscles, then of the organic fleshy tunics and of parenchymatous organs, lastly of bones; whilst, even in the highest grades of the affection, the nervous system, so far as relates to its constituent elements, remains exempt. This order is, however, subject to many exceptions. The wasting of several formations low in the scale above laid down, occurs primitively, and offers the starting-point for the atrophy of the rest; for example, atrophy of bone.

The causes are loss of fluids of whatever kind, deficient reproduction of organic substance, fasting, various affections of the digestive organs, bodily and mental exertion, inordinate activity of the nervous system in various ways, excessive heterologous development, dyscrasis of the blood.

It is not rarely combined with the hypertrophy—for the most part, false hypertrophy—of internal organs, especially of the liver, spleen, and lymphatic glands.

A peculiar form of partial diminution is represented in the *coarctation of canals and cavities*. It is often, indeed, essentially a concentrical atrophy. It may, however, arise from external pressure, from deficiency of contents, from continued irritation of the sensitive parietes, or even from hypertrophy of,—or from various heterologous luxuriations and changes of texture implicating,—the said parietes. The highest grade manifests itself as morbid closure (atresia).

CHAPTER III.

ANOMALIES OF FORM.

Anomaly of form, or *deformity*, affects either the entire body or portions of it only,—*general* or *partial deformity*. It is either *primitive* or *acquired; simple* or *complicated*, that is, conjoined with anomalies of a different nature.

General deformity is rare, even as relates to very faulty abortions.

To *primitive, simple partial deformities* belong—

(*a.*) Those in which any part is preternaturally long, broad, thick, spherical, angular, curved, &c.; for example, oval, vertical pupil, oblique uterus.

(*b.*) The division of parenchymatous organs into two or more parts, by extraordinary lobulation (the lungs, liver, spleen, kidneys); the section of hollow organs by the inordinately sharp partition of a naturally

double cavity, or by septformation in a cavity normally single; for example, double apex of the heart, bilocular uterus.

Many of the former, and still more of the latter kinds bear the impress of arrest of development, and present the images of brutes.

Amongst *primitive complicated deformities* may be classed most of the instances of disproportion and of absence of symmetry manifested in the preternatural volume of individual organs or sections of organs; secondly, those consisting in abnormal position, abnormal association, coalition or cleftformation; and, lastly, those founded in the superfluity or in the deficiency of parts.

Pre-eminent amongst them are hermaphrodites. The forms of hermaphroditism, strictly considered, range under several of the heads just specified as conditional upon anomalies of shape. It would, however, appear most suitable to discuss them here under a single head, seeing that from one or more fundamental anomalies inductive of hermaphroditism there often results a marked deformity of the generative organs; seeing also that the character of many of them consists *essentially* in a departure from the normal type. They are, in the great majority of cases, arrests of development.

In strict analogy with the relations of lower orders of animals, those malformations should be designated as hermaphrodites, in which the generative organs of both sexes are found united in a single individual. Such monstrosities have, from time immemorial, been abundantly described. We must, however, unite with Joh. Müller and Th. Bischoff in rejecting the great majority of these examples. Bischoff has pointed out the numerous sources of error by which, in such cases, a judgment may be warped; as, for instance, the great resemblance between the generative organs of the two sexes at an early period, the uniform type in the development of both, the coalition of the corpora Wolffiana, the errors formerly prevalent as to the primitive identity of both sexes. It is, therefore, easily intelligible that a judgment to be relied upon can alone be based upon a familiar knowledge of the progressive development of the genital organs, and of their elementary structure. The coexistence of testicles and of ovaries on the same side has been thrown into entire discredit by the arguments of Joh. Müller, who nevertheless admits the occurrence of ovaries on one side and of testicles on the other. Th. Bischoff, however, impugns the accuracy even of the latter observation. Nor will Bischoff unconditionally admit the numerous cases of other portions of the genital organs alleged to have been found bisexual on the same side, or male on the one side and female on the other. The history of development, he affirms, sufficiently teaches us that this species of simulation may be the result partly of an arrest, partly of a peculiar modification in the type of development. Moreover, the progressive development of the uterus, of the seminal vesicles, of the prostate gland, and of Cowper's glands, in both sexes, still remains, notwithstanding the skilful investigations of J. Müller, Rathke, Valentin, so far matter of uncertainty that we can hardly derive any support from analogy with the normal state.

Strictly speaking, therefore, neither in man nor in the higher animals, can hermaphroditism, that is, the coexistence of testicles with ovaries,

occur. So far as relates to these essential organs of generation, there can be but male or but female individuals. On the other hand, the rest of the genital organs, which in their rudimental condition closely resemble each other in the two sexes, may, owing to some anomaly in the mode of their development, assume in a male individual more or less of the feminine, in a female individual more or less of the masculine form—and thus, in either case, the semblance of both combined.

If, with Bischoff, we rightly discard from hermaphroditism, cases of individuals with throughout female organs but masculine habit, and, again, with perfect male organs and feminine habit—irrespectively of a simply undersized penis or a preternaturally developed clitoris—we may, consistently with our usual classification, divide hermaphrodites into—

1. Those which being, as to the essential organs of generation (testicles and ovaries), distinctly male or female, exhibit nevertheless some anomaly of development [be it arrest, overgrowth (up to the masculine type), or disproportion of some other kind] more or less typical of the opposite sex.

(a.) *Hypospadia* in its highest grades, namely, on the one side with cleft scrotum and the formation of a vagina-like sinus—on the other side, as its analogue, diminutive vagina, closure thereof into a raphé or suture, partial or entire absence of this organ, with a clitoris developed into the semblance of a penis hypospadiæus, or one completely channelled with a urethra.

(b.) *Cryptorchism* : concealed testicles in the one case; in the other its parallel condition, descent of the ovaries into the greater labia pudendi. Now and then associated with the foregoing form.

High grades of these anomalies constitute the so-called transverse hermaphroditism, implying external organs of the one and internal of the other sex. The case of externally female and internally male organs is by far the more common, because due to an arrest in the development of the male organs, whilst the opposite case depends upon the ulterior development of the female organs into the male type.

(c.) The occurrence in the male sex of a womb-like organ.

These cases collectively constitute what is termed spurious hermaphroditism.

2. *Lateral hermaphroditism.* The presence of testicles and vas deferens, with or without seminal vesicles, on one side, and of ovarium and tube on the other. It has been before stated that Bischoff attaches little credit to these alleged cases of the coexistence of testicle with ovary.

3. True hermaphroditism (hermaphrodite per excessum, androgynus, coexistence of male and of female organs on the same side). With reference to these cases, recorded by Meckel and by Gurlt, Bischoff remarks that not a single one offers conclusive evidence of the union of the two main organs of generation, the testicle and ovary, and that the seeming dualism of the rest of the organs is explicable according to principles of normal development.

Amongst *acquired deviations* of form are to be enumerated, first, those conditional upon hypertrophy and atrophy; upon change of locality and of connection—as, for instance, hernia, prolapse, oblique position of the

uterus from one-sided traction, luxation; upon mechanical interference—for example, amputation, extirpation; upon cicatrization; and, lastly, those malformations of organs which essentially depend upon alterations of texture—misshappen liver, for instance.

The most frequent and marked kinds of deformity are founded upon anomalies of the osseous system; for instance, curvatures of the spine, of the long cylindrical bones, dislocations, preternatural articulations, &c.

CHAPTER IV.

ANOMALIES OF POSITION.

PRETERNATURAL position—situs mutatus, inversus, alienus, dislocatio, ectopia—is either congenital or acquired. In either case, it may affect a single organ or implicate several.

To congenital anomalies of the kind belong:

1. The re-establishment of symmetry, in lateral asymmetria. For example, each lung is found to have two lobes only, with both liver and heart in the centre. This is probably an arrest of development, these organs originally occupying the median line, and being in appearance symmetrically constituted.

2. *Lateral transposition, displacement from side to side*, affects either only individual organs of the thoracic or abdominal cavities—the cæcum being, for instance, on the left, the heart on the right side; or else it affects the aggregate of the thoracic or of the abdominal viscera; or, lastly, and most commonly, the collective organs of both these cavities at once. The type of formation is reversed, the right greater lobe of the liver, for instance, becoming the left, the left becoming the right, the gall-bladder lying to the left of the longitudinal fissure. As regards the cause, it appears to Bischoff that in the embryo at an early period, the umbilical vesicle, after development of the intestine, verges towards the left, and the allantois towards the right, whereby a peculiar spiral revolution of the embryo is effected, which may possibly influence the position of the internal organs. It is conceivable that a change in the position of the germinal vesicle in the ovum might, in like manner, give rise to a transposition of organs.

3. *Transposition from above and below.* Thoracic organs in the abdomen; abdominal in the thorax.

4. *Transposition from front to back;* for instance, in the case of teeth, in distortion of the extremities.

5. *Displacement of individual organs from the median line*, as for, instance, of the falx (cerebri),—of the uterus. *Displacement upwards*, as in cervical position of the heart. *Displacement downwards*, as in abdominal site of the heart, pelvic position of the kidneys.

Anomalous origin and distribution of arteries and veins. The more important examples hereof will be discussed under the head of special anomalies of the heart. They originate, for the most part, from blood-

vessels which should have become further developed stopping short in their progress, whilst others which should have remained diminutive, or even have disappeared, persist and become more strongly developed. The majority represent types proper to different vertebrata—to fishes, amphibia, birds, and mammalia.

The preternatural position of certain organs which, in their development, undergo locomotion to a considerable extent, is specially termed *deviation, aberration*. An example offers in the descent of the testicle beneath the femoral arch or into the perinæum. In truth, many anomalies of position are founded in an early aberration in this sense. The same designation is applied to anomalies in the origin, course, and ramification of vessels.

Acquired transposition is of various kinds, and many of these so closely resemble the congenital forms, as with difficulty to be distinguished from them. Their import varies greatly, proportionally to—

(*a.*) The importance of the organ displaced;

(*b.*) The number of organs displaced;

(*c.*) The extent of the displacement; and

(*d.*) Especially to the rapidity with which the dislodgment takes place, and to the corresponding strain upon various formations, more particularly bloodvessels and nerves.

(*e.*) The extent of the morbid complication to which the displacement is due; for example, mechanical injury to the surrounding parts.

(*f.*) The degree of embarrassment to which the dislodged organs become subject; for instance, limitation of space, incarceration, exposure to the external air, &c.

(*g.*) The amount of functional embarrassment inflicted upon organs by the displaced parts; for example, upon the lungs by the intrusion of abdominal viscera into the thoracic cavity.

These transpositions are, moreover, *spontaneous*, where the organ changes its position owing to increase of volume, of mass, or of weight, in which case it commonly sinks into a lower region. Or else they depend upon conditions extraneous to the organ displaced; to which class belong dislodgments consequent upon atony of investing, supporting, attaching formations, especially when of a muscular and fibrous nature. Or they are referable to tonic spasm and retraction of fleshy, of tendinous, and of ligamentous formations, as exemplified in hernia, in curvature and distortion of the spine, in luxations, in club-foot, &c. Lastly, we have to mention the displacement of organs, through tumors, through dislodged or enlarged neighboring organs, through accumulated fluids, and the like.

The more important forms of displacement are:

1. *Hernia ;* the extrusion of one or more viscera, or of merely a portion of a viscus, out of its natural cavity into a sac formed by the circumscribed dilatation of the membranous investments of that cavity (hernial sac).

2. *Prolapsus ;* the naked extrusion of a viscus through a natural orifice. It is either complete or only partial; the former case occurs in hollow organs—for example, in the rectum, in the prolapsed and inverted womb. At an external opening of the body intussusception

becomes prolapsus, which is intussusception minus the external layer or sheath.

3. *Protrusion*, propendentia, of viscera, owing to congenital fissure, or to rupture or penetrating wounds of the parietes of cavities.

Again, the position of organs may be anomalous, independently of any change of place, simply by preternatural inclination, especially in the shape of obliquity. This species of deviation is sometimes primitive and congenital, sometimes acquired. It affects the eye, the heart, the stomach, the uterus, the teeth, &c. It is frequently coupled with obliquity of form, as in the case of the uterus.

CHAPTER V.

ANOMALIES OF CONNECTION.

THESE anomalies (vitia nexûs) consist in diminution or total absence, or else in enhancement of the natural connection and contiguity of organs. They are both primitive and acquired, and, in either case, exceedingly various in degree and extent. To the former belong the opposite extremes of cleft-formation, and of malformation from fusion, together with atresia.

1. Cleft-formations.

(*a*.) A considerable number of these have their foundation in the germ being originally a membranous expansion, the edges of which incline towards each other, eventually meet, and thus form into cavities or cylinders.

The two cavities developed out of the animal layer of the germ, for the inclosure of the central nervous system and of the organs of the neck, the thorax, and the abdomen, are formed out of the union of the so-termed abdominal and visceral plates. Now, supposing the union of the edges of these plates not to take place at all, or to take place but imperfectly; or supposing consummated union to become redissolved through some agency, like the accumulation of watery fluid; there would result, either anteriorly or posteriorly, and commonly at the median line, although often elsewhere, a cleft or gap, attended by prolapse, or even by destruction of the implicated viscera.

Clefts of this kind are :—
    Cleft skull (hemicephalia).
    Cleft spine (spina bifida).
    Cleft countenance.
    Cleft cheek.
    Cleft palate.
    Cleft upper-lip.
    Cleft tongue.
    Cleft in the thorax.
    Cleft in the abdomen.
    Cleft in the pelvis.

Cleft urinary bladder, so termed prolapsus, inversio vesicæ.

Cleft dorsum penis (epispadiasis).

The last two are generally combined with cleft pelvis.

The intestinal canal is in like manner developed out of an expansive formation, the united vascular and vegetative layers of the germ, by the approximation of its edges, out of a groove in front of the vertebral column. Hence, clefts occur in the intestinal canal, in the stomach, as arrested growths.

(b.) Other clefts, besides those mentioned, originate in the gaps which occur during the normal development of particular parts, not closed at the proper time. To these belong:

Cleft choroid membrane and iris (*coloboma iridis*). In the embryos of all vertebrata we meet, at an early period, at the inner, lower angle of the eye, with a narrow colorless stripe in the choroid membrane, which commonly disappears before the iris becomes developed. When this stripe continues beyond this period, it often abides in the iris, and is perceptible after birth.

Cleft at the side of the neck, congenital fistula of the neck, founded in the mode of development of the visceral cavity of the head. The visceral edges of the animal layer of the germ do not grow towards each other in continuity, but in ridges, termed visceral or branchial arches, which are parted by fissures, termed visceral or branchial clefts. When the early closure of these does not take place, occasion is given to the somewhat rare malformation in question.

Cleft urethra and scrotum (*hypospadiasis*) of various grades. At an early period is discoverable, at the lower side of the rudiment of the penis, a groove, which extends to the common orifice of the urinary and sexual organs. In the male, the edges of this groove being brought into apposition, coalesce into a raphe or suture, and thus form at once the scrotum and the urethra. Where this process wholly or partially fails, there arises a malformation which, if the penis be at the same time short and the testicles retained within the abdomen, simulates female development—a form of spurious hermaphroditism.

To this malformation succeeds—

Cloacal formation, junction of the orifice of the anus and of the external orifice of the urinary and sexual organs—a formation which, being at an early period normal, may, through an arrest of development, become persistent. In the male it is necessarily associated with the last-mentioned vice of formation, that is, with hypospadiasis, frequently also with cryptorchism.

(c.) As cleft-formations may likewise be reckoned the persistence of certain communicating apertures between parts which, at a later period, ought to remain separate, as also the abiding patency of certain canals, namely:

Defective development of the septa of the heart's ventricles and auricles; permanent patency of the foramen ovale. These septa form only gradually within the heart, the septum of the auricles not arriving at its full development until after birth. Defective development of the septum of the ventricles occasions a resemblance with the hearts of fishes and of reptiles (the crocodile excepted), and especially of serpents and tortoises;

absence of the septum of the auricles a resemblance, in particular, with the hearts of fishes. It is often quite evident that the arrest of development has been caused by endocarditic changes in the valves—the residue of fœtal valvular inflammation.

Abiding patency of the ductus arteriosus, ulterior dilatation thereof.

Abiding patency of the ductus venosus Arantii, giving rise to the abduction of a portion of blood from the vena portæ into the vena cava.

Abiding patency of the processus vaginalis peritonæi (the upper portion of the tunica vaginalis testis), so commonly the cause of congenital hernia or hydrocele. Generally speaking, the inguinal canal closes immediately after the testis has, in the seventh month, descended into the scrotal sac, carrying with it a process or continuation of the peritonæum. Occasionally an arrest of development prevents the said closure from taking place.

Abiding patency of the urachus, allowing the escape of urine through the umbilicus. Urachus and urinary bladder are the portions of the allantois internal to the embryo, which is destined to convey the umbilical vessels from the embryo to the external membrane of the ovum (chorion), for the formation of the placenta. The portion of the allantois external to the umbilicus becomes obliterated at an early period. Of the internal remnant the inferior portion becomes developed into the urinary bladder, whilst the portion intervening between that and the umbilicus, contracts into a cord, the urachus.

Cleft member (*schistomelus*) commonly appears from between the third and fourth fingers or toes to the wrist or ankle. It is probably derived from external causes, and, as Gurlt infers from an examination of the fœtus of a dog, from adhesion to the amnion.

2. *Malformations through fusion* (*symphysis*). To these belong:

(*a.*) *Cyclopia.* In this malformation we find in the forehead a single eye, or the two eyes blended into one. It is met with under every gradation of the fusion of both eyes. The nose is either wanting, or defective, being frequently represented by an imperforate proboscis-like appendix, which overhangs the one eye or the two united. The mouth is sometimes normal, sometimes misshapen—nay, the entire infra-frontal countenance may be wanting. The ethmoid, nasal, lachrymal, turbinated bones, the vomer, the superior maxillary and palatine bones, the pterygoid processes, are often all or severally absent; the anterior lobes of the brain invariably so. One explanation of this deformity is based upon Huschke's hypothesis, of both eyes being developed out of a single primitive rudiment, subsequently divided in twain by the interposition of the nasal and facial parts. An arrest in the development of these parts might then, indeed, suffice to occasion the mischief. Bischoff, however, firmly maintains that the two eyes originate at once, distinct and separate, from the anterior primitive brain-cell, and he derives the cyclopian deformity from an arrest in the development of this cell causing the too close approximation and eventual fusion of the rudiments of the two eyes. As this defective development of the brain-cell frequently causes a defective development of the anterior portion of the plastic material for the chorda dorsalis, and often for the anterior process of the first

visceral arch, it would thus occasion the absence, before alluded to, of the aforesaid facial bones.

(c.) *Monotia, agnathus, otocephalus.* The two ears approach each other more or less below the skull, and finally coalesce. The inferior maxilla is wanting. The superior maxillary, the zygomatic, the palatine bones, along with the pterygoid processes, are either, in like manner, wanting, or else inadequately developed. The mouth is absent or very diminutive. The skull is normal, but the face small, and in brutes projects after the fashion of a proboscis. Bischoff considers this deformity referable to an arrest of development of the first visceral arch, intercepting or impairing the growth of all the said bones, and thus promoting the mutual approximation of the two ears beneath the skull. Were the internal organs of hearing implicated, the source would needs reside in a defective development of the third primitive brain-cell.

(d.) *Monopodia,* M. Syrenomeles,—Siren-malformation. The two lower extremities, more or less perfectly developed as to their individual parts, are blended into a single one. The pelvis, the sexual and urinary organs, are wanting or imperfect; the intestinal canal is defective beyond the cæcum, and the anus invariably absent. The extremities, moreover, have revolved upon their axes, the direction of the patella and of the poples of the knee being reversed. It is founded in a faulty development of the lower end of the trunk and of its organs, the rudiments of which approximate too closely towards each other, and ultimately coalesce.

Syndactylus, aschysto-dactylus. Here the fingers or toes are imperfectly separated. It is an arrest of development, the rudiment of hand and foot, even when distinctly cognizable, not manifesting at first any division of fingers and toes.

(e.) *Fusion of kidneys, testicles, and ovaries.* This, according to Bischoff, is not due to arrest of development,—even these organs not originating from a single rudiment,—but rather to a defective development of the intermediate formations occasioning fusion of the rudiments.

3. *Atresiæ.*

(a.) *Atresia palpebrarum.* The eyelids are said to coalesce naturally towards the end of the third or the commencement of the fourth month, and to separate afterwards. Accordingly this malformation would be an arrest of development.

(b.) *Atresia oris.* According to Burdach, the lips coalesce in the fourth month, closing the mouth until the sixth, when they again separate. According to Bischoff, however, this malformation might have a different origin. At a very early period, namely, the visceral edges of the animal layer mutually incline towards each other inferiorly, unite, and form, through the medium of Rathke's so-called inferior bond-membrane, the visceral cavity of the embryo. Not until the visceral arches break forth above, does the upper portal to the nutritive canal open, and not until still later the mouth. The atresy might, therefore, depend upon the abiding of the *bond-membrane.* In either case it would be an arrest of development.

(c.) *Atresia pupillæ.* Until the seventh month the pupil is closed by the membrana pupillaris, the anterior section of a vascular sac in which

the lens, with its capsule, is inclosed. Its persistence determines the atresy.

(*d.*) *Atresia nasi.* According to Burdach, the nostril becomes closed during the fifth week by a saccular plug, which gradually disappears during the fifth month. Its persistence would occasion the atresy.

(*e.*) *Atresia auris externæ.* The external meatus auditorius is developed out of the posterior upper portion of the first visceral fissure. Previously to birth it is upon the whole little developed. A slight anomaly of formation may give rise to its closure, although at no period is the latter normal.

(*f.*) *Atresia ani.* The anus is not present at first, even where the terminal intestine has formed. A stand-still at this period, however, would involve the simultaneous closure of the urinary and sexual organs, seeing that their external orifices are all developed out of the primitive orifice of the terminal intestine,—the cloaca. Where, therefore, the anus is alone closed, the mischief must date from a later period, namely, after the separation adverted to has already taken place. Some physiologists believe it to be, at a certain epoch, the natural condition.

(*g.*) *Atresia vulvæ.* Probably conditional upon the turgescent edges of the external orifice of the uro-genital canal being brought into apposition, and coalescing in the female, as they ordinarily do only in the male sex, for the formation of the scrotum. Where the anus is at the same time deficient, we have here again non-development of the cloacal outlet.

(*h.*) *Atresia vaginæ*, frequently due to a preternaturally large hymen, although occasionally to a partial deficiency thereof, causing two blind sacs to overlap and compress each other.

(*i.*) *Atresia uteri*, not being derivable from the mode of development of the uterus, is to be regarded as a vice of formation, or else as the result of inflammation.

(*k.*) *Atresia urethræ*, in the male, an arrest of development,—the groove at the nether part of the penis, out of which the urethra is developed, not extending to the glans. In the fourth month the glans becomes perforated, in the natural course: if this process be checked, this part will remain imperforate.

To *acquired* anomalies of connection belong, firstly, actual interruptions of continuity, together with their not invariable but frequent and obvious associate, *diastasis,*—that is, the parting, through loosening or lesion of continuity of the binding material of two bones immovably connected together, and again the estrangement and deviation of the articular ends of two bones,—luxation. Secondly, agglutination and concrescence of two or more formations originally contiguous, or brought by accident or design into mutual association, and abiding contact with one another. Agglutination is effected through the binding property of recently exuded fibrin ;—concrescence through the medium of textures newly formed out of exuded protein substances, and like unto the normal textures, for example, areolar tissue ; or through the medium of such as differ in certain respects, for instance, in the degree of density of aggregation,—in the arrangement of their form-elements,—in chemical composition,—cancer, for example,—and, lastly, through the medium

of vessels. This species of concrescence, in accordance with the cha-
racter of the binding material, is effected by loose, filamentous adhe-
sions, admitting of a certain degree of motion in the affected organs;
or by tense and intimate conglutinations.

The adhesion of the parietes of hollow organs, and the obliteration
of canals and of their mouths, represent *acquired morbid atresy*. This
originates in various ways: for example, in concentric atrophy conse-
quent upon deficient expansive power. Thus, in ducts, it follows the
extinction of the gland; in bloodvessels, the cutting off of the blood
stream; or it is the effect of abiding compression and mutual contact of
the parietes; or, again, it may be the result of the deposition of
organic matter out of the contents of the canal, or of textural changes
in its walls, produced by inflammation, cicatrization, and the like; or,
lastly, of luxuriating heterologous growths.

The coalition of the two bones in mutual contact within an articula-
tion is specially termed articular adhesion, anchylosis.

CHAPTER VI.

ANOMALIES OF COLOR.

ANOMALIES in the color of organs are either essentially conjoined
with or independent of change of texture. Our concern here is princi-
pally with the latter kind. We shall content ourselves with a simple
allusion to the former, as the true pathological production of pigment
will be separately discussed in the sequel.

The said anomalies consist in diminution, in augmented depth, or in
altered quality, alienation, of color. They affect the totality, or simply
the majority of textures and organs, or, again, individual organs only,
or mere circumscribed portions of these latter. Their causes, it will
immediately appear, are numerous.

Diminished coloration is sometimes primitive; various organs, owing
to an arrest of development, not acquiring their natural amount of
color, a condition very commonly associated with the defective develop-
ment of the organs in respect both of texture and of bulk, as in the
instance of muscles. We may specify a well-known species of this
anomaly, namely albinoism (leukæthiopia, leukopathia), wherein the
pigment is wanting in the rete mucosum of Malpighi, in the hair, in the
iris, and in the choroid membrane of the eye.

It is, however, more frequent as an acquired morbid condition—in a
word, as *decoloration, blanching*. Under this head are to be reckoned,
firstly, the pallor of texture consequent upon anæmia, or upon changes
suffered in various dyscrases, as chlorosis, albuminosis, dropsy, tabescent
diseases, by those carriers of coloring matter, the blood-corpuscles;
secondly, the blanching of textures produced by their maceration in the
serum of dropsy, by atrophy, by fatty degeneration, the muscles being

here the organs principally affected; lastly, that for the most part local, gradual, or rapid decoloration, due, in the former case, to cessation of the development of pigment, in the latter to rapid withdrawal, or more probably to destruction, by some unknown means, of the existing pigment. As an example we may cite the topical blanching of the common integuments in colored tribes, in parts rich in pigment (the scrotum) in whites, the progressive or sudden hoariness of the hair, &c.

Augmented or deepened coloration is, in certain colored textures, in the animal muscles, for instance, the consequence of and the attendant upon hypertrophy. In the outer integuments, it is conditional upon the excessive development of pigment in the rete mucosum; in scars, as a purple tint, caused by the capillary vessels appearing through the texture of the cicatrix, in the embryonic stage, with its thin layer of epidermis. It is usually a consequence:

(*a.*) Of hyperæmia (congestion) and stasis, more especially where the blood is dark-colored, as in cyanosis, in asphyxia, in a typhous crasis, in an inspissated condition of the blood, consequent upon loss of serum, &c.

(*b.*) Secondly, of hemorrhage, extravasation of substantive blood into textures (apoplexy); sugillation or suffusion founded in rupture, which latter may be the consequence either of traumatic influences, such as contusion and concussion, or of excessive hyperæmia, mechanically produced; of blood stasis; of disease of bloodvessels; or else the consequence of the patency of bloodvessels, engendered by the liquefaction, the breaking down of textures.

(*c.*) Finally, of the *exudation of blood-serum*, with an *appendage*, so to speak, of *blood-pigment*—to which category belong ecchymosis, petechiæ in decomposition of the blood, in scurvy, in putrid typhus, in acute exanthematous decomposition, in acute alcohol dyscrasis (the scurvy of drunkards), &c. As subordinate to this we may also mention those outward signs of death which assume essentially the guise of red coloring, namely, death patches, death livor, spurious sugillations. Their character varies:

1. They are dependent upon local hyperæmia and stasis, brought about during the death-struggle and the period immediately following death, as the consequence of unequable palsy of the small and capillary vessels,—whence it arises that the blood accumulates in distinct patches of the capillary system various in extent, whilst in others, owing to the continued contractility of those vessels, it is urged onward into the veins. Hence they consist in injection, and, where the usual accompaniment of the livor, from imbibition, is wanting, they are for the most part marked by being sharply bounded by a blanched texture. They are particularly frequent in the mucous membrane of the intestinal tract, and in the lungs.

2. Other death-patches originate after death, being the result of cadaverous hyperæmia or hypostasis, which signifies the descent of the blood within the vessels, conformably with the laws of gravitation, to the most dependent parts. These patches are mostly of great extent, deeply saturated at their most dependent parts, and less and less so higher up. Their seat, answering to the usually supine position of the dead body, is the occiput, the posterior part of the trunk and limbs;

and it includes, not alone the common integuments, but also the subcutaneous soft parts, and even the posterior portions of the viscera contained in the great cavities. Under different circumstances, they affect other regions of the body; in a lateral position of the corpse, the nether lateral half of the organs—for example, in hemi-lateral hyperæmia, the one hemisphere of the brain; in the prone position they appear in the front; in those hung by the neck, at the lower half of the subject and internally, in the organs of the pelvis and hypogastrium.

They are the more developed the greater the amount of blood and the smaller the degree of coagulation which the previous illness and the mode of death have produced in the blood during the mortal struggle. Accordingly, after acute or chronic decomposition of the fibrine in the blood, after asphyxia, they are especially marked by their rapid development after death, by their extent, and by their depth of color.

3. A third species of death-marks arises from the imbibition, by the coats of bloodvessels, and the transuding from thence into the neighboring tissues, of blood-serum, which, owing to decomposition, has taken up a portion of the pigment of the blood-globules. In this manner are produced the livid striæ which follow the course of the subcutaneous veins in the common integuments, the red coloration of the endocardium, and of the internal membranous strata of the vascular trunks, the diffuse reddening of serous and mucous membranes, the red tinge observed in parenchymata and seemingly inherent in their textures. Not only does imbibition pass from one organ to others contiguous,—even fluids, contained within hollow organs, as also in muco-membranous canals and in serous sacs, receive the blood-tinged serum, thereby acquiring the same cadaverous hue; or, again, the blood-tinged serum is found in the said cavities, pure, and unmingled with pre-existing fluids in the form of cadaverous exudations.

The reddening of imbibition is, of course, most readily derived from vessels, the seat of hyperæmia and of stasis; therefore very commonly from the death-patches of the two species above named. The redness of injection characteristic of hyperæmia and stasis merges in, or becomes masked and disguised by, that of imbibition.

Death-spots of this kind are marked by absence of injection; by the obvious cause, namely, blood being discoverable at the point of the deepest saturation, and by the stain being washed out towards the circumference.

Where the previous disease involves liquefaction of the blood-plasma—they are rapidly developed, and they increase in saturation and extent in proportion as, favored by various external influences, cadaverous decomposition gains ground.

It would appear, from the above, that death-spots are, for the most part, stains resulting from a combination of hyperæmia with imbibition.

Amongst the number of imbibition stains, with the character of death-marks, is to be reckoned the yellow tinge imparted to the membranes of the gall-bladder and of the adjacent membranes of the stomach and intestine by the imbibition of bile.

To *qualitative alienations* of color belong more especially, as cadaveric stains subordinate to the above—

1. The original changes of tone in death-stains to blue, purple, and violet, dependent upon the blood-crasis.

2. The brownish and greenish tints, and the dark green dye developed out of the reddening of imbibition, both in the common integuments and in other soft parts, as also in an especial manner in the intestinal membranes and their contiguous formations—namely, the peritoneum, the areolar, adipose, and muscular tissues, and the liver. These varieties of color are produced by certain gases—hydrosulphuric acid and sulphide of ammonia,—evolved in the abdominal cavity, and within the tissues themselves. These gases react thus upon the red pigment of the blood within the tissues generally, and in the muscles most of all.

3. The dark brown, black, green, and ink-black discoloration of the spleen, from its fissured surface to various depths, as also of the ramification of bloodvessels in the fluid sac of the stomach from the imbibition of gastric juice.

4. The more rare violet-red, iodine-colored, diffuse lividity of the intestinal membranes.

Other preternatural colorations, for the most part equally cognizable in the living body, are, in particular—

1. The deep red tinge characteristic of thin watery blood, as also of all the tissues, down to the common integuments, in cases of poisoning with carbonic oxide and carbonic acid gas.

2. The copper-red tint of the skin in venereal stains, and in the circumference of venereal ulcers and skin eruptions.

3. The diffuse sallowness, and the circumscribed freckle-spots, termed liver-stains or ephelides, in cachexia.

4. The violet hue of typhous hyperæmia and stasis.

5. The greenish and yellowish tones of sugillation of the common integuments, arising from deep-seated extravasation of blood.

6. The yellow tinge of the solids and fluids, assuming manifold shades, the most intense of which are a brazen- and a greenish-yellow, engendered by the coloring matter of the bile, where the secretion and excretion of that fluid are intercepted, or where bile mingles with the blood, as in the typhous crasis. It is frequently superinduced by pyæmia, and occurs as the substantive and essential dyscrasy in yellow atrophy of the liver, and probably in yellow fever. As this pigment generally associates itself with the exsuding plasma, the majority of the soft parts, more especially the vascular and succulent—the secretions and incidental products of inflammation—are all dyed yellow.

7. The rust-yellow, rust-brown, black-brown, and black tints of certain organs, resulting from a corresponding granular pigment, partly contained within pigment-cells—a formation, which will, in the sequel, be considered more at large.

8. In conclusion, those anomalous dyes, produced by the assimilation of pigments, or of substances which, either with or without the intervention of some specific influence—light, for example—enter into peculiarly tinged combinations with animal tissues. As instances of the decoloration of both fluids and solids, we may cite the yellow appearance of the urine from the ingestion of rhubarb, the reddening of the bones from feeding upon the root of rubia tinctorum, the yellowness of the skin and of the mucous membranes produced by nitric acid, the swarthy complexion which follows the internal use of nitrate of silver.

CHAPTER VII.

ANOMALIES OF CONSISTENCE.

CONSISTENCE [the normal degree of mutual cohesion and of resisting power pertaining to the elements constituting a texture] is either augmented or diminished. In either case the gradations and also the forms vary greatly, cannot be estimated but in relation to the amount of mechanical violence exerted, and exist, to a certain extent, in combination with each other.

Diminution of consistence is based upon—

1. *Loosening* of the mutual cohesion between the form-elements composing a texture, through the interposition of a fluid or solidified substance. Instances are afforded in the loosening of texture through serous effusion (dropsy), and, in hyperæmia and inflammation, through their products. Such loosening of texture is generally considerable in proportion to the rapidity with which the said products form.

2. *Atrophy*, both primary and secondary, provided the density of the texture be diminished.

3. Liquefaction and breaking down of the elementary forms of the texture, as in suppuration, in gangrene, but most especially and most variously in liquid exudation from mucous membranes, which by its chemical properties proves destructive to the underlayer. Liquefaction of the substance of the liver, through anomalous or intercepted bile, offers another instance in point.

4. Next to these rank the softenings of certain organs, particularly of the mucous membrane lining the stomach, of the lungs, and of the brain, brought about by the resolvent agency upon the textures of a free acid. They represent those processes to which, in conjunction, perhaps, with the foregoing, the term softening ought properly to be restricted.

The abstraction of earthy salts from the bones, in rickets and osteomalacia, belongs to this class.

5. It is brought about by a transformation of textures, the nature of which is most probably a breaking up, with conversion of the chemical constituents ;—for example, the breaking up of the primitive muscular fibrils, or of the texture of the annulo-fibrous tunic of the arteries, in fatty degeneration.

Diminished consistency manifests itself as irregular softening, compressibility, lacerability, maceration, liquefaction, and solution ; or else as pulpiness, putrescence, friability, fragility—the latter property being frequent in the osseous system, in muscles, and in the annulo-fibrous tunic of the arteries.

Increase of consistence varies, in like manner, as to its character and cause. It is based—

1. Upon diminution of the humecting plasma, by which the texture is pervaded (water).

2. Upon hypertrophy. Those augmentations of consistence are par-

ticularly marked which depend upon true hypertrophy without increase of volume, and upon various kinds of spurious hypertrophy.

3. Upon atrophy, the reduction of volume being here accompanied by condensation,—concentrical hypertrophy,—of the brain, for instance.

4. Upon inflammation,—through the solidification and textural transformation of coagulable products;—in other words, by the issue of the inflammation in induration.

5. Upon what is termed ossification, so common in the aforesaid products of inflammation.

Increase of consistence manifests itself as preternatural toughness, hardness, rigidity. Relatively to the normal condition of the textures, it often appears less in the shape of absolute increase, than of a change in the character of the consistence. Thus the friable liver, the kidneys, under certain conditions, in spanæmia, for example, toughen through defibrination of the sanguineous fluid. The increase of consistence is often, moreover, but a seeming one, and even such only with certain restrictions. Thus the organ concerned will exercise, against ordinary external influences, a resistance exceeding the natural, and yet be powerless against more forcible impressions, because, although with increased density it has become harder and firmer, it has at the same time lost its toughness, and become morbidly fragile and brittle. Muscle affords an example.

CHAPTER VIII.

SEPARATIONS OF CONTINUITY.

They are engendered either by external and especially by mechanical influences, or else by various internal causes which may, in like manner, operate mechanically.

To the former belong:

1. Simple or complicated injuries from penetrating mechanical violence, with or without loss of substance;—incised, punctured, contused, gunshot, bitten, and lacerated wounds;—solutions of continuity occasioned by fire and cautery.

2. Imperfect and complete laceration and rupture of solid as also of hollow organs, consequent upon concussive violence,—especially when in the condition of repletion and of distension,—lesions of continuity frequently unaccompanied by perceptible injury to the common integuments and to the parietes of the implicated cavities of the body. The casting of the envelopes of particular organs caused by similar violence, as the separation of periosteum, of the dura mater, from bone, of the fibrous capsules (tunicæ albugineæ) of certain viscera, as of the spleen or kidneys,—is of like significance.

3. Simple and complicated fractures of bones, incurvation of soft, rickety bones, casting of the epiphyses.

Separations of continuity *from internal causes are dependent upon*

various contingencies. Where mechanical influence is simultaneously at work, the two influences co-operate in such wise that where the one predominates, less of the other suffices to produce the effect. Their occurrence may be rapid or slow. They are brought about—

1. By violent exercise of the voluntary and involuntary muscles, lacerating either these or their tendons, or even affecting the bones,—in convulsions, for instance.

2. By excessive distensions of hollow organs, as in laceration of the intestine, of the urinary bladder from accumulation of its contents through paralysis, through mechanical obstruction, stricture, closure, &c.

3. By hemorrhage. Here the lesio continui consists in great laceration, contusion, disruption, destruction of texture ; take, for example, apoplexy of the brain, of the liver, of the muscles ; the forcible separation of the strata composing a membranous organ ; the loosening of the enveloping membrane of organs, of the periosteum from bone, of the tunica albuginea, through extravasated blood.

4. By atrophy. When favored by a mechanical influence it occasions a rapid lesion of continuity in the shape of laceration,—or else, being itself caused by pressure and tension, it serves in the long run to produce lesion of continuity, more especially in the muscles and nerves. Under this head should be mentioned the spontaneous casting of normal and of morbid formations, owing to defective nutrition, as of the hair, the nails, the teeth, horny excrescences, and the like.

5. Separation of continuity is the final result of high degrees of diminution of consistency, especially in true softening, the consequence of textural disease. If, therefore, any mechanical cause be requisite at all, the slightest,—even the degree ordinarily in operation, as for example, repletion of a hollow organ, suffices to produce the effect. Amongst textural diseases, inflammation—from its effects in the loosening of tissues,—and the fatty degeneration of muscular organs, more especially of the annulo-fibrous tunic of arteries—stand pre-eminent.

6. In fine, lesions of continuity are engendered in primary textures, as well as in new growths, by various processes of liquefaction and dissolution, especially by suppuration and gangrene. To this head belongs, amongst others, the spontaneous separation of dead parts, for instance of fingers, of entire limbs, of heterologous products, such as fibroid and cancerous growths. Lesions of continuity are, upon the whole, simple, or else more or less associated with loss of substance. Their cure is effected by the immediate union of the edges or surfaces of the wound, or, in the case last alluded to, by regeneration.

CHAPTER IX.

ANOMALIES OF TEXTURE.

These are the most important of all. They affect the solids and the fluids, especially the blood, in so far as certain form-elements enter as

essential ingredients into its composition, and so far as this general source of nutrition, under particular circumstances, contains and supplies formative matter anomalous in many respects as to its embryonic character and primitive forms, and also as to all its ulterior stages of development. They are commonly connected, in the relation either of cause or of effect, with various other of the anomalies of volume, of consistence, of form and of color already discussed.

Every change of texture being founded in an anomaly of general nutrition, the proximate causes of this anomaly have to be investigated.

As such are directly demonstrable or at least deducible from analogy, —alterations of the blood, as the general fluid of nutrition, and anomalous character of the nutritive process itself. Accordingly, textural disease of the solids is in the one case the reflex of constitutional disease, in the other case a mere local ailment.

Quantitative anomalies of nutrition having been considered under the heads of hypertrophy and atrophy, the present chapter will comprise those textural diseases alone which depend upon qualitative anomalies of nutrition.

Textural diseases may be primitive arrests in the development of the texture—for example, in bones, in muscles, in pigment,—such as we may observe at any time in tissues adapted for regeneration. The majority are, however, acquired during intra-uterine, and more especially during extra-uterine life. In the former case the textural anomaly is congenital.

Textural anomalies manifest themselves—

1. As *new growths*. The most numerous of the class.

2. As a *breaking down of texture*. The disruption involves both the primitive physiological and the new-formed pathological texture,—the latter, by their frequent persistence, at the embryonic stage of textural development, or even in the condition of the primitive rude blastema, are particularly predisposed to this sort of conversion of their elements. To this subdivision belong, besides the reduction of textures in atrophy—besides the breaking up of textures in genuine softening, in particular the liquefaction of textures in various processes of exudation, in suppuration, and in gangrene. And to these are yet to be added other processes which seem to be conversions of various complex substances constituting, now a rude blastema, now a definite texture,—the breaking up of fibrinous, albuminous blastemata, of muscular fibrils, of yellow artery-fibres, with conversion of their elements into fat, &c. They will be considered, together with their attendant circumstances, under the heads partly of general, partly of special anatomy.

New growths, as already observed, furnish forth the great majority of textural affections. For, apart from their mere local characters, almost all constitutional diseases are prone to localization and to the deposition, within an area more or less defined, of products in the shape of blastemata. Even the processes of liquefaction are in part ultimately reducible to a new growth, for example, to an exudation destructive of the texture, to the production of acid, &c. Textural diseases assuming the shape of new growths were formerly distinguished in a manner which here calls for a few remarks. Pathologists discriminated between :

1. Changes (metamorphoses) of textures.
2. Genuine *new* or *heterologous* growths.

With reference to this distinction which, before the adoption of the present auxiliary methods of research was highly estimated, we have to observe that, strictly speaking, the conversion of one texture into another only occurs in isolated instances, which will be hereafter specified. With these exceptions, all conversions are but seeming ones, and consist in the anomalous growth becoming developed betwixt the elementary particles and filling up the interstices of the original normal texture, so as to occasion the reduction and resorption of the latter.

This process of reduction and absorption may be so complete as ultimately to cause the original texture to be altogether replaced by the new growth, which now presents one uniform mass, corresponding in volume with the texture expelled, or even exceeding it,—in a word, constituting a tumor.

But although this process apparently disproves any conversion, it still remains matter for inquiry whether, in a certain sense, a conversion do not take place,—whether the original but reduced texture do not, under the determining influence of the heterologous development, furnish the blastema for the new growth? Several circumstances afford decisive evidence of a metamorphosis of this kind.

As true conversions are to be regarded—

(*a.*) The ossification of cartilages intended for permanent ones; as also of pathological cartilage—of certain euchondromata. Again

(*b.*) The fibrillation of the hyaline intercellular substance of cartilage.

(*c.*) A metamorphosis of muscular fibre into areolar fibrils, such as takes place, in the organic muscles obviously through a splitting of the muscle fibres,—in the animal muscles, probably through fibrillation of the collapsed sheaths of the primitive muscle fibres, after the breaking up and resorption of their contents (the primitive fibrils).

(*d.*) A transformation of the organic muscle-fibres into the annulo-fibrous membrane-texture of arteries.

(*e.*) A transformation of primitive muscle-fibrils, of the fibrous web upon the layers constituting the annulo-fibrous membrane-texture of arteries, into free fat (see fatty degeneration). We are not indisposed to believe in a conversion of hepatic cells into the elementary cells of medullary cancer.

Let us, after this disquisition, turn to the division of new growths:

1. Into organized and organizable new growths.
2. Into unorganized new growths.

I. ORGANIZED NEW GROWTHS.

A. OF ORGANIZED NEW GROWTHS IN GENERAL.

These resemble normal textures, at least in their elementary composition,—and very frequently in the (secondary) arrangement of their form-elements. Where they appear amorphous, the character of the blastema attaches to such amorphous growths. They are occasionally united with unorganized new formations, and that commonly in a con-

secutive manner, the latter supervening upon the new growth, as in the instance of so-termed ossification in the shape of concretion or incrustation.

New growths present great and important differences in relation not alone to the form-elements—especially cell and fibre,—but also to the secondary arrangement of these elements into a texture.

Nor do they differ less widely and essentially as to origin and development. In this respect they often, it is true, follow the laws of cell-formation—cytoblastema, elementary granule, nucleus, cell, fibre. The field, however, is equally extensive of fibrillation out of nuclei and granules, and especially that of the independent development of fibre directly out of solid blastemata, intercellular substance, primitive structureless membrane, and membranaceous coagula. Compare, with reference to this and to what next follows, *Blastemata.*

With reference to the grade of development attained by their elements, new growths are classed, if such a classification be feasible, considering the variety of elementary bases coexisting in a single growth, and the want of uniformity in their ulterior development, as follows:

1. Such as exist in the condition of formless liquid, or at that phase of coagulation—the consolidating blastema. They are susceptible of further development, or they abide at this stage, many ultimately breaking up. They comprise some very malignant new growths—for example, tubercle.

2. Such as attain to nucleus and cell-formation only,—perhaps to fibre or caudate cells. They consist of isolated cells within a fluid, semi-fluid, intercellular substance (pus; colloid, encephaloid substance); or, again, the cells are imbedded in a paucity of firmer, amorphous intercellular substance, which acts as a bond-mass. Along with them are numerous nuclei and elementary granules—embryonic formations readily broken up. Some of the most malignant new growths are thus constituted.

3. Such as have their texture represented by fibres of different kinds, variously arranged, and arising out of cells, nuclei, elementary granules, or directly out of blastema. To this class belong many quite benign, and a few eminently malignant formations: for example, fibrous cancer.

4. New growths, which, in their full development, consist of fibres, cells, nuclei, blastema, although the disposition and the mode of development of these elements may greatly vary. They include new formations, both benign and the reverse. The progressive development of the form-elements is accompanied by a succession of chemical changes. Under every mode of development the reactions vary with every phase, from the primitive blastema to the completion of a texture, the difference between the perfect texture and the primitive blastema being very marked indeed.

With reference to the development of bloodvessels, new growths are either *vascular* or *non-vascular.* The former present every gradation, from poverty in bloodvessels to luxuriant vascularity. Nor does the number of its bloodvessels stand in any direct relation either to the bulk or volume of the new growth, or to the stage of its development in other respects. Accordingly, there are, on the one side, new formations of

very considerable magnitude, which, devoid of all bloodvessels, vegetate freely in the cavities of the body—for example, the frequent fibroid concretions in serous cavities, certain cancers, &c. On the other side, there are blastems in which blood and bloodvessel-formation so predominate that the new growth consists of little else. (See *Pathological Bloodvessel-Formation.*)

With reference to their state of aggregation, new formations are either *fluid* or *semifluid*,—for example, the plasma containing dropsical fluids, pus, ichor, encephaloid fluid, colloid, the gelatinous substance of collonema, of areolar cancer, &c., or they are solid.

Not long since there existed a classification of new growths, which, though not altogether available, is nevertheless deserving of mention—namely, their division into *homœoplasiæ* and *heteroplasiæ*. The former (according to the earlier views of Meckel) are repetitions or imitations of normal textures, the latter alien to the normal composition of the organs and textures. With respect to this division, it is to be observed, that:

(*a.*) The chief argument against the assumption of heteroplasiæ is afforded in the evidence recently obtained, that all new growths essentially imitate normal formations, not alone in their origin, development, and growth, but also in their chemical composition.

(*b.*) Even the secondary arrangement of their textural elements, that is, their coarser texture, very frequently offers analogies with normal textures. Finally, their general aspect exhibits to the naked eye analogies which formerly served for the basis of certain classifications and denominations. Take for example the comparisons with various glandular structures made by Abernethy and others.

(*c.*) It might appear from the above, that homœoplasiæ alone existed. Still, in many heterologous formations the external aspect, the structure, and even the textural elements, especially cell and fibre, differ, not only in themselves, but in the progress of their development and in their chemical composition, so materially from the normal type, that the existence of heteroplasiæ cannot be altogether rejected. As regards the relation of new growths to normal texture, it should be stated that,

1. The heterologous formation lodges more or less uniformly between the elementary parts of a texture, the latter becoming infiltrated. The mass (and commonly the volume also) of the organ increases—*false hypertrophy.*

2. Where, on the other hand, the heterologous formation is developed and increases from an interstitial point, or even from an originally circumscribed infiltration, so that at its circumference it rather displaces than involves or embraces the elements of the affected organ, and so that its periphery becomes more or less sharply defined, it forms an individual independent heterologous mass, termed a tumor.

The distinctive characters of the two are, however, by no means strongly marked. Akin to the above division is another, almost essential to the medical practitioner, however little tenable in a scientific point of view, namely, into benign and malignant new growths (benign and malignant tumors). The connection between the two classifications consists in this, that, with certain exceptions, homœoplasiæ appear to

and actually do answer to the character of benign, whilst a grade of malignancy may be predicated of a new growth proportionate to the degree of its heterogeneous nature. In this classification it is essential to determine,

1. What constitutes a benign, what a malignant new growth?
2. What are the distinctive marks of the one and the other?

In the first place, we would signify by malignant new growths, those the origin and continuance of which either are essentially bound up with, or else eventually lead to, a definite constitutional dyscrasis, a general disease giving rise to a peculiar impairment of nutrition, and a multiplication of specific new growths. No new formation is, therefore, in itself malignant, but becomes so either through a specific, pre-existent, and predetermining, or through a consecutive, general dyscrasial affection. This is perhaps the proper explanation of a malignant new growth. It does not preclude that occasional purely local relation of a malignant formation upon which the cure of the latter, spontaneous or artificial, often depends. It will be seen that whatever else is adduced as an attribute of malignant tumors ceases to be distinctive.

(*a*.) It is very difficult to recognize a constitutional disease as a definite one reflected in a new growth, and to discriminate between this and a cachexia engendered by the luxuriation and ichorous vent of a new growth essentially local, and pronounced benign. Besides, the constitutional affection may as yet be altogether wanting. Even where several growths coexist of the same character, or rapidly succeed each other, they need not necessarily be based upon any general dyscrasis. They may be simply so many mere local occurrences.

Certain other characteristics are indispensable for a diagnosis, and, at the same time, difficult to establish: for example, that—

(*b*.) Benign growths are curable by extirpation, whilst the malignant recur at the same spot, or at other spots, or even at both.

In opposition to this, it is to be urged, that many benignant new growths recur after extirpation, where the disposition to them remains, whilst, under certain conditions, many a malignant new growth does not recur, but enters upon a spontaneous process of retrogression, and becomes extinct.

(*c*.) Malignant new growths have a marked tendency to draw within their formative range—to convert to their own similitude—contiguous and neighboring textures.

It is to be observed, on the other hand, first, that the most malignant growths thrive and flourish as independent tumors upon a new-formed vascular apparatus of their own, without otherwise molesting the surrounding textures than by forcing them from their positions; secondly, that where the original normal textures merge in the heterologous growth, this is brought about in the malignant, precisely as it is in the benignant ones—namely, by a conversion in the sense before adverted to; that is, through reduction, disintegration, and resorption of the normal textural elements.

(*d*.) When malignant growths have attained their highest point of development, they break up and enter upon a process of softening, which

implicates or involves surrounding and included textures, and thus serves to exhaust the organism.

In connection with this process, the following subjects for consideration suggest themselves : namely,

1st. This so-called stage of metamorphosis—this breaking up very frequently fails to occur, even in the most malignant new growths.

2d. That apart from the general difficulty of establishing the epoch of the highest development of a new formation, the act of breaking up should seem a fortunate event as regards the growth itself, which, by virtue of the elementary transformations thereby engendered, becomes deprived of its importance, and in many instances is excreted from the body.

3d. That this metamorphosis for the most part simply implies inflammation terminating in ichorous degeneration, and death of the new growth.

4th. That this destructive process frequently attacks the surrounding textures merely in the character of suppuration, and that, as such, it may, whether based upon a benignant or upon a malignant new formation, either exhaust the organism, or, on the contrary, lead to a cure,—to the expulsion of the heterologous product.

5th. That where a fresh development of heterologous substance is excited and kept up in neighboring parts by inflammation connected with sustained ichorous secretion, the malignancy of the growth may be at least strongly suspected.

(e.) Malignant new growths are said to abound in albumen and casein; benignant new growths, in fibrin and gluten.

Were such a distinction of new formations not rendered nugatory by the convertibility of those organic substances, it would become in a great measure deprived of its value by the number and weight of the exceptions,—for example, those of fibrinous tubercle, of fibrous cancer, the composition of which is marked by a considerable amount of gluten, &c.

(f.) Homœoplastic formations are, for the most part, benignant, heteroplastic growths malignant.

Supposing such a distinction admitted, its utility would still be doubtful, seeing that in a given case the decision frequently depends upon the method followed in the examination, and upon individual opinion; and again, that in many new formations, homœoplasia and heteroplasia coexist in various gradations. For the more marked repetitions of normal textures, namely, areolar, cartilaginous, osseous new growths, the character of benignancy might indeed be predicated; whilst, on the other hand, certain forms of *malignant* fibrous cancer bear so close a resemblance to the *benignant* fibroid new growth as to set discrimination at nought.

Microscopic analysis, therefore, from which important disclosures in relation to the diagnosis of benignant and of malignant growths, and tenable grounds for the establishment of a system were expected, has in reality thrown but an uncertain light upon the subject.

Certain new growths are especially intended for the more or less perfect restitution of loss of substance, howsoever occasioned. These regenerated textures are sometimes perfectly identical with the lost ones, in formal and chemical composition, as also in function; sometimes entirely

dissimilar. The latter kind are represented in scar-texture, which, again, may have an evanescent existence, as in *provisional cicatrix*, out of which is developed, and which merges in, a texture identical with the lost one; as, for instance, the fibroid scar-texture that ensues upon loss of substance in the bones of the skull; the scar-callus occurring at the point of a lesion of continuity in a nerve. Or again, the cicatrix may be permanent, consisting throughout of a fibrous texture of various degrees of perfection, in which the elements of the lost texture are never reproduced; such is the muscular, the glandular cicatrix.

New growths once established either sustain themselves without alteration of bulk, or else wane and shrivel in various ways, or even disappear altogether. Products of inflammation, even such as have assumed a textural character, unquestionably become reabsorbed; so, in like manner, do new growths of embryonic structure.

Again, they liquefy under various transformations of their chemical components, or they become diseased in manifold ways.

Finally, new growths increase. This increment takes place through juxtaposition; that is, through the accession of blastema upon the periphery of the existing structure. Such is the growth of non-vascular formations, especially of those which do not rise above the lowest grade of development; as, for example, tubercle. Or the increase takes place through the intussusception of new blastema from those bloodvessels of the diseased organ which supply the new growth, or from an adventitious vascular apparatus newly developed for the supply of the new formation. Finally, an increase of volume may be based upon the variety of chemical conversions attending the development of textural elements out of blastema, and, in vascularized heterologous products, attending the growth of those elements themselves.

Growth and intrinsic development by no means keep pace with, but rather stand in an inverse ratio to, each other. Rapidly vegetating heterologous growths are mostly distinguished by an embryonic structure.

Upon the rapidity of its growth depends, in a great measure, the degree of influence exercised by the heterologous product upon the affected organ, upon its vicinity, and upon the organism generally.

This influence, considered locally, consists in pressure and tension of textures and of entire organs; in displacement and extinction of textures; consequently, in the production of pain, and in embarrassment or complete hindrance of function.

The influence upon the entire organism is sometimes deducible from that which is local, shaping itself differently according to the different seat of the new formation. In the instance of heterologous products luxuriating by growth and multiplication, this influence consists in causing the wasting of organic matter and of power, or in the establishment of a consecutive dyscrasial state. This latter may be brought about in a twofold manner:

(*a.*) Either through the withdrawal from the fluid of nutrition of some particular substance employed as a material in the heterologous structure —as in defibrination of the blood and œdema in tubercle—in dropsy consequent upon albuminuria.

(*b.*) Or else in a positive manner, namely, through reception into the

blood and lymph of substances generated in the interchange of matter that constitutes the nutritive process of the heterologous product, and still more through reception of the heterologous matter itself, in the shape of intercellular substance, or of elementary cells, and the like. This directly leads to contamination of the fluid of nutrition, and thereby to a dyscrasy reflecting the character of the heterologous growth. It is the more speedily brought about where circumstances are generally favorable to endosmosis or resorption, and particularly so in the locality of the heterologous growth, where this latter is bulky or highly vascular, or situate in organs rich in blood and lymph-vessels, where its mass (its intercellular substance) is more or less fluid. It occurs, however, in heterologous growths, both solid and poor in bloodvessels, when their texture has become disintegrated and liquefied by hyperæmia and inflammation. To sum up, new growths possess sometimes a general, sometimes a local character. Nay, one and the same new formation may, at various, successive periods, acquire now the one, now the other character. A growth, originally of general import, may in particular assume a local one instead.

New growths vary considerably as to the organs and texture which they affect by preference; each possessing, in this respect, a scale of frequency of its own. Some organs are pre-eminently subject to one particular kind of new formation.

Certain new formations become developed and subsist unmistakably in concurrence; certain others never cohabit, the presence of the one serving to exclude the other,—the appearance of the one arresting the development of the other. Exclusiveness or repulsiveness of this kind is, as might be expected, mutually evinced by new growths based upon dyscrases of opposite characters. On the other hand, new formations rooted in kindred dyscrases, do exist confederately, and purely local new formations enter into every phase of combination.

Let us now turn from the consideration of confirmed new formations to that of their blastema and of its metamorphoses.

OF BLASTEMA AND ITS METAMORPHOSES, WITH AN ESPECIAL REFERENCE TO FIBRINE.

The blastema for pathological new growths ultimately proceeds from the general fluid of nutrition, the plasma of the blood. Accordingly, its source is that out of which all normal textures are developed. Its bodily detection and demonstration in its simple, primitive form, are, however, mostly a matter of difficulty, except, perhaps, in cases where it is somewhat copiously produced, in the train of peculiar and often rapidly fatal processes, which may be experimentally analyzed grade for grade,—for example, in inflammation and hyperæmia. It exudes through the parietes of vessels wherever capillaries exist, or it appears as an endogenous segregation from the blood within the circulating system. In rarer instances, it is deposited by extravasation out of lacerated vessels.

The blastema is originally fluid, and it may either abide in this condition or solidify. The earlier or later solidification, that is, its becoming

a fixed elementary body, and the degree of the resulting density and consistence depend mainly upon the presence of coagulable protein, and upon the degree of its coagulability, as also upon the absence of those counter-checks to coagulation, alkalies, acids, and certain salts.

Rapidly solidifying blastemata, especially when products of inflammation, are very commonly termed plastic,—improperly, however, because coagulability of the blastema stands by no means in any direct relation to the faculty of development. Many blastemata, distinguished for their coagulability, do not rise above the lowest grade of form-development, and not alone do they stop at the grade marked out by the process of coagulation, but their ulterior tendency is to liquefy. An example is afforded in tubercle.

As a fluid, primitive blastema recently secreted is amorphous. Sooner or later, however, it is marked by the development of form-elements, in the shape of molecular granule, nucleus, cell. Solidified blastema is either at the outset amorphous, or displays, from the moment of coagulation, certain, and, indeed, higher elementary forms,—more especially fibrillation.

The blastemata are colorless, or they assume the tint of the plasma, or they are of a reddish gray,—the fibrinous of various tones of yellow, —the albuminous, whitish, particularly when fat enters simultaneously into their composition,—or they display various shades of red from adhering blood-pigment, or from the presence of blood-globules, &c.

Chemically considered, all blastemata for pathological new growths are protein compounds, for the most part in various degrees of oxidation.

The main conversion which the blastema undergoes is its development into textures. It is capable, however, of abiding in its rude primitive condition—of remaining dormant—or of breaking up, or lastly, even of becoming reabsorbed.

Before we proceed to consider these several attributes of blastemata, it seems desirable for us to render ourselves familiar with the main conditions for its development or non-development.

If, participating in the current opinions as to the conditions necessary for the development of blastema, we admit

(*a.*) A faculty of development originally and essentially inherent in the blastema, and inseparable from the idea conveyed by the term.

(*b.*) The necessity of certain outward and general conditions, particularly a mean temperature, the presence of water (moisture) and of oxygen.

(*c.*) *The necessity of extant life in the textures* into which the blastema is effused, and *à fortiori*, in the individual. In necrose textures no development takes place at all.

(*d.*) The necessity, in order to become developed, that the blastema should abide in close contact with the living textures; for beyond this the influence of the vital power certainly appears to be limited. The development of blastema usually commences close to the living textures, and bulky effusions of blastema remain in a backward state when removed from these textures lingering either in their rude primitive condition, or at the stage of form-development, determined by coagulation, or lastly, breaking up.

(*e.*) The specific influence exerted by circumjacent textures upon the mode of development, and upon the form of blastema. We know that,

in the act of nutrition, of regeneration, even in pathological processes, blastema in areolar tissue becomes developed into areolar tissue; blastema in serous membranes into areolar tissue, nay, even into serous layers and sacs; blastema in bone, into bone; we know that, in tumors, fibroid textures often imitate the texture of the organ; that fibroid tumors of the uterus, for example, represent the elementary forms of organic muscular fibre;—that in bones, cartilaginous new growths are wont to assume the form of enchondroma.

All this generally admitted, the failure of such influence does not, as regards many, and the more momentous cases, appear satisfactorily to explain either the non-development of blastema, its tarrying in its rude primitive condition, its arrest at an inferior stage of embryonic development, its disintegration, or its development to unwonted heterogeneous textures. This becomes the more obvious if, in relation to the aforesaid conditions, we reflect:

(*a.*) That, as a rule, the absence of moisture is not absolute, and that it is also in other ways conditional.

(*b.*) That the absence of an adequate, general, and specific influence in the circumjacent textures can never be assumed directly, but only through the one-sided conclusion that, notwithstanding the existence of other requirements, a blastema has failed to become developed, a fact which might admit of a very different interpretation.

Thus, to discuss a matter of the greatest importance in the arena of facts, the sojourn of certain fixed blastemata,—for example, tubercle—in the primitive condition, is not ultimately referable to the absence of moisture; the absence or rather paucity of water depending upon the high degree of coagulability proper to the said blastema. This coagulability must, however, be inherent in the blastema itself. Again, there are blastemata which never get beyond the embryonic grades of development,—for example, the pus-blastema, the blastema of medullary and of colloid cancer. Deficiency of vital power, or of determining influence on the part of surrounding textures cannot, in every instance, furnish grounds for the non-development of blastema. Thus we see very minute portions of blastema,—for instance, of tubercle—in robust individuals, in the closest contact, with, nay, in the centre, of vigorous textures, undeveloped. On the other side, in a very low degree of vital power, where one might rather expect little or no blastema to be produced, we meet with enormous masses of it under various forms of heterologous growths, engaged in the process of development. The phenomenon so commonly regarded as an arrest of textural development,—founded in deficiency of vital energy, of adequate working power,—namely, fibrous callus, in the regeneration of bone,—cicatrix in muscle, &c.,—is, we think, interpretable as qualitative alienation, the blastemata not abiding at the embryonic stages of development of the textures to be regenerated, but forming into other textures, perfect after their kind.

Still less is this *deficiency* calculated to illustrate that qualitative variety in the development of blastemata, exemplified in so many heterologous growths. How should we therefrom apprehend the derivation of a cyst, of an areolar-carcinoma, and the like? How often should we not be driven, instead of *deficiency*, to assume an equally unintelligible

excess of power, where we find, in textures of an inferior grade, new growths developed, the elements of which belong to textures of a higher order.

These remarks of themselves lead to the conclusion:

1. That the abnormal development of the blastemata is founded, not in a deficiency, but in an anomaly, of determining influence.

2. That the different blastemata themselves, at the outset, possess indwelling properties of their own. We can have little hesitation in establishing, as a basis of the doctrine of new formations, a native anomaly in the blastemata, this being practically demonstrable. Such, for example, are the various morbid relations of protein substances, and in particular the anatomically demonstrable anomalies in the constitution of fibrin in the blood itself, with which anomalies the different exudation, (as blastemata) correspond both as to form and chemical composition.

In this manner certain blastemata bear, in their primitive character and composition, the grounds for their non-development,—the seeds of their dissolution,—for example, croupous fibrin,—tubercle,—pus-blastema.

Other blastemata, on the contrary, possess the indwelling faculty of development in so exalted and inextinguishable a degree, as to form, in large serous cavities, into free aggregations of blastema without any abiding contact with textures—free fibroid concretions.

Areolar new growths are so frequent, simply, in our opinion, because their blastemata are so frequent, and their production consequent upon so many different processes of exudation.

The blastema for animal muscular fibre appears, on the contrary, to exude only in the normal process of nutrition, or where this process is exaggerated to hypertrophy.

It is very common for mixed blastemata to exude. Hence the frequent coincidence, in one and the same new formation, of such various elementary forms, and of such different modes of development.

Primitive anomalies of blastema may be occasioned in a twofold manner:

(*a*.) They may be rooted in a general dyscrasy of the sanguineous mass. The effusion of blastema coincides with manifest anomaly of general nutrition. The blastema bears the impress of dyscrasial adulteration. This is particularly the case with blastemata deposited in mass as inflammatory products. Indeed, the copious production of blastema in reduced, enfeebled subjects, admits of no other conclusion than that of a dyscrasial condition as the source of such effusion; the specific character of the latter being simultaneously manifested by its obvious preference for particular organs.

(*b*.) Again, the said primitive anomalies may, where the general crasis is untainted, be based in an altered admixture of the plasma (the blood) owing to local changes of innervation; or else in an anomalous act of nutrition, for example, inflammation.

In the former case, the blastema is a symptomatic manifestation of a constitutional disease, and is of general—in the latter case it is of mere

local—import. The same blastema, pus, cancer-blastema, for instance, may, in one case, imply general, in another, local disease.

A new growth would, however, be equally of local significance if it resulted from a blastema of originally normal character through an anomalous determining influence on the part of the nerves or textures, or of an anomalous interchange of matter (resorption, &c.)

Amongst the many causal relations, the mode of operation of which is unknown, mechanical influences are by no means the least frequent originators of anomalous blastemata through local changes of innervation and of textural influence.

We shall now leave this discussion, and proceed to an inquiry concerning the *metamorphoses* of blastema. Amongst these, the first rank is taken up with the development of blastema into textures. Here *solid* blastemata, as the groundwork of pathological new growths, present so great a difference from *fluid*, that the two must, as far as possible, be separately considered.

The latter are all developed according to the laws of the cell theory, whilst the former disclose, besides a variety of granule and of fibro-nucleus formations, a direct, and, for the most part, preter-physiological development, in various ways, to higher elements in the shape of fibre.

Blastemata of this description are represented by coagulated fibrin and coagulated albumen. In the identity, however, of the process of development in both, fibrin is pre-eminently adapted for experimental study, owing to the greater frequency of its occurrence, especially in an aggregate and bulky form. Under certain conditions, its coagulation alone suffices to constitute textural formation. We shall, therefore, do well to preface an inquiry into the nature of solid blastemata by the study of coagulate fibrin in its relation to the doctrine of exudation and of blood disease.

COAGULATED FIBRIN.

The simple coagulum met with in the heart or great vessels after death, and in blood drawn from bloodvessels during life, furnishes the chief groundwork for this inquiry.

These coagulations, which vary essentially, both as to external appearance and to elementary constitution, form the basis of the different qualitative fibrin crases. We should, however, begin by stating that the individual forms seldom, if ever, occur in their perfect simplicity, owing both to the mutable nature of the substance, and to the fibrin not becoming throughout equally influenced by the disease. This corresponds to the numerous exudates composed of differently constituted fibrinous materials, as also to the frequent impurity of blastemata in general.

The more important forms, described from the most perfect specimens, are as follows:—

1. Fibrin taken from the dead bodies of healthy individuals, presents tolerably compact and tough, moderately adhesive coagulations of a yellowish white. These are separable into membranous layers, and their torn surface exhibits a delicately villous character. Viewed under the microscope, they display a transparent basement, capable of membranous expansion, or else stratified. Upon this is a dense felt, freely

erect at the edges of the preparation, and consisting of very minute, very elastic, ramified fibres, visible in black outline, and rapidly soluble in acetic acid. Hard by on the preparation, are seen numerous round polished nuclei, which, when treated with acetic acid, are brought more distinctly into relief. Beside these lie scattered minutely granular, dull, round, and elliptic nuclei, and similar cells, the size of pus-cells, colorless blood-globules, lymph-globules (fibrin-globules, according to *Mandel*), the same form-elements which, in exudates, are termed plastic corpuscles (Bennett) exudation cells (Henle). (See also Pappenheim, Addison, and others.)

The soft, jelly-like coagula of so-called *spurious fibrin*, found to accompany the above-mentioned compact coagula, and in certain cases to constitute the whole of the impoverished fund of fibrin, show the same composition. They constitute, we think, a transition form from albumen to fibrin, of great moment in relation to the normal process of nutrition.

2. Fibrin the coagula of which, though of the ordinary appearance, possess the property of adhesiveness in a more marked degree, and frequently inclose not inconsiderable quantities of serum. Examined with the microscope, they present a laminated basement, and one splitting into fibres, flattened or roundish, rough, and firm, or resembling organic muscular fibres; or else a membranous basement invested with delicate wavy fibres, upon which, amongst elementary granules, are seen numerous round, black-edged nuclei, sometimes rod-shaped, or drawn out into fibres, and again, more especially in the moisture poured out, dull, round, and oval nuclei, and analogous cells. This fibrin enters—along with rudiments of the preceding one—into frequent combinations with those about to follow.

This and the preceding fibroid together furnish the basis of numerous areolar or fibrinous new growths, whether simple or combined with other blastemata, both within and without the vascular system; textural development having set in with the process of coagulation itself. The exudation of the last specified form of fibrin is especially wont to accompany morbid processes; for example, inflammation, and frequently in considerable abundance. It might be designated, for distinction's sake, as *plastic* or *organizable fibrin*.

3. Fibrin, the coagula of which are marked by opacity, and by a dull-white aspect shaded with yellowish, or with yellowish-green. They frequently include, besides blood-serum, blood-corpuscles in considerable amount, thus giving proof both of augmented coagulability, and greater rapidity of coagulation. They are opaque, and of various shades of red. Microscopically examined, the coagulum presents a stratiform or fibro-laminated basement, or else a faintly striated membrane, both being, however, opaque, owing to delicate granulation (Punktmasse). Upon this, as also in the serum, are seen a vast number of nucleus-like formations, of developed, dull granulated nuclei, and of similar more or less developed cells. Frequently the coagulum appears to consist altogether of the two last-mentioned elements, with a proportion of granulated structure. The nucleus-formations all manifest the usual neutral relations towards acetic acid. This fibrin possesses little adhesive property.

4. *Fibrin* presenting in a higher degree the morbid characters manifested in the preceding variety. The coagula are extremely opaque, and, where they inclose no blood-corpuscles, of a more marked greenish-yellow tinge. Frequently, however, they do inclose vast quantities of blood-corpuscles, and are of a reddish-gray or a reddish-brown, denoting rapid coagulation. Upon a closer examination, they are found to consist of a compact, delicately granulated mass of nucleus and cell-formations (assimilating in various degrees to the pus-cell and pus-nucleus), all held together by a tough amorphous intercellular substance. There is neither fibrous tissue nor any fibrillation. These coagula possess still less of a cementing property.

These two latter forms we would designate as *croupous fibrin*. Here the fibrin borders upon that in pyæmia, and has the croupous character. The cells and nuclei included in the coagulum are genuine pus-nuclei and pus-cells. Other morbid conditions of fibrin—for example, the milky white opaque fibrin—are of little moment as regards the present subject. They will be considered under the head of Crases.

These forms of fibrin possess, from the very first, an indwelling proneness to textural formation, and a disposition to molecular disintegration —nay, they have already entered upon both the one and the other transformation. The fibrin 1 and 2, are organizable; the fibrin 3 and 4 suffer disintegration; portions of the fibrin 1 and 2 that mingle with it being alone susceptible of textural transformation, as is so frequently witnessed, extraneously to the vascular system, in exudations of a kindred stamp. Fibrin 4 presents no definite coagulum at all.

These forms of fibrin correspond in some measure with Mulder's gradations of the oxidation of protein. Here, however, chemical analysis has assuredly not kept pace with anatomical facts.

Coagula assuming as it were the form of intercellular substance, are liable to both kinds of metamorphosis. The differently apportioned nucleus and cell-formations here play a subordinate part, their importance varying, as has been stated, from the nucleus employed in the fabric of textures, to the true pus-nucleus and pus-cell. Hence they are the manifestation either of a quantitative endogenous development of textural rudiments, or else of a qualitative affection of the plasma.

1. The structural transformation comprised in the process of coagulation, consists in the afore-mentioned diverse fibre-and membrane-formation. The nuclei themselves, sometimes appear elongated into rod- or perhaps spindle-shaped fibre-stems. In the cells the caudate form of development is seldom observable.

2. The second metamorphosis is disintegration. It is foreshadowed in the granular mass that enters into the coagulation. After probably a brief interval, the entire coagulum resolves itself into a pulpy, cream-like, whitish, or yellowish-white, or, if containing blood-corpuscles, into a proportionally faint reddish-gray, reddish-brown, or chocolate-colored liquor, pregnant with granulated substance along with the nucleus and cell-formations originally admitted into the coagulation, and becoming, where the latter are numerous, relatively analogous to, and where they assume the character of pus-nuclei and pus-cells, identical with, pus. This breaking down may, under certain external conditions, unfavorable

to textural formation, or owing to some indwelling peculiarity, affect fibrin generally. In croupous fibrin it is of unfailing occurrence. The results of Gulliver's experiments concerning the liquefaction of fibrinous coagula, under the sustained influence of the animal degree of heat, out of the animal body, are not applicable to the process as occurring within the living body, where certain kinds of fibrin of necessity become converted into textures, whilst others as invariably liquefy.

This process is witnessed with especial frequency in the coagula occurring within the heart, and which Laennec designated as "végétations globuleuses," as also in the coagula occurring within bloodvessels, both great and small.

Liquefied fibrin is capable of undergoing inspissation and cretaceous conversion.

Other transformations of fibrin are:

3. The abiding of the organizable fibrin at the primitive stage of formation, and its eventual extinction. Here the coagulum is, with loss of its moisture, reduced to a compact, unyielding, semi-translucent, or opaque and horny substance. It is capable of eventually ossifying.

4. *Fatty conversion*, in the shape of a reduction to fat-molecules of various circumference, a metamorphosis which coagulate fibrin shares with liquid and coagulate albumen.

5. Within the vascular apparatus solidified albumen, of whatever form, may again become incorporated with the circulating fluid. Where this liquefaction of the coagulum is not the consequence of inherent disposition, it is wrought by gradual solution in the plasma, becoming, so to say, corroded, layer for layer, by the liquor sanguinis. Examples offer in the progressive resolution of solidified vegetations upon the heart's valves, or of the thrombus in arteries. It corresponds to the resorption of the consolidated fibrin of exudation and of extravasation.

We have hitherto expressly restricted ourselves to an inquiry concerning the consolidation and the metamorphoses of fibrin *within the vascular apparatus*, as exemplified in the diverse spontaneous coagulations which occur in the heart, not rarely during life; and again in coagulations within the larger vessels (more especially the veins), and also in the capillaries.

The relations of the fibrin of exudation are precisely the same.

The organizable nature of the fibrin of exudation might be confidently assumed a priori; it is, however, as shown under the head of hemorrhage, directly demonstrable by facts.

Contrasting the frequency with which solid blastemata constitute the basis of pathological new growths, with their rareness in the physiological condition; reflecting, at the same time, upon the predominance of cell-development in physiological structures; and lastly, upon the absence of fibrin in the embryo, we feel somewhat disposed to concur with Zimmermann, in regarding fibrin as a genuine excretive formation; a substance carried by oxidation to the verge of disintegration,—albumen worn out by oxidation, and associated with albumen for the purposes of nutrition, only in the shape of pseudo-fibrin.

METAMORPHOSIS OF BLASTEMA.

1. Textural development.—Organization.

Solidified Blastemata, at their very development, either constitute various pure and unmingled new growths, or enter in the shape of inter-cellular substance, basement- and bond-mass, as the stroma into the composition of complex heterologous structures. Their development is, for the most part, foreshadowed in the types cast in the process of coagulation, and which were partly discussed in the foregoing chapter.

The principal abiding form-element that enters into the composition of new growths is the *anastomosing, delicate fibrous network* of consolidated fibrin. This, together with a hyaline intercellular substance, speckled throughout with shining nuclei, we have seen in old inflammatory indurations in the brain, as also composing an extensive fibrous cancer in the stomach.

Solid blastema either appears originally as a compact mass, or else takes up a considerable amount of moisture, and establishes a sort of skeleton-work with variously shaped gaps, offering a specific type of much interest. From a central mass, namely, arises a trelliswork, the rods of which are sometimes isolated, sometimes anastomose with each other, constituting a network with largish, and for the most part, oval meshes. This type characterizes in particular the opaque accumulations found upon the internal coat of arteries, as also certain fibrous tumors, especially when seated upon the dura mater. Or, again, solid blastema assumes the form of a *membrane*, either superficially spread out, or folded and rolled up in a tubular form,—a *cylindrical* fibre.

The blastema is here amorphous, laminated; or it presents upon laceration, a striated, fibrous aspect. It may assume, owing to the presence of elementary molecules, various degrees of opacity, or a granulated look; or it may display crystalline clearness. Lastly, it may or may not include nuclei and cells, in various proportions.

Solid blastema of each of the specified forms is worked out into fibres by splitting:

1. Either *directly into areolar fibre and fibril*, or else

2. Into flat, riband-like, rough-surfaced, jagged, or into roundish, oval, mostly felt-like fibres, of from $\frac{1}{100}$ to $\frac{1}{85}$th of a millimetre in their broad diameter.

3. *Into fibres perfectly identical with those of the organic muscles.*

By renewed splitting, for the most part commencing at their ends, the two latter kinds may give rise indirectly to *areolar fibrillation.*

Where there are nuclei present, engaged in the development into oblong nuclei, the splitting takes place in the direction of their longitudinal axis.

The aforesaid *cylindrical fibres* represent little tubular bodies of from $\frac{1}{100}$th to $\frac{1}{10}$th of a millimetre in diameter, which end either in bulb-like dilatations, or in sharp points, frequently inosculate, and constitute a wide-meshed villous network. Their parietes are formed by a transparent, structureless, often wavy membrane, in a single or double fold. Their contents are elementary granules,—in hemorrhagic blastema, pigment-granules additionally,—nucleus formations, cells, together with an ampho-

rous blastema in varying quantity. Cylindrical fibre at its parietes becomes areolar fibril, or perhaps this fibrillation is first developed in the blastema within the canal, as a delicately fibrous wave-curled axiscylinder. Such fibres are commonly coincident with the primitive forms of blastema, more especially with the trelliswork described. We have frequently examined them, and we regard them as analogous with the cylindrical formations occurring in fluid blastemata (Engels germ-tubes.)

Other kinds of fibre arise directly out of splitting, but more slowly, it would appear, and only after the blastema has entered upon essential chemical changes (as partial, glutinous, or horny conversion). They are characterized by their neutral relation to acetic acid, or at least by their stubborn resistance to its influence. Through progressive transitions they ultimately attain to uniformity with elastic and nucleus-fibre with which they further accord in blackness of outline, in solidity, and in elasticity. Of this nature are

1. *A transparent fibrillation for the most part solid*, the fibre varying in diameter from that of the areolar fibril to one of undefinable minuteness.

2. A fibrillation in black outline, vibrating *in lengthy deviations*.

3. *A twig-like fibrillation arising out of a short stem*, with black contours.

4. *A fibre-felt*, resembling the intercellular substance of reticulated cartilage.

The appearance in the blastema of *roundish gaps*, created by resorption, is likewise deserving of notice. In this manner solid masses of blastema acquire a *porous, honeycombed aspect*, whilst membranous blastemata become pierced or loopholed tunics. This does not, however, prevent the blastema either from remaining amorphous or from undergoing fibrillation. Amongst the elementary granules, nuclei, and cells which occur in various number in solid blastema, it is more especially the two former, and *most* frequently the nuclei, that undergo further elaboration.

1. Even in recent fibrinous coagula, within the vascular system, rodlike *nucleus* formations are discoverable. They enter into the composition of many delicately fibred textures.

2. The nucleus is developed through the oblong form to the caudated nucleus, and from thence directly into nucleus-fibre. Upon basement membranes, we often meet with serpentine, creeper-like nucleus-fibre stems. The caudated nuclei often constitute, when held together by an amphorous intercellular mass,—in rarer instances independently,—the fibrous element of not a few heterologous growths. More frequently, however, they enter singly into the composition of fibrous textures of other kinds.

3. Contiguous nuclei, in progess of fibrous development, conjoin and merge in the *varicose nucleus-fibre*, which by degrees acquires uniformity, and in rare instances forms the main component of fibrous new growths.

4. The nuclei form the basis of the *true elastic* splitting fibre.

Elementary granules forming in collateral array, become confluent, and establish in various directions, more or less delicate, dark-looking, longitudinal, or reticulated fibres, which resist the influence of acetic acid. They are most conspicuous upon basement membranes.

In solid blastemata the elaboration of cells into fibre occurs, for the most part, slowly and in the ordinary routine. The majority of the cells, however, remain undeveloped, and become reabsorbed. Still the development of primary cells into parent-cells, however rare, does occasionally happen.

Fluid blastemata, in their development to textures, obey the laws of the cell theory (Schwann's). The perfect nucleated cell, however, originates in two different ways :

(*a.*) The union of several elementary granules gives rise to the nucleus, and around this to the cell, with the nucleus impinging upon the wall,—the ordinary mode. Or else—

(*b.*) The cell originates first,—its primitive limpid contents giving rise to an *endogenous nucleo-genesis*—for example, in the blood,—in exudation—in colloid and medullary cancer.

Generally speaking, the nuclei equal in size those proper to physiological textures. Larger nuclei, however, and in particular oblong, free nuclei $\frac{1}{100}$th to $\frac{1}{50}$th of a millimetre in length, occur likewise,—in medullary cancer, for instance. Inclosed within cells, their further development, so far as we know, commences only after the conversion of the cell into fibre. They are round, oblong, lustrous, black-edged, or dull and granulated.

The cells present every variety of size, from that of the exudation- and the pus-cell to that of the largest ganglion-cell, and upwards. They are in shape spherical, oval, lengthened by branch-like processes, rhomboidal, polyedrical.

They mostly contain one, often two, occasionally several (three, four, or five), nuclei.

The propagation of nuclei and cells occurs either immediately out of the fluid intercellular substance, as blastema, or within a parent-cell. Endogenous nuclei and cells [brood-nuclei and cells; filial cells; intra-utricular cell-formation] from within a primary cell, and distend it into a structureless vesicle, by the eventual bursting of which they become released.

In rarer instances, we meet with secondary cell-formation around a primary cell,—an *incasing of the primary* cell.

The primary cell is either permanent or adapted for ulterior development, namely—

1. The ordinary development of the cell into fibre. This is brought about by the spontaneous elongation of a cell to a wedge- or spindle-shaped, or a caudated cell; or by the fusion of several cells, arrayed in rows or columns, and engaged in the act of elongation to a varicose fibre, the protuberances of which are eventually reduced. Fibre produced in either way may, by splitting lengthwise, subsequently break up into fibrils. In form, the fibre corresponds with that of areolar tissue, or of organic muscle. The cell-nuclei immediately form into nucleus-fibre, into elastic fibre. In this wise do fluid blastemata, under the progressive consumption of the intercellular substance, give rise to fibrous new growths.

2. The above transformation differs from the working out of the primary cell into the *parent-cell*, and to the production of *pouch-like formations with endogenous nucleus and cell-development*.

(*a.*) The parent-cell is a *cyst-like dilatation of the primary cell*, and its contents furnish the blastema for the creation of filial cells, in either of the two modes before described. When the latter have greatly increased in number, the parent-cell frequently, but not invariably, bursts, and is destroyed. Not rarely, however, it becomes the groundwork for very remarkable textures. (See " Cyst.")

(*a.*) The *structureless parietes of the growing parent-cell acquire a fibrous texture*, and thus become fundamental to the type of the *alveolar* texture, and to cyst formation. (See " Cyst.")

(*β.*) The *parent-cell is singly*, or it may be in fusion with others, developed into a *gibbous, lobulated, hollow body, resembling a glandular acinus.*

The *filial-cells enter occasionally, even within the parent-cell, into a fibrous development.* Upon the dura mater, tumors are often met with seemingly of glandular texture. These consist of conglomerations of caudated cells, imbedded in a layer composed of the same elements. They are the products of a single parent-cell.

(*b.*) In fluid blastemata, utricular or pouch-like formations occur, similar to the tubular fibres mentioned under the head of solid blastemata, and they inclose nuclei and cells in various number. Their walls appear structureless; although, on a closer inspection, one or two nuclei, —occasionally several movable nuclei,—may be detected upon them. They occur in colloid, in scirrhus, and in sarcoma, with a fluid intercellular substance. Their functional import is, in our opinion, identical with that of the parent-cell with its brood-elements. They present the greatest analogy with the capillary vessel and its contents, the more so that they probably originate through the fusion of nucleated or non-nucleated cells, arrayed in columnar juxtaposition. Their diameter ranges from the $\frac{1}{100}$th to the $\frac{1}{10}$th of a millimetre, and upwards.

We have now examined the essential elementary forms arising out of both solid and fluid blastemata. Their secondary arrangement into a texture offers equal diversity. Nuclei, cells, nay, elementary granules, display infinite variety in their arrangement, as do, in like manner, caudated nuclei and cells, and the different descriptions of fibres, in their course and in their co-ordination with other concurrent elements. These relations will have to be pointed out in the special analysis of new growths, to certain of which, peculiar arrangements naturally belong.

Other changes suffered by blastemata, either in their primitive state, or after having attained to different stages of development, are:

1. Resolution into a molecular point-mass. Blastema breaks up, in its primitive state, owing either to positive intrinsic relations, or to the absence of compulsory extrinsic conditions for its evolution. Or, again, it breaks up, after having already entered upon a course of development, owing to the cessation of the external conditions necessary to its maintenance and further elaboration.

In the state of disintegration, it may undergo complete or partial resorption, with or without entailing constitutional mischief. This process is often attended with cretaceous deposition, often with fatty conversion of the protein-substances.

2. The blastemata stop short at different stages, retrograde, and

perish. This may happen at any epoch of their development, from the primitive state upwards. The causes may be either inherent in, or extrinsical to, the blastema. In some instances it is a natural death, certain elements, epidermis-cells, for instance, dying off, after having attained their highest development. This of course applies more especially to solid blastemata. In their primitive condition, they part with their water, condense, and shrink into horn-like masses, and frequently ossify. When more advanced in their development,—for example, to fibre—such elements waste, and become reduced to primitive amorphous blastema, which immediately shrivels, often disengaging calcareous salts, that is, ossifying. Within the cell there occurs incrustation, with amorphous granules (a kind of granule-cells), or in stratiform deposition. Here the blastema has become bereft of all faculty for further development.

3. *Conversion into fat*, occurs both in primitive blastema and in tissues, and it is frequently accompanied by the disengagement of salts of lime,—by cretefaction and ossification. The protein substances undergo a transformation into free fat in little molecules, and into cholesterine crystals. To this conversion both solid and liquid blastemata are liable. Where cells exist, it occurs in the shape of granule-cells.

Vogel describes it as a peculiar granule-cell development, established for the resorption of an inflammatory exudation. "The exudation," says he, "is converted into nucleated cells of $\frac{1}{300}$th to $\frac{1}{100}$th of a millimetre in diameter. These cells progressively enlarge, until they have attained the size of from $\frac{1}{80}$th to $\frac{1}{50}$th of a millimetre, and gradually fill, at first with a few, afterwards with very numerous little dark granules, until the cell, originally transparent and colorless, becomes thoroughly opaque, assuming the brownish or blackish coloration of its contents, and appearing as an aggregation of granules, which cover and conceal the cell-nucleus, and frequently even the cell's walls."

The concomitant chemical changes consist in the formation, or at least eduction, of a new (reckoning the cell's walls and the cell-nucleus,—of a third) substance within the granules, possessing the characters of fat, and occasionally of salts of lime. Vogel says, further on, "the matured granule-cells are not susceptible of ulterior organic development. After they have attained their full size, and filled with granule-cells, their further metamorphosis is a retrograde one. The cell-nuclei disappear, becoming, like the cell's walls, reabsorbed, whilst the granules, which alone remain, and are at first held together by a viscid medium, finally separate. After the complete breaking up of the granule-cells, the entire exudate originally present is converted into a semi-fluid, pultaceous mass, which, with the aid of the microscope, is found to consist of, as yet, unchanged granules out of the broken granule-cells, natant in a fluid,—the original serum of the exuded blood-plasma."

With reference to this process, which affects not alone inflammatory products, but every kind of blastema, we have additionally to state:

1. The process of granule-cell development consists not, we apprehend, in a development of fresh nucleated cells, and of granules within these. The granules become developed rather within the already exist-

ing cells, and also externally to them in the intercellular substance. There are seen distinct granules, which here and there collect in smaller or greater number, and occasionally assume an investment, not distinguishable from the bond-mass by which they are held together. Those developed within the cells accumulate and distend the cell's walls, until these give away and allow the granules to escape. This process may be directly witnessed, but it is further corroborated by the following circumstances:

(*a.*) Where the blastema is devoid of pre-existent cells, it does not contain any nucleated granule-cells either, but simply aggregates of granules; for example, in the fatty conversion of certain fibrinous coagula, of primitive muscle-fibrils, and of fibrous new growths.

(*b.*) The exudation-cell, the pus-cell, the cancer-cell, as the case may be, becomes the granule-cell, which retains the form of the pre-existent cell,—for example, the spherical, wedge-like, spindle-shaped, fibro-elongated granule-cell.

2. This process is, in point of fact, the fatty conversion of the contents of the cell. It is the counterpart of the fatty conversion of protein substances in every variety of blastema, and even in tissues generally. It gives rise to emulsive and saponaceous combinations, thus proving destructive to both blastemata and new growths, which latter it would indeed render fitted for resorption, were this not often hindered by the simultaneous disengagement of phosphate of lime with cholesterine crystals.

These changes run parallel with chemical ones, consisting in the development of different kinds of gluten, in horny conversion, and the like.

3. Finally, blastemata (like physiological textures) become reabsorbed at various stages of development, having become adapted for the process by a previous disintegration or fatty conversion, although, in the case of fluid blastemata, without any intermediate change. Solid blastemata may become gradually dissolved and fitted for resorption by blood serum percolating the textures, for example, in solid, fibrinous, inflammatory products. Occasionally some of their nuclei are left behind, presenting the only visible residue of comparatively extensive blastema masses.

Our next inquiry concerns the conditions which favor the throwing out of pathological blastemata in particular localities. These may consist in an exudatory process, not differing from that which presides over the normal act of nutrition, or else in processes which, though akin to physiological, are, in strict parlance, pathological. Such are hyperæmia, and inflammation in its numerous modifications. Again, blastemata become consolidated within the vascular system through the coagulation of fibrin, as metastases, or deposits.

HYPERÆMIA.

It is to be understood that we have to deal only with local hyperæmia —congestion, so called.

It consists in an excessive amount of blood in the capillaries of an organ; that is, in an injected condition of this latter, exceeding what experience has shown to be its average. This is not possible without dilatation of the vessels, nor can we admit the existence of congestion

with coarctation of the vessels, and a *consequent* accelerated passage of the blood through them, which some have designated as active congestion.

A simple comprehensive view of the development of hyperæmia is not feasible, the conditions not being always the same. It will, perhaps, suffice to distinguish between *active, passive*, and *mechanical* hyperæmia. We shall here, however, not consider these individually—more especially, the two former—further than may be requisite for the establishment of a principle. Their closer investigation will be more appropriate under the head of Inflammation.

1. *Active hyperæmia* is the result of external or internal stimuli acting immediately upon an organ, or reflected to it from other organs, —irritating the sensitive nerves, and thereby causing antagonistic palsy of the nervi vasorum or (according to another theory), evoking an increased afflux of blood, a preternatural affinity of the parenchyma for the blood.

To this category belong, for the most part, those hyperæmiæ, dependent upon external or internal causes, which precede inflammations; most of those habitual, constitutional hyperæmia, rooted directly in the nervous system or in the blood; again, those hyperæmiæ occasioned by augmented activity, by overwrought function, or concurrent with excited conditions of the organs.

2. *Passive Congestion* depends upon direct palsy of the nervi vasorum, wherewith is commonly associated a depressed energy in the remainder of the nervous system. The palsy may originate in the centres, or it may be peripheral. It is often determined by dyscrasis, and especially by the higher degrees of decomposition of the blood.

To this class must be referred those hyperæmiæ, introductory to so-called asthenic inflammations, in organs exhausted by excess of functional activity, enfeebled by hyperæmiæ and inflammation, or paralyzed; as also in those hypostatic hyperæmiæ of the lungs, of the abdominal and pelvic organs, of the common integuments, which are developed under diminished impulse from the heart, in dependent parts of the body, during the progress of various adynamiæ and marasmi.

3. Mechanical hyperæmiæ, namely,—

(*a.*) Hyperæmiæ arising from palpable mechanical impediments to the return of the blood through the veins, or to the ultimate disgorging of the venous trunks into the heart. The extension of the hyperæmiæ varies with the locality of the impediment. It affects single organs and sections of organs; for example, a portion of intestine strangulated, invaginated through tension and compression of its bloodvessels. Or it may have a more comprehensive range in impermeability of the liver, of the lungs, in stenosis (coarctation) of the heart's valves. The hyperæmiæ occasioned by spontaneous coagulation within the capillaries, by various elements obstructive of these vessels, as pus-corpuscle, cancer-cell, injected mercury globule, are commonly referred to the same head.

(*b.*) Hyperæmiæ ex vacuo, as they occur in atrophy of the brain within the unyielding skull, or in the gravid uterus after rapid delivery, often to the extent of producing hemorrhage; hyperæmiæ due to the eccentrical rarefying atrophy of organs.

The hyperæmiæ is either of a more or less *transitory* or of an *abiding* nature, of which latter kind mechanical hyperæmiæ, from heart disease or from induration of the liver, present the most frequent and the most marked examples.

The sequelæ of hyperæmiæ are multiplex, varying with the duration, the repetition, the degree, of the congestion. Much likewise depends upon the character of the affected organ, the congestion being significant in proportion to the general importance of such organs, and to the vulnerability of its texture. Organs are prone to congestion proportionately to their vascularity and to the degree of their functional activity. Under particular circumstances of life, of occupation, of civilization, certain organs, such as the brain and its membranes, and the lungs, are hardly ever entirely free from congestion. Hyperæmiæ affects morbid growths equally with normal formations.

Intense congestion suddenly developed in organs essential to life (the brain or lungs) may prove fatal directly, as so termed *vascular* apoplexy, or through the sudden effusion of blood serum into the textures—acute œdema.

High degrees of congestion occasion laceration of capillaries and parenchymatous hemorrhage (apoplexy with bloody extravasation) in the brain, the lungs, and other organs.

The same causes lead, by an overloading of the bloodvessels, to absolute palsy of the bloodvessels, to stasis, inflammation, and gangrene.

Moderate but habitual or repeated congestion gradually engenders œdema and the dropsy of serous cavities—*genuine dropsy*, increased exudation of blood plasma, preternatural nutrition of the textures—*hypertrophy, augmented secretion.*

In this relation, abiding mechanical hyperæmiæ, from heart disease, are worthy of especially notice, with their unfailing consequences, hypertrophy of the glandular abdominal viscera ; preternatural secretion of the intestinal and bronchial mucous membrane ; excessive, saturated secretion of bile.

Hyperæmiæ create and bequeath permanent dilatation and elongation with coil-like or serpentine deflection—properly termed varicosity—of the bloodvessels, as more particularly exemplified in the less resilient veins.

Hyperæmiæ frequently occasion and obviously accompany the development of various heterologous growths. Finally, in some organs, a proportion of blood-pigment, effused with the plasma, constitutes the basis of rust-colored, slate-gray, bluish-black coloration, as in the lungs or on the intestinal mucous membrane.

Organs attacked by a high degree of hyperæmia present different shades of dark red, become swollen, loosened in texture, and consequently friable, lacerable. In organs of a porous spongy texture, the swelling seems due to a bloated condition of the tissue itself.

HEMORRHAGE.

Hemorrhage consists in the extravasation of blood bodily, and in its entirety, from the bloodvessels, consequent upon a breach of their con-

tinuity. Herein it differs from red effusions resulting from the transudation through the parietes of vessels of blood serum, which, owing to various changes of admixture, has taken up blood pigment. Hemorrhage occurs either within textures, when it is with reference to its attendant paralysis, somewhat inappropriately termed apoplexy; or else within natural or preternatural cavities or canals; for example, in serous sacs, in muco-membranous cavities or canals, pus-reservoirs, &c. The two kinds frequently coexist.

Heterologous growths are, in the same degree as normal formations, subject to hemorrhage, those at least which are highly vascular; as, for instance, adventitious membranes, carcinoma (in a high degree), the interior of cysts, &c.

Hemorrhage depends upon various causes, the most common cause being, as before stated, a breach of continuity in the bloodvessels.

Apart from hemorrhages produced by external injury inflicted upon bloodvessels, whether alone or in conjunction with other formations, those resulting from the following momenta, more especially capillary (parenchymatous) hemorrhages, possess a high degree of interest.

1. Hemorrhage the result of intense hyperæmia, of whatever kind. Thus, active hyperæmia has a marked tendency to create bronchial hemorrhage, passive hyperæmia uterine hemorrhages, hyperæmia from mechanical causes, in particular the bronchial and intestinal hemorrhages that result from heart disease, and the cerebral hemorrhages induced by a vacuum within the skull.

Finally, the excessive accumulation of blood determines rupture of the capillaries.

2. Another step conducts us to hemorrhage as occurring during the progress of inflammation, namely, in the stages of congestion and of stasis. In this combination we have hemorrhagic inflammation, and the in many respects remarkable exudation designated *hemorrhagic*. Here, as in *simple* hyperæmia, the hemorrhage is unfailing, and considerable in proportion to the extent of the congestion and stasis, as also to the delicate, lax, and vulnerable, nature of the implicated texture. Any one of these influences may predominate to a various extent. There are organs in which, owing to the nature of the texture, inflammation never takes place without hemorrhage—for instance, the brain, the lungs, many heterologous formations, and especially exudates undergoing a change of structure, and loose cancerous textures. The hemorrhage is capillary.

3. Hemorrhage from the laceration of vessels, produced in atrophied organs by the laxity and diminished resistance of surrounding textures—apoplexy of the decrepit uterus.

4. Hemorrhage from the spontaneous laceration of organs diseased in texture, pulpy and friable—laceration of the heart.

5. Hemorrhage from laceration of vessels consequent upon impaired texture of their coats, with or without dilatation of their calibre. It affects the smallest bloodvessels as readily as the main trunks, and more especially the arteries. Other coincident causes—hyperæmia, for example—greatly favor its occurrence.

6. Hemorrhage consequent upon the destruction of bloodvessels by ulceration, or by contact with a free acid, as in softening of the stomach.

It is questionable whether, and in what way, dyscrasial states can give rise to hemorrhage. A relaxation of the coats of bloodvessels sufficient to admit of the passage through them not only of plasma, but also of blood-corpuscles, is by no means proved, and its assumption, to explain the hemorrhage occurring in scurvy or typhus, needless. The spontaneous hemorrhage arising in the progress of such maladies, is the result either of local hyperæmia and inflammation, into which those general diseases have resolved themselves, or else of preternatural expansion (increased volume) of the blood itself, and of the consequent rupture of bloodvessels in the looser textures, such as the gums, the mucous membranes, and the lungs. Hence the occurrence of hemorrhage in several organs simultaneously, and again the predominant invasion of a few particular organs. That hemorrhage, having once set in under such circumstances, is apt to become excessive, is due, without any doubt, to the slender coagulability of the dyscrasial blood.

*Hæmorrhophilis—habitual hemorrhage—*depends, so far as we at present know, upon a preternaturally delicate and vulnerable structure of the coats of the vessels, coupled with a thin, watery, condition of the blood.

Hemorrhage greatly varies in intensity. Its character is to be estimated not merely by the quantity of blood thrown out either externally or into internal cavities and canals, nor by the degree of anæmia that follows, but also, where the parenchyma of organs is its seat, by the anatomical condition in which we find the diseased parenchyma. In slight hemorrhage, the texture appears here and there dotted or streaked with extravasated blood,—*Capillary apoplexy*. As the hemorrhage increases, these dots or streaks become more crowded, the parenchyma, more turgid, until its interstices and cavities having at length become uniformly surcharged with blood, it appears throughout red. Or, where the blood has become coagulated, the rough texture seems as if converted into a blood placenta, whilst a diminished coherence of its molecules, and numberless lesions of continuity have rendered it friable, easily torn [mucous membrane, lung]. Such is the condition of the denser, more resisting textures; in those of a more lax nature, or where the hemorrhage has been sudden and violent, the texture is completely swamped and crushed into a red pulp of various shades, or else more or less laceration of texture has taken place, and the gap so occasioned become the recipient for the extravasated blood—*apoplectic foyer*.

The extravasated blood varies in deportment according as it is circumstanced subsequently to its extravasation. Certain influences cause it to undergo rapid and unusual changes: for example, in the stomach and intestines, black coloration and liquefaction of its fibrin, through the influence of gastric and enteric acid.

Blood poured out into cavities and canals, or into textures, is either fluid or in various phases of coagulation. Of these phases, coagulation with central or peripheral *encysting* separation of fibrin is the most important, owing to the increased impediment which it offers to the process of absorption.

The immediate effects of hemorrhage, besides the anæmia consequent upon great effusion, either out of the body, or into its cavities, are lesion

of continuity in textures, in the shape of the swamping, crushing, or extensive laceration before referred to—impaired or destroyed function, paralysis of the organ affected—*cerebral, muscular hemorrhage.* A less immediate effect is the inflammation of surrounding textures, occasioned by actual injury, and by the irritation of the extravasated fluid, as a foreign body, with eventual organization of the products effused, callous condensation of the nether layer, and capsular isolation of the hemorrhagic clot. Inflammation, resulting in purulent and ichorous products, in parts broken up by hemorrhage, is of rarer occurrence.

Hemorrhage is both in itself, and in its results, of significance commensurate with the importance of the organ affected.

The cure of hemorrhage is a process simple or complex in a degree corresponding to the amount of blood effused, to its character as coagulable fluid, and to the extent of the injury suffered by the parenchyma.

Slight hemorrhage is readily cured through resorption of the effused fluid, enabling the distended textures to recover their resiliency. The liberated red pigment, however, frequently resists absorption, even in slight hemorrhage, remaining strewn, for the most part in a state of minute molecular dispersion, over membranous formations, or between the elementary parts of a texture, as a brown or black pigment.

The remedial process is difficult and complicated proportionately to the amount of blood extravasated, to the resulting destruction of texture, and to the solidity of the coagulated fibrin as a central or a peripheral secretion from the fluid thrown out. It is a process of slow gradation, involving the changes produced not only in the effused blood with its red pigment and its fibrin, but also in the surrounding textures. These changes occur simultaneously, and we have frequent opportunities of observing them in areolar tissue, in muscle, and especially in the brain.

The crushed and disorganized texture within the walls of the foyer, together with the extravasate itself, undergoes liquefaction, at the same time that hyperæmia and stasis become developed in the adjacent texture-layer. The medium is probably almost exclusively supplied by the blood serum of the extravasated fluid which undergoes many changes in composition adapting it for the liquefaction of the different substances; namely, the solidified fibrin and the remaining elements proper to the effusion—blood-globules, nucleated and cell-formations, *debris* of tissues. How important a part the liberation of adipose, and of saline substances out of their primitive combinations herein plays, is sufficiently attested by microscopic analysis. The blood-pigment incurs a special change. It is converted, partly within the blood-corpuscles, partly extraneously to them, into a brown, rusty yellow, or into a blackish-brown, or a black pigment. This is shown as well in the form of spherical corpuscles, which resemble blood-globules and are frequently seen accumulated in compact congeries, as also in the shape of elementary granules (granulated pigment), either discrete or in circular groups. These are commonly free, but now and then inclosed within cells, or suspended from little prismatic crystals of ammonio-phosphate of magnesia. Together with them is found, at the part involved, fat in a free state, fat in the form of little black-edged, discrete or aggregate molecules, of limpid drops, of cholesterine crystals. Again, there are found elementary

molecules down to the minutest pulverulent molecular mass, consisting of minutely subdivided, suspended fibrin, albumen, and fat, with calcareous salts. Finally, we have amorphous, membranaceous, stratiform coagula, nuclei, and blood-disks (the as yet integral elements of the effusion), and amongst them all detritus of the involved texture.

In this manner the greater portion of the hemorrhagic effusion would have become fitted for resorption. The process is, however, impeded at this juncture, by the inflamed condition, and at a later period by the hardened character of surrounding textures.

Hence the ulterior metamorphosis of the effused mass, namely, its progressive thinning and clarifying into a mere pale buff, or, it may be, colorless liquid.

What, amongst other things, has become of the pigment? Partially it may have perished amid the unknown, final conversions of the fluid above characterized. To some extent, however, it is preliminarily taken up into a formation which,—derived from the coagulable contents of the effused fluid—invests the walls of the foyer.

This colored, soft, jelly-like, loosely adherent lining, eventually becomes endowed with a minutely fibrillated structure, and even with blood-vessels, and is at length converted into a delicate, and, if the pigment be destroyed, into a colorless membrane, resembling a serous tunic.

Meanwhile, the inflammatory process has engendered in the walls of the foyer products which serve, in the shape of a nucleated blastema (to be afterwards developed into fibrin texture of various kinds, and, into areolar tissue), to condense and harden the textures. Thus, the original hemorrhagic foyer is changed into a capsule or cyst, which, when it occurs in the cerebrum, is termed *apoplectic cyst*.

This cyst is susceptible of diminution, and eventual closure, through resorption of its contents,—of closure to a cicatrix which often contains a certain kernel consisting of the aforesaid residuary pigment.

It must be confessed, however, that the complete closure of the cyst is a work of time and difficulty. This is intelligible from the slender absorbent faculty of the surrounding textures in their condensed and hardened condition. Occasionally, special obstacles stand in the way of this process of reduction; for example—

1. Great extent of the hemorrhagic foyer, and of the resulting cyst.

2. A vacuum, created either through original retraction, or through subsequent wasting of the texture involved, in muscles and especially in the cerebrum. In the brain, indeed, a later supplementary enlargement may take place in the apoplectic cyst, as an expletive of the vacuum created within the skull by consecutive atrophy of the cerebral organ; for the internal capsular membrane is, by reason of its vascularity, adapted alike for secretion and for absorption.

3. A very remarkable obstacle to the collapse and closure of the cyst consists in the secretion of fibrin in the shape either of central bulky, or else of peripheral isolating coagula, for the most part tinged by no inconsiderable proportion of embodied blood-corpuscles. These coagula, being originally very dense, and retaining their solidity even when converted into a fibrous texture, resist, when central, the liquefaction, when peripheral and encysting, the resorption of their contents.

Certain cases offer various exceptions to the processes hitherto described. Thus, some cysts having, in spite of the peripheral encysting coagula, parted with their blood-serum by early resorption, are found replete with a dark-colored, inspissated, dry blood-plug; or else with fibrinous, stratiform, villous masses, developed out of the partially absorbed vehicle.

The frequency of hemorrhage varies greatly in the different textures of the organs. A scale of frequency is indeed but of very limited use, since hemorrhage is the result of various disturbances, and, in most instances, of the concurrence of several. Generally speaking, hemorrhages of the brain and of the bronchial mucous membrane are distinguished by their frequency; those of serous membranes are very rare, if we except the cerebral arachnoid sac.

It was signified at the outset, that the mere exudation of colored (red) serum, devoid of blood-corpusles, is perfectly distinct from hemorrhage. It is found as so called petechiæ (ecchymoses) in all textures, and in serous and mucous cavities as colored effusion. It is due to decomposition of the blood.

ANÆMIA.

The chapter on hyperæmia naturally leads to a passing consideration of the opposite state, namely *anæmia*. Just as we have before treated only of *local* hyperæmia, we shall here, in like manner, limit ourselves to the subject of *local* anæmia. It comprehends both oligæmia, or an insufficient measure of blood in relation to what experience has shown to be its just standard, and true anæmia of an organ.

It is present under various conditions:

1. As the partial manifestation of general anæmia.
2. As the consequence of hyperæmia of one or more other organs.
3. As the result of coarctation and closure, or orificial obstruction of the vascular trunk supplying the diseased organ or part, pending the establishment of a compensating collateral circulation.
4. As the effect of external or internal pressure upon an organ, and its consequent inadequate injection; anæmia of the lungs from pleuritic effusion; anæmia of textures the interstices of which are filled up with morbid products, as in hepatization of the lungs, in fatty infiltration of the liver.
5. As a consequence of decay of the vascular apparatus of an organ affected with atrophy, whether primary or secondary, more especially atrophy with condensation (concentrical wasting).

The effects of anæmia are pallor, collapse, and shrivelling of the textures, weakening and eventual extinction of their function.

Anæmia is momentous proportionately to the vital importance of the diseased organ, and to its exigencies with respect to the supply of blood. Thus, anæmia of the brain, of the lungs, of muscle, is of the highest import.

INFLAMMATION, PHLOGOSIS.

This pathological process is of paramount interest, not only on account of its great frequency, and of the great variety of external causes by

which it is called forth, but also as being that in which most, and in a certain sense all, general diseases become localized. It is a process which leads incontinently to the most various and most extensive new growths, and associates itself, equally often, to other anomalous formative efforts. In fine, it is a process which, on the one hand productive, on the other hand frequently proves destructive of both normal and anomalous formations.

The inflammatory process is capable of being experimentally called forth and observed, in all its phases, in transparent textures. Thus studied, it has furnished the groundwork for the most varied interpretations, but, at the same time, for researches respecting other exudatory processes. Nor have these experiments failed in a certain measure to elucidate the connection that prevails between blastema and the endogenous formative processes carried on in the blood itself.

We seek not to deprive this process of its time-honored name, *inflammation*, because it has become naturalized in science beyond all others. It is applicable enough, if in using it we simply dismiss the theories which first led to its adoption. It comprehends the entire process, and the efforts made to designate the latter differently have utterly failed. Andral's hyperæmia, and Eisenmann's stasis have not advanced the subject by a single step.

It is impossible to define inflammation suitably, owing, on the one side, to our imperfect knowledge of its proximate causes; and, on the other, to the complex nature of its consecutive phenomena. These latter, variously modified in type, point to processes equally varied, whilst they at the same time furnish the most striking analogies with other processes which issue in exudation (production of blastema).

Let us now proceed to a descriptive examination of the phenomena which constitute so many stages of the process, a due regard being had to the results of experiment, that is, to the observation of the inflammatory process as artificially called forth in animals. And in this description, together with the analytical remarks annexed to it, we will take for our basis the so-called pure, legitimate, inflammation, which yields essentially a coagulable, fibrinous, plastic product, as developed in sound organisms without the co-operation of a pre-existent dyscrasis, and simply as a consequence of moderate local stimulation; such being the most marked of any in its manifestations and stages.

The phenomena of the inflammatory process present the following sequence.

1. The moderate influence of mechanical or chemical stimuli is followed by contraction of *the capillaries, and simultaneous quickening of the blood-stream through them*. This phenomenon may be wanting as an effect of most causes of inflammation in the human species; and even in experiments upon animals it is either transitory or entirely absent if the stimuli applied be potent.

Contraction of the vessels is succeeded sooner or later by—

2. *Dilatation of the capillaries*, if this be not, indeed, the very first cognizable phenomenon. Unlike contraction, it is invariably present, readily seen both in the living animal and in the dead subject, as is, in like manner, the simultaneous loading of the vessels with an increase of

blood. It determines capillary injection, and therefore the redness of injection proper to inflamed textures.

This dilatation of the vessels is attended by a *retarding of the blood-stream*, which sooner or later, although not always visibly, merges in an oscillating movement of the blood in the capillaries. The contained blood-columns move forward and backward by turns, the onward movement, however, predominating. The blood-corpuscles begin to adhere to one another, like rolls of coins, the outer linear layer of plasma (the lymph space) within the vessel still remaining unchanged.

This twofold proceeding establishes the stage of congestion.

3. Hereupon ensues, sometimes so rapidly as to prevent the retardment of the blood-stream and its oscillation from being noticed, *stagnation of the blood-stream,—stasis.* The bloodvessels are completely filled up with blood-corpuscles, so that the transparent, so termed, lymph space, near the circumference of the vessel and before occupied by plasma, has vanished. Meanwhile the blood-corpuscles have assumed greater intensity of color, have become flattened, contracted, and firmly glued to each other, and to the vessel's walls, so as to form a homogeneous red mass, with irregular translucent intervals.

The nuclei and nucleated cells (so-called lymph-globules and colorless blood-corpuscles) have increased in number to an extraordinary extent, often adhering together in groups connected by delicate transparent coagula, and forming either thus, or singly, the aforesaid translucent intervals. The blood has assumed a dark tile-colored aspect, verging upon cherry-red.

In the preceding stage (see hyperæmia), as well as in this, two notable phenomena are witnessed, namely:

(*a.*) Laceration of bloodvessels, and extravasation into the textures, or into the free spaces,—lung-cells; muco-membranous cavities and canals; serous sacs. The hemorrhage is frequent and considerable, proportionately to the degree of congestion and stasis, and also to the delicacy and the textural looseness of the diseased part. For the most part it takes the form of capillary apoplexy, and only in very delicate normal and anomalous textures, as, for example, the brain or encephaloid cancer, the form of the isolated clot.

(*b.*) Transudation of blood-serum through the thinned bloodvessel walls into the parenchyma, and from membranous expansions into the cavities and canals of which they form the lining. This implies, in the parenchymata, pervading moisture,—in expansive structures, diffuse exudation or circumscribed accumulations beneath the epidermis,—for example, in burns, in vesication artificially called forth, in erysipelas, and the like. The exuded serum resembles essentially blood-serum, only that it is, for the most part, less rich in albumen. This phenomenon often merges at once in the exudatory process next succeeding, the two acts being simultaneous.

4. The step to which stasis ultimately leads is *genuine effusion*, that is, the exudation of blood-plasma, a fluid holding in solution fibrin, albumen, and salts. It is thrown out into parenchymata, filling up their interstices to a various extent, either as a fluid, or as a more or less solidified product. Or, again, it is partially, or, it may be, wholly

expended upon the free surfaces of natural cavities and canals, or of such as have arisen out of the previous transudation of serum, for example, in vesication of the epidermis, or in antecedent suppuration of textures,—in abscess cavities.

With exudation, the inflammatory process is to be looked upon as closed. It is immediately followed by an endosmotic current of the serous portion of the effusion, causing the blood-corpuscles to float in a thinner medium, to exchange their now flattened for a more spherical shape, to become separated, to part with a portion of their pigment, and finally, by dint of a returning resilience in the bloodvessels, to move, conjointly with the aforesaid form-elements, onward again in the circulating stream.

We have now furnished the reader with a substantial description of the several acts which make up the process of inflammation. We shall further endeavor to show how these may be reconciled with the present standard of pathological science.

1. The first experimental phenomenon adverted to, namely, contraction of the capillaries, with acceleration of the blood-stream, has been stated to be inconstant, and indeed absent altogether, when potent stimuli have been used in the first instance. Where it does occur, it is to be regarded as a vital phenomenon. The contraction of the bloodvessels is moreover independent of any simultaneous collapse of the parenchyma, like that produced, for example, by the action of cold.

2. The dilatation of bloodvessels and the retardment of the blood-stream are, on the other hand, constant and essential. The very fact of their being so commonly the primitive phenomena, or at any rate of their succeeding very rapidly to a previous contraction, refutes the notion of their consisting in a secondary relaxation, resulting from exhaustion. With these, and with the subsequent stasis, the theories of inflammation hitherto advanced are mainly concerned.

Henle reduces these to an attraction-theory, and to a neuropathological theory.

(a.) The attraction-theory refers the essential phenomena of inflammation to an augmented affinity between the parenchyma and the blood, and especially to an anormal attraction of the blood, and of the blood-corpuscles in particular, by the affected parenchyma. It assumes the retardation of the blood-stream, and the crowding together of the blood-disks to be the primary—the dilatation of the capillaries a secondary phenomenon. It explains even the stasis as a continued increment of that attraction.

The augmented attraction is effected through the intervention of the nerves, and either by direct influence upon the peripheral nerves, or else by reflection from the centres of the nervous system.

The attraction-theory carries the problem of congestion and stasis a step farther, without solving it. Together with its common attribute, namely, an increased afflux of blood to the diseased part, as due to a dilatation, and to a more frequent contraction of the afferent artery, it has been met by many valid objections. It is only the stasis and inflammation engendered by a pre-existent dyscrasis that can, provided the attraction be not limited to the blood-corpuscles, but embrace the

diseased plasma, warrant the conclusion of a preternaturally strong affinity between blood and parenchyma. There would be some analogy between such a localization of general disease, and the indwelling relation of secreting parenchymata to certain normal or anomalous ingredients of the blood. But these inflammations, like the rest, are aptly expounded in the neuropathological theory.

The neuropathological theory, on the contrary, assigns to the nerves an important part, and ascribes the accumulation of the blood to the dilatation of the bloodvessels, this being set down as the primary—that as the consecutive phenomenon.

The dilatation of the vessels is the consequence of paralysis of the nerves. Respecting the cause and the conditions of this paralysis, there exist two different opinions.

According to Henle, an antagonistic relation prevails between the states of irritation of the sensitive nerves, and of the nerves of the bloodvessels: a high degree of inflammatory irritation in a sensitive nerve producing a state of depression—in a word, paralysis of the implicated bloodvessel nerves.

According to Stilling, the sensitive and the bloodvessel nerves bear, on the contrary, a direct sympathetic relation to each other. He assumes a continual reflex action to be kept up by the sensitive, upon the bloodvessel nerves, whereby the tone of the latter is sustained. With paralysis of the former, the tone of the latter is destroyed; whereas by excitation of the former their reflective power is augmented, and the tone of the latter thereby raised. In accordance with this reciprocity, two different kinds of inflammation are made out.

Griesinger was led to the adoption of a similar view, imagining pain to result from a qualitative disturbance of the texture of the nerve.

Against this hypothesis it is to be objected that a continued reflex action of the nerves of sensation upon those of the bloodvessels as a necessary condition for the undisturbed function of those vessels is not proved; and that the assumption of two different characters of inflammation, involves both a contraditio in adjecto, and a disregard of the results of observation. An inflammation with augmented tone of the bloodvessels cannot exist, and this admitted, inflammation with and through paralysis (diminished tone), must invariably ensue from influences paralyzing to the sensitive nerves. This is, however, opposed to daily experience.

Our own opinions accord with those of Henle, whose theory we shall therefore adopt as the groundwork of any future remarks on this subject.

Even here the causal momentum influences the peripheral nerves, producing either at the spot itself, or through the intervention of the nervous centres, in other sympathetically allied structures, excitation and depression. Or again, the influence affects the nerves within the centres, the impression being conveyed from *thence* to the corresponding peripheral organs.

The stasis is not accounted for in the neuropathological theory. It is, indeed,—

3. Not intelligible upon the ground of paralysis and dilatation of the bloodvessels, even though [as we must admit a certain off-flowing to

take place to the veins] we may not regard the stasis as an absolute one. For our own part, we hold stasis to be dependent upon the following momenta :

(*a.*) The cohering, crowding, and impaction of the blood-disks within the capillaries, the blood-plasma being partly withdrawn into the veins.

(*b.*) The thickening of the plasma, and its saturation with fibrin and albumen, owing to the transuding of blood-serum through the distended and thinned bloodvessel walls.

(*c.*) The accumulation of the colorless globules—that is, nucleus and cell-formations—along with the blood-corpuscles, their conglutination, and the delicate transparent fibrinous coagula collaterally developed. This is, perhaps, the most important stage in the inflammatory process, as at once illustrating the stasis itself, and embracing the plastic processes in which the blood engages when arrived at this point. A line of distinction is thus drawn between the inflammatory process, and a simple process of exudation. The form-elements adverted to are not merely washed together within the range of the stasis ; they are new creations out of the blood so arrested, which at the same time undergoes other remarkable changes. Thus it is of a dark red, with a tile-colored shade, contains red flocculent particles of cruor, visible to the naked eye, teems with the aforesaid elementary bodies, and with coagula, most of which latter have incorporated a number of the former, as well as of dark-colored flattened blood-corpuscles. The accumulation as well as the general importance of those (new) elements, for the inflammatory process, and especially the stasis, have been recognized by Addison and others.

The momentous question, as to the cause of the said formative process of blood in the condition of stasis, will be answered, so far as it is possible, in the sequel.

4. *Exudation.*—The thinning and permeability of the walls of bloodvessels, produced by their distension, must be regarded as the basis of this phenomenon, even in the instance of a condensed plasma ; perhaps, additionally, the effort at equalization betwixt the latter and any thinner blood-serum before exuded.

A question of peculiar interest here suggests itself, namely : wherefore in the inflammation of membranous expansions does exudation always take place upon their surface, and into the cavities which they invest, whilst the effusion beneath is limited to the infiltration of the implicated parenchyma, or of the subjacent areolar tissue—with, for the most part, an inconsiderable amount of plastic serum? This applies not only to mucous and serous membranes, but even to other more delicate hollow bodies—for example, follicles. The problem, like that of Johannes Müller, as to natural secretions affecting the free surfaces, is only to be solved upon the ground of less resistance being offered in this direction. The said infiltration of parenchymata and of contiguous textures, is co-significant with the œdema that surrounds patches of inflammation.

To an indefinite distance beyond the range of true inflammation, and lessening in intensity as the distance increases, congestion takes place, and with it the effusion of serum. And the serum becomes, in like man-

ner, poorer in plastic substances towards its periphery. Such is *inflammatory œdema; œdema encompassing the range of inflammation.*

The accompaniments of *pain, redness, heat, swelling*, are explicable as follows:

Pain is determined—

1. In external injuries, as wounds, burns, cauterization, either by the immediate action of the cause of the inflammation upon the peripheral nerves, or else by reflection from the central organs. In no instance is it determined by the inflammation *itself.*

2. By the pressure and tension which the dilated and overladen vessels, and the effused fluid exercise upon the nerves, *true inflammatory pain.*

3. Finally, pain of a certain degree, or rather of particular kinds, is to be referred to augmented temperature in an inflamed part. In the absence of increased warmth, which characterizes certain inflammations, pain is generally absent, also.

The redness is a consequence of the overloading of the dilated capillaries with blood-corpuscles; it is therefore to be designated as the *redness of injection.* A new creation of bloodvessels does not, as was once supposed, ever accompany the inflammatory process itself, and cannot, therefore, be taken into account here.

The *redness* is also, in some measure, due to the blood thrown out during the stage of congestion and stasis.

In some inflammations, one great source of the redness is the drenching of the tissues with dissolved blood-pigment,—*redness of imbibition.*

In form, the redness of injection varies in different textures, according to the order in which their capillaries are disposed. Take for example the linear redness of injection in inflamed fibrous structures. In the most vascular structures, however, the naked eye is no longer cognizant of aught but a uniform red tint.

Lastly, the redness is subject to many gradations of color, being deeper in proportion as the organ is vascular, and the congestion intense. Much depends, moreover, upon the constitution of the blood, and more especially of its red pigment; take, for instance, the copper-redness of the syphilitic, the violet hue of the typhous stasis. The *elevated temperature* has its source partly in the formative processes, in which blood in the condition of stasis becomes engaged, but for the most part in the excitation produced upon the sensitive nerves.

The swelling is dependent—

(*a.*) Upon dilatation and repletion of the capillaries;

(*b.*) More especially upon exudation of blood-serum and plasma;

(*c.*) Upon concurrent hemorrhagic effusion (extravasation).

The two latter conditions give rise, in like manner, to the loose, lacerable condition of inflamed textures.

After this description of the inflammatory process, and this interpretation of its phenomena, we should proceed to investigate the so-called issues of inflammation. These, however, resolution excepted, cannot be satisfactorily considered without an insight into the various products of inflammation, nor these products themselves be rendered intelligible but by a knowledge of the manifold varieties and anomalies of the process, and of its relation to the blood-crasis.

VARIETIES OF INFLAMMATION.

Inflammation is characterized by much variety and anomaly. On the one side, it recedes so far from the foregoing description of the process, that it has been attempted to distinguish certain forms as *spurious inflammation*. On the other side, its gradations into mere hyperæmia and a preternatural amount of plasma-exudation (nutritive irritation) are so imperceptible, that discrimination becomes a matter of difficulty, the greater, perhaps, that such processes frequently do become exalted into, and do alternate with, inflammation.

Every hyperæmia may attain the point of inflammatory stasis.

(*a.*) *Active hyperæmia*, the same in origin with inflammatory congestion, is developed into *sthenic*, or active, *inflammation*.

(*b.*) *Passive hyperæmia*,—the result of direct, centrical or peripheral palsy of the entire nerve-apparatus of an organ,—becomes *passive, asthenic inflammation*. To it belong, amongst others, all hypostatic inflammations, occurring in dependent parts during the progress of adynamiæ and marasmi, and the asthenic inflammations in organs paralyzed by concussion, complex injuries, or by direct central influence,—for example, of the bladder, in paraplegia. Many of them have a humoral origin, a dyscrasial disturbance giving rise to paralysis either central or directly peripheral. The hyperæmia and stasis are characterized, in the absence of pain and increased temperature, by very dark livid redness, partly of injection, but for the most part of imbibition. Their products, conformably with the humoral elements, are poor in coagulable material, discolored by adherent blood-pigment, spuriously reddened, sero-albuminous, sero-purulent. Frequently the stasis becomes absolute, degenerating into necrosis of the blood and of the diseased texture ; in one word, into gangrene.

(*c.*) *Mechanical hyperæmia*, as we have seen, commonly determines exudation of serum [œdema], and this not alone from the true capillaries, but also from the larger veins. By intense mechanical obstruction, the hyperæmia is raised to a stasis marked by very deep redness, great tumefaction, numerous lacerations of vessels, and hemorrhage. It deposits the usual coagulable products, but often degenerates, with complete paralysis of the organ affected, into absolute stasis and gangrene.

The course of inflammation is *acute* or *chronic ;* all else being equal, sthenic, and traumatic inflammations, and (amongst those due to internal causes) such as result from a fibrino-croupous crasis, are marked by their acute character. Asthenic inflammations, on the other side, and of these, more especially the ordinary hypostatic, incline to a chronic course. *Chronic inflammation* is variously modified.

(*a.*) Inflammation may tarry unwontedly long at any one of its stages. It is in unison with the causal conditions upon which hypostatic inflammations depend, that their stasis should be abiding. The congestion is, moreover, often only very gradually brought about.

(*b.*) There are inflammations in which a decided stasis is most probably never arrived at ; the process consisting in prolonged congestion, with a slackening of the circulation, bordering upon stasis. Arrested processes of this kind give rise to exudations, poor in coagulable mate-

rials, serous in kind, and prone to accumulate either by slow degrees or by sudden impulses.

(c.) Chronic inflammation consists in a linked succession, stage for stage, of inflammations more or less acute. Amongst the most marked are those occurring in the vascularized products of an antecedent inflammatory process, more particularly in the pseudo-membranous products of inflammation upon serous membranes.

(d.) Inflammations productive of pus and ichor, whether of external or internal origin, are, for the most part, essentially chronic processes imitative of a secretive function.

In point of degree and extension, inflammations offer the greatest possible variety. For the degree of intensity of the inflammation, the intensity of the external appearances, especially redness and tumefaction, together with the quantity and quality of the products, affords a criterion. This, however, applies exclusively to inflammations due to local external causes. In inflammations kindled out of an internal humoral element, the mildness of the stasis and of its symptoms is often strikingly disproportionate to the quantity and character of its products. Even the former kind is subject to frequent exceptions: thus, purulent exudation, which is commonly regarded as the product of a very intense stasis, not unfrequently occurs in great abundance under slight symptoms.

The extent of an inflammation, that is, its diffusion over one or more organs, depends both upon its occasional cause and upon accessory circumstances. Inflammations evoked by dyscrasial agency, very commonly assault large organs, or at least considerable sections thereof, very frequently several like-named or kindred organs, at once, or in rapid succession,—for example, serous, mucous, follicular formations.

Inflammation is not essentially modified by anatomical differences of organs and tissues. We may here, however, remark that—

(a.) Organs differ greatly in the proneness to inflammation from an internal humoral cause, there being a certain relation of organs to special crases. Frequently, however, peculiar extraneous impulses concur to localize a crasis upon a particular formation; take, for instance, the localization of the puerperal crasis upon the peritoneum, a part enlisted both in the act of parturition and in the puerperal uterine process.

(b.) Vascularized new growths, like normal textures, may become the seat of inflammation, luxuriate in growth, and increase in substance, or else decline and perish. Sloughing and luxuriance of growth are here occasionally concurrent processes. Tender, budding formations are especially apt to become destroyed by inflammation, at the same time that a more vigorous germination is becoming established in their vicinity.

The distinctive characters of regeneration and of suppuration, destructive inflammation, will be considered under the head of Terminations or Issues.

Both the causes and the products of inflammation constitute very important grounds of distinction, and a rigid discrimination is necessary between inflammations from external, and inflammations from internal causes. To the former class belong not alone the inflammations locally engendered by direct external influences, but also those awakened in remote but sympathetically allied formations, through the mediation of

the nerves. To the latter class belong those hyperæmiæ and stases based upon a greatly overwrought condition of the nervous centres, be it irritation or paralysis, and more particularly inflammations arising out of a pre-existent dyscrasial or humoral element.

RELATION OF THE INFLAMMATORY PROCESS TO CRASIS.

A twofold relation exists between inflammation and an anomalous crasis, the latter being either a mere result of the inflammatory process and secondary, or else pre-existent and primary, and the inflammation a consecutive, symptomatic phenomenon,—the localization of the crasis.

1. The undeniable development of a dyscrasial condition of the blood, as the sequel to mere local inflammation produced by external and, it may be, traumatic influences, justifies the inference that inflammation is in itself a dyscrasial process. All doubt as to this point is, however, overcome if we consider—

(*a.*) The qualitative variations in the products of a local inflammation; and still more—

(*b.*) The fact that, where the blood is in the condition of stasis, the elements of the exudation are found preformed within the vessels. The character of the products is thus shown to be mainly dependent upon inbred transformations of the stagnant blood, and more particularly of its plasma.

Inflammatory stasis is thus shown to be no mere passive congestion; effusion to be no mere percolation of an altered plasma simply divorced from the blood-globules. On the contrary, both are shown to imply every variety of conversion, from simple exaltation of the formative process carried on in normal plasma, to the generation of totally heterogeneous combinations and of corresponding form-elements. For the same reason the plasma effused, and the fibrinous matter thrown out as the result of inflammatory stasis, differ from mere extravasated plasma, or extravasated fibrin. The same arguments, in fine, serve for the solution of the question: in what wise does an anomalous constitution of the entire blood-mass result from local inflammation?

Consecutive dyscrasy, as infection of the blood, is brought about—

(*a.*) Through direct readmission of effused matter into the circulation by endosmosis, or through the indirect way of resorption through lymphatics;

(*b.*) Through a more copious reception of the products of inflammation into bloodvessels laid open by external injury, or by ulceration and necrosis;

(*c.*) And (besides these commonly-taught channels) through the ebbing back to the roots of the veins of the altered plasma, issuing from bloodvessels involved in the process of inflammation.

This last mode of infection is decidedly the most common. It is, indeed, the only one possible where endosmosis and resorption of the substances effused are, owing to the density of the latter surpassing that of the lymph or of the blood, excluded. It resembles, on a small scale, that infection of the blood due to inflammatory products directly thrown

out into the cavities and canals of the circulation from their internal membranes.

The interest and the practical importance of the subject warrant us, whilst referring to the chapter on blood diseases and on pyæmia, in remarking here that,—if we except cases engendered by very heterogeneous substances, the result of decomposition within or without the vessels,—infection stands in direct relation to the magnitude and number of the inflamed parts, and, as a consequence, to the amount of inflammatory products taken up into the blood. In opposition to a fermentation theory, we may affirm that a minimum of inflammatory product does *not* suffice to determine a perceptible alteration of its admixture, very small quantities merging in the normal processes of the circulating mass. The most pregnant source of infection is the inflammation of the internal membrane of bloodvessels.

The *character* of the infection and of the consequent crasis corresponds with that of the products of the inflammatory stasis.

Consummated infection of the blood-mass bears towards every inflammation which it may subsequently call forth, the relation of a primitive crasis localized in congenial inflammation.

2. In every crasis, hyperæmia and stasis may occur accidentally. Here, however, we are more particularly concerned with inflammations resulting from a special crasis as their internal and sole cause. To this class belong a multiplicity of anomalous crases, more especially, however, the fibrin-crasis and pyæmia, the former being usually comprised under the so-called phlogistic crasis. Inflammations dependent upon dyscrasial impulses are marked by their preference for particular organs, and, for the most part, by the acuteness of their career, as well as by the rapid formation of their products.

This last circumstance is, to a certain extent, practically demonstrable with respect to the fibrin-crases and pyæmia. In these, the examination of the blood shows that, the same changes occur in its totality, as in any portion of it in the condition of stasis; that both the plasma and the fibrin are constituted identically with the products of the stasis. Herein a source is supplied for the rapid development of the stasis, and, consequently, of the effusion. In the stasis, a simple increase of the processes carried on in the totality of the blood suffices to furnish forth, in exquisite form, a characteristic exudation.

Inflammation of humoral origin is determined, according to the neuropathological theory, by antagonistic palsy of the nerves of the bloodvessels, brought about, at the periphery or at the centres, by the irritating effect of the dyscrasial blood upon the sensitive nerves; or, if passive, by direct paralysis. To such inflammations unquestionably belong many commonly ascribed to an external cause; for example, pneumoniæ. These we believe to be, for the most part, the localization of a crasis modified by atmospherical influences.

The relation of the crasis to particular domains of the nervous system, determines the localization of certain crases in particular organs, almost as a definite rule. Still, the modifying power of concurrent external influences is not to be lost sight of.

Inflammation arising out of humoral elements manifests itself not

unfrequently as a metastasis, exhausting the crasis and resolving itself into a local evil.

The above becomes invested with greater significance when applied to the exudatory processes upon which the production of various heterologous formations rests.

EXUDATION.

The greatest and most marked differences of inflammation are manifested in its products. In inflammations due to a pre-existent crasis, such difference is intelligible enough, the product of the stasis having its germs, in part at least, preformed in the general circulation. It is less clear with respect to inflammations devoid of a pre-existent crasis. Mere local inflammation, however, independent of any dyscrasial materials, will yield products presenting a repetition of many, if not of all, the characters which mark the products of dyscrasial inflammation, such as simple (plastic) fibrinous exudation, fibrino-croupous (pyin-holding) exudation, pus, ichor. Here the question is: wherein are the different characters founded? how elicited by stasis out of normal matter? Their ultimate source can be no other than the stasis itself, of which it might be predicated—

(*a.*) As stasis it promotes, and, within its range, condenses upon inconsiderable elementary materials, the changes and formative processes which take place in the general circulation.

(*b.*) When feeble in intensity it determines mechanically the display of thin, serous, sero-fibrinous, sero-albuminous substances, poor in plastic materials.

(*c.*) Greater intensity and duration occasion relatively, through the continued afflux of oxygen to a given quantity of plasma engaged in stasis, excessive fibrin formation, and ulterior oxidation of the fibrin. Hence result fibrinous exudation, and, as a higher degree of oxidation of the protein (Mulder), croupous constitution of the fibrin, croupous exudates, pus- and ichor-exudates.

(*d.*) *Absolute stasis*, deficiency of oxygen, leads to necrosis of the blood and of the implicated textures,—*gangrene*.

The exudate is, accordingly, now of general, now of local import. Exudates, when formed, suffer various changes through external influences, and take various impressions not inherently theirs; thus not unfrequently entering upon anomalous modes of development. Like blastemata, they do not exude unalloyed; croupous fibrin and pus, for example, being always mingled with a certain amount of so-called plastic fibrin and normal plasma. Nevertheless, the study of exudates in their utmost simplicity of forms is indispensable.

Exudates, indeed, both in their primitive condition, and in the changes which they undergo, irrespectively of impressions from without, afford an insight into the local processes occurring in blood involved in stasis, as also into the processes carried on in the general circulation, that is to say, into the crasis itself.

The most instructive of all exudates are those occurring upon serous and mucous membranes, upon which many, if not most, crases are wont to localize.

The doctrine of inflammatory exudation is, in many points, applicable to the products of other processes of exudation, nor shall we, in the sequel, neglect the opportunity of generalizing in this sense.

Let us pass, in the mean time, to the enumeration of exudates as established according to anatomical research, taking at the same time, a general survey of the various changes which they undergo.

1. *Fibrinous exudation* comprises several varieties, each corresponding to a particular constitution of the fibrin of blood involved in stasis, or of the general circulation. They are, for the most part, not pure, one kind being always alloyed with a proportion, however minute, of another kind, and a certain amount of normally constituted fibrin attaching to all.

(a.) *Simple or plastic fibrinous exudation.*—A grayish, yellowish-gray, or if containing blood-corpuscles, even red, reddish-gray exudate which, in great part, speedily solidifies, into bulky, membranaceous, plug-like, or frame-like coagula, whilst the remainder, where the proportion of blood-serum is considerable, coagulates in the latter into flocculi, thus parting into a solid and a fluid constituent. It presents itself, on a closer inspection, as a clodded, fibrous, diaphanous blastema, of very tenacious properties, the broken surface of which is felt-like and studded with nuclei and nucleated cells. It answers to fibrin 2. It is observed in its purest form in wounds, when these agglutinate and heal by the first intention. Besides this, it occurs upon the pleura, in areolar tissue, in muscles, in bones, upon the endocardium and the internal bloodvessel membrane, in the brain, occasionally, as pneumonia, in the parenchyma of the lung, which either slowly recovers its natural state through absorption, or ends in induration with extinction of the pulmonary texture. Upon serous membranes it constitutes the peripherous coagula lining the inner surface of serous sacs. It frequently enters, in common with a certain proportion of normally constituted fibrin, into the composition of other exudates, as that portion of them upon which a change of texture depends, for example, into the croupous, the purulent, &c.

The following are its metamorphoses, the most remarkable amongst which is its textural conversion.

(a.) It becomes partially or wholly reabsorbed; this occurs slowly, through the instrumentality partly of the serous portion of the exudation itself, partly of a supplementary effusion of serum, succeeding to the resolution of the stasis. These humors furnish the solvent—the corroding media, so to speak, for the solidified fibrin effused, having incorporated which, layer by layer, either in solution, or in a state of minute subdivision, they forthwith become reabsorbed. In like manner, the fibrinous coagula within the vascular system; for example, vegetations, thrombi, &c., are progressively taken up again into the blood.

(β.) It wastes and hardens, with the loss of its fluid part and with lessening of its volume, to a horny, and eventually, perhaps, to an ossified mass.

(γ.) It undergoes a change of texture, commonly, and for the greater part, consisting in an ulterior development of that fibrillation of the blastema which ensues upon the solidifying of the exudate. To avoid

repetition, we refer for the details of this process to the heads—New Growth of Areolar Tissue, and Fibroid Formations.

In rare instances, the new formation of bloodvessels is predominant therein, more especially in exudates upon the arachnoid membrane. In the vicinity of serous membranes, there result serous layers, a new serous sac; in contiguity with bone, bony texture.

The time requisite for these transformations is scarcely definable in a general way. Under favorable circumstances, they are wont, even in voluminous exudates, to attain an advanced or nearly completed state, within the space of six weeks.

The constitution of the exudate corresponds, as before stated, with that of fibrin 2, having either been acquired in the stasis or pre-existed as a blood crase. It is a quantitative anomaly consisting in an exaggeration of the formative processes which occur in normal plasma,—and especially in the locally and generally increased generation of a fibrin (hyperinosis) marked by its coagulable and plastic properties.

(*b.*) *Croupous exudation* has several varieties, dependent upon a qualitative impairment of the fibrin. It is, with the utmost impropriety, confounded with the former and its kindred crases. It is marked by a high degree of coagulability; by a yellow, or greenish-yellow coloration; by its opacity; by its inorganizable nature; by its early tendency to break down, to liquefy; frequently by a corrosive, texture-softening power. The quantity of serum simultaneously thrown out is relatively insignificant.

The croupous process of exudation and its product are further distinguished,

1. By the commonly excessive, exhausting, quantity of the exudate, and its extension over wide ranges of organs and textures.

2. By the rapidity with which the effusion is brought about, where the stasis depends upon a pre-existent crasis.

3. By the often slight vascularity of the diseased texture; a circumstance due, it may be, to the blood-corpuscles not appearing prominently in the opaque, over vigorous plasma, or else to the exudate, by its excessive quantity, soon leaving the bloodvessels exsanguine and collapsed.

4. By less adhesiveness.

5. By considerable fattiness of the exudate.

The principal *metamorphosis* of the croupous exudate is the aforesaid breaking down, and liquefying to a fluid more or less analogous to pus, which constitutes the so-called *liquid, purulent exudate*.

This metamorphosis first of all affects the solidified blastema intervening between certain form-elements of the exudate; only in an adherent portion of organizable fibrin does the latter undergo a change of texture—namely, to areolar tissue. When liquefied, it may be wholly reabsorbed, or may leave a residue in the shape of a fatty, curd-like, cretaceous pap, or, lastly, of a glutinous fluid, abounding in free fat and in salts of lime (in elementary granules, granulated cells, cholesterine crystals), which speedily thickens into a cretaceous concrement.

(*a.*) *Croupous Exudation*, an abundant, proportionately to its contents in blood-globules or in blood-pigment, more or less red (red hepatization of lung), or grayish-yellow mingling with green, opaque exudate,

consisting of a sod-like, fibro-laminated, or striated membranous base-
ment, a large proportion of dotted substance, nucleated formations, dull
granulated nuclei and nucleated cells. The nuclei are not influenced by
acetic acid, beyond some little shrivelling, together with a sharpening
of their contours. This answers to the constitution of fibrin 3. It lique-
fies first in its basement mass, with change of texture of any adherent
portion of organizable fibrin, to a pus-like fluid.

(β.) *Croupous Exudation.* The above-mentioned characters, espe-
cially the opacity and the greenish coloration, are here more strongly
developed. It consists, together with an amorphous blastema, of nucleus
and cell-formations, more or less akin to pus-nuclei and pus-cells, and of a
predominating quantity of granulated substance. It adheres loosely to
the exudation-surfaces and deliquesces rapidly. It answers to fibrin 4.

These exudates occur most of all upon membranous expansions, upon
mucous membranes (as the well-known croup), upon serous and synovial
membranes, and in the substance of the lung. Again, they occur in
areolar tissue, in the pia mater, in the convexity of the cerebral hemi-
spheres, upon the endocardium and the internal bloodvessel membranes,
in parenchymata, and upon the surface of both internal and external
sores. Almost all the pneumoniæ, except those ending in slow resorption
or induration of their product, belong to this class, and pre-eminently
those in which the lung becomes enormously distended with a very copi-
ous, rapidly deliquescent yellow effusion, the stage of red hepatization
being, in Hodgkin's opinion, here altogether wanting.

As the exudates break down, they exert, especially after long-con-
tinued contact, the aforesaid corrosive liquefying influence upon their
substrata, occasioning ulcerous loss of substance, pulmonary abscess,
destruction of serous membranes, ulcerous perforation of the thoracic,
the abdominal, parietes, &c.

(γ.) *Croupous Exudation.*—*Aphthous Exudation*, a yellow, green-
ish-yellow, dingy-grayish, opaque product, wont, upon surfaces, to
solidify into tough membranes, and then melt down, reducing the impli-
cated textures to the same condition. This said product consists some-
times in simple destruction of the textures, sometimes in a blending into
a variously discolored, fetid, ichorous pulp, or into a tenacious, greasy
slough, which tears like tinder.

These exudates affect, with especial frequency, the mucous membranes,
particularly those of the alimentary canal, of the urine bladder, of the
female sexual organs, with their follicles; secondly, external sores and
ulcers; thirdly, the common integuments. Under this head belong
thrush or aphthæ, diphtheritis, exudates upon the intestinal mucous
membrane of the intestines, and of the colon in particular as represent-
ing one form of dysentery, and of the uterus after childbirth; corroding
exudates upon external and internal wounded and ulcerated surfaces, for
example, on the base of the typhous ulcer, white gangrene of the com-
mon integuments, hospital gangrene.

Croupous exudation is occasionally the product of an intense inflam-
matory stasis, unconnected with any pre-existent crasis. It is, however,
much more frequently the product of a stasis in which the croupous
crasis has suddenly become localized.

(c.) A special form of fibrinous exudation is the tuberculous—the true

fibrino-tuberculous. It is a repetition both of simple, and, when engendered by inflammation, of croupous exudation. It is characterized by a proneness to tarry long in its primitive, crude state, and eventually to soften down.

It exudes almost, if not wholly, pure, or else blended with a certain proportion of organizable fibrin. In the former case it represents a homogeneous gray, or yellow, curd-like, brittle, fissured mass; upon serous membranes, a similar uniform layer, of uneven stellate surface. In the second case, the tuberculous matter is imbedded in the shape of more or less crowded tubera clusters, or larger masses, within the other portion of the effusion, which has attained to various grades of textural development. In the last-mentioned case, more especially, tuberculous exudation is, upon serous membranes, always characterized by clustering of the granules.

Tuberculo-croupous exudation affects serous and mucous membranes, evincing a preference for those of the bronchial, of the alimentary, canal, of the uterus, and of the cavity of the tympanum. It invades the parenchyma of organs at all points, but most of all that of the lungs, as infiltrated tuberculous pneumonia of the lobar, or still more commonly the lobular form, the substance of lymphatic glands; lastly, the interior of follicles, and particularly the Peyerian capsules.

There are cases in which it may be but the local produce of a stasis. More commonly, however, this stasis is itself a localization of the tubercle crasis, and the tuberculous exudate the prodcct of general dyscrasial processes.

2. *Albuminous exudation* occurs united to a proportion of fibrin as a fibrino-albuminous; or as a mere albuminous; or again, mingled with a certain proportion of serum, as a sero-albuminous exudate.

Albuminous exudates are marked by their fluidity, by their tenacious, ropy consistence, often by their abundance. They are colorless and resemble a thick synovia; or milk-white and opaque; or again blended with croupous fibrin and of a yellowish-white. Their turbidness, and also their thick porridge-like nature, are due to their quota of form-elements (elementary granules, nuclei, cells), to their proportion of fat, and to fatty conversion (granule-cells); or again, they are due to an act of coagulation, the albumen assuming, by virtue of a chemical conversion of the entire exudate, the coagulate form. Coagula of this kind are for the most part soft, flocculent, forming, upon serous membranes, velvety deposits,—not unfrequently as the inner coating to a peripherous fibrinous coagulation.

The form-elements, present in various amount in the albuminous exudate, are identical with those found in the fibrinous exudate, nucleus and cell exhibiting themselves in multiform variety, ending with the perfect pus-cell.

It is very rare for albuminous exudates to assume at once—that is, immediately on becoming effused, the solid form.

The changes which albuminous exudates undergo, vary with the character of the albumen. In the one case, they enter into a progressive transformation of texture, which in fluid albumen obeys—in solidified

albumen evades—the laws of the cell theory, thus determining hyper-
trophy of the areolar tissue, and induration and extinction of the paren-
chyma, for example, in the lungs.   In dyscrasial constitution of the albu-
men, on the other hand, they have a decided tendency to liquefaction, to
fatty conversion, whereby they become either fitted for resorption, or
endowed with a corrosive power.

Their appearance is for the most part connected with a crasis, involv-
ing either a simple predominance of albumen (deficiency of fibrin) in
the blood, or concurrently a dyscrasial condition of the former.   To the
first description belongs, for instance, the venous diathesis (Venosität),
the concomitant of heart disease, of infancy, and of extreme age, of
atrophy, of defibrination.   The products are represented by colorless,
adhesive, pasty exudates, poor in form-elements, and, owing to the often
low intensity of the stasis, largely diluted with serum ;—sero-albuminous.
To the second description belong the crases which attach to cutaneous
affections, to typhus and the like, with their dull whitish effusion ; and,
again, the anomalous quality of albumen pertaining to the fibrino-croup-
ous crasis, to pyæmia and the like, with their pus-like effusion.   The
stases are often of the asthenic or hypostatic kind, and run a protracted
course.

3. *The serous, dropsical exudate.*

Serous effusions are, generally speaking, either *merely serous* (blood-
serum) ; or again from their containing a larger proportion of albumen,
albumino-serous ; or, lastly, owing to an admixture of fibrin, *fibrino-
serous.*   This gives rise to several important distinctions in their physical
bearings.   The mere serous effusion is a thin, watery, limpid, colorless,
or pale yellowish, now and then reddish-yellow fluid, salt to the taste,
and containing little, if any, albumen.   A large proportion of albumen
renders it tenacious, like a thin synovia.   An admixture of fibrin mani-
fests itself upon serous membranes as a peripherous coagulum of incon-
siderable thickness, as a villous deposit, as a filamentous, wide-meshed,
network or finely membraned honeycomb, or as a flocculent cloud within
the serous fluid.   Or again, it may, in the shape of the so-called spurious
fibrin, which solidifies tardily, perhaps only after cooling and coming in
contact with the air, appear in the effused serum as soft, jelly-like, trans-
parent, coagulate pellets, which are often found lodged within the afore-
said true fibrinous network, or honeycombed structure.   Fibrino-serous
effusion may be said to be invariably the product of an inflammatory
stasis.   It is simply fibrin-exudate, with a notable preponderance of
serum.   It might well be denominated *fibrinated dropsy.*

Albumino-serous effusion is sometimes, like that previously described,
the product of a not very intense, often of a protracted, stasis or simple
congestion.   As an example, may be cited the (inflammatory) œdema,
encompassing the range of an inflammation.   It occurs more commonly
independent of the latter, *in the albumino-serous crasis,* as general
œdema.

Mere serous effusion is the result of an excess of serum in the blood—
the serous crasis ; or else it is the product of acute and more frequently
of chronic hyperæmia.   In the latter case, the effusion seems to origi-
nate less from the capillaries than from the small thin-membraned veins.

It represents genuine dropsy, and does not at all imply a foregone inflammatory stasis.

Serous effusion, as such, is not organizable. Albumen and fibrin, however, when blended with it in sufficient quantity, are susceptible of a somewhat tardy, structural change. It relaxes and tumefies the textures on becoming imbibed, destroys their contractility, and by long-continued contact exerts, more especially upon the muscular fibres, a remarkable power-bereaving influence.

4. *Purulent and ichorous effusion.* These range immediately with fibrinous and albuminous exudation, as also with their fibrino-serous, albumino-serous combinations. Purulent effusion seems, however, to be more especially akin to the fibrino-croupous exudate.

The importance of these products renders it desirable to discuss them at greater length.

PUS, ICHOR.

No product of disease has, perhaps, been the subject of such zealous research as pus and ichor, and yet nowhere has a greater number of shortcomings been overlooked or glossed over than here. These we may be incompetent to remedy; we may, however, render some slight service to pathology if we can succeed in simply directing attention to them.

There are so many kinds of purulent-looking fluids, and there is so great an affinity of these fluids amongst each other, that, for the sake of discrimination, it is indispensable to establish one standard form of pus. Such a standard form is furnished in the pus of granulating, healing wounds, as well as in that of certain abscesses.

This *normal pus* is a homogeneous, cream-like, fatty, glutinous fluid, of a yellowish color, of a flat, sweetish smell and taste, of a specific gravity of from 1030 to 1333 (Vogel), and when recent, of alkaline reaction.

It consists essentially of *pus-serum*, with certain form-elements, these being, besides molecular granules (elementary granules), the *pus-nucleus*, and the *pus-cell*. To these is to be added the pus-placenta, of which more hereafter.

The *pus-cell* is a spherical or oval—now smooth and even, transparent, thin membraned—now granulated and opaque, nucleated cell, which, under a magnifying power of 400 diameters, appears colorless, or faintly yellow, and measures from $\frac{1}{100}$ to $\frac{1}{80}$th of a millimetre in diameter.

Its granulated nucleus, firmly attached to the cell-wall, is, in the translucent cell, visible without the aid of artificial expedients. In the granulated cell, on the contrary, it is rendered indistinct, if not totally obscured, by the contents of the cell, but is readily discernible on the application of acetic acid. It generally occupies from one-half to two-thirds of the cell's cavity, and in rare instances almost fills it up. Generally speaking, it is single; not unfrequently, however, it is manifestly composed of from two to five smaller corpuscles. Normal pus only rarely contains larger cells, with two, three, or four nuclei. Under

the action of acetic acid, each pus-nucleus being brought out with sharper contours, as a spherical (according to Vogel, cupped) body, presents the well-known characteristic phenomenon of indentation and eventual split-ting. In other words, the nucleus, after passing through sundry modi-fications of shape, down to that of a trefoil, finally breaks up into two, three, or four sharply-defined corpuscles, no further soluble in acetic acid.

Besides the cell-inclosed nuclei there are present free nuclei. These are in like manner either single (perfected), or made up of from two to five corpuscles, and they exhibit the same phenomenon of indentation and splitting, when treated with acetic acid.

The molecular granules are present in various numbers, some scat-tered, others grouped together.

The contents of the cells are in some cases limpid, in others, owing to very minute granulations, nebulous. It is very common to find one compact group of pus-cells presenting every known gradation in the quality of their contents.

The development of the pus-cell is easily demonstrable, falsifying the assertion that the nuclei are artificially produced by chemical agency. For the most part from two to five of the larger molecules associate themselves into a group, and constitute, thus aggregated, an imperfect nucleus. By and by they coalesce, and present a simple, finished nu-cleus—a fabric reducible, by the agency of acetic acid, to the very same elements.

The nucleus now becomes surrounded, often immediately, with a cell wall, so closely fitted at first, as to require the endosmotic agency of water, or dilute acetic acid to disconnect it, and render it cognizable. Many nuclei, however, become previously endued with a delicate nebu-lous deposit, which by and by puts on a circumscribing cell-envelope, and assumes the contour of the cell.

These formations are, to a greater or smaller extent, always dis-cernible in genuine pus, in the progress of germination.

Cells devoid of nuclei,—clear, transparent cells, which have to create a nucleus out of their own materials, are rare.

The phenomena of endosmosis and exosmosis bring out the pus-glo-bules with great clearness.

The chemical relations of the pus-cell are not without their weight in reference to its constitution, and to its recognition.

Dilute acids, for example, dilute hydrochloric, oxalic, tartaric, but especially acetic acids, have the effects of tumefying, loosening, attenua-ting, bursting, without entirely dissolving the pus-cells, whilst upon the nuclei they produce the above-mentioned appearance, first, of greater distinctness of outline, then of indentation, and lastly, of disruption.

Caustic alkalies and their carbonates convert the pus-cells into a jelly-like, granulated substance.

Thin solutions of certain saline substances, as for instance, of chloride of sodium, hydrochlorate of ammonia, nitrate of potash, iodide of potas-sium, with many others, cause first the disappearance of the sheaths, and secondly, the swelling up of the nuclei into a shapeless grume.

A solution of borax acts like the alkalies, only less rapidly. Metallic

salts, alcohol, tannic acid, &c., which coagulate fluid albumen, render the pus-globules shrunken, nebulous, and opaque.

In the blood, in urine, in mucus, and in saliva, the pus-cells are preserved unchanged ; bile, on the contrary, occasions a disappearance of the sheaths, and a bloated aspect of the nuclei.

From these facts, and from further experiments in the same direction, Lehmann and Messerschmidt draw the following conclusions :

1. The sheath of pus-cells, turgescent in acids, soluble in solutions of caustic alkalies, and of their saline conjunctions, is identical with a protein-compound which may be artificially produced out of albumen, deposited by water, and redissolved by alkaline salts, and acetic acid—a modified albumen, poor in salts, constituting a transition stage to fibrin —fibrin *a*.

2. The nucleus, insoluble in acetic acid, soluble in solutions of alkalies, turgescent in solutions of salts, a protein-compound similar to the venous fibrin, turgescent in salines—fibrin *b*.

3. The third substance, namely, the molecules accompanying the pus-cells, forming part of the contents of the opaque granulated cells, and even exhibited in the nuclei (the nucleus-corpuscles of Lehmann and Messerschmidt), are uninfluenced by alkalies or borax, and are regarded by Lehmann and Messerschmidt, as a substance analogous to the essential constituent of horny texture. They, however, partly consist, as Vogel rightly maintains, of fat.

Besides these elements, pus not unfrequently contains cholesterine-crystals, crystals of ammonio-phosphate of magnesia, animalcules, &c.

Like Henle, we have been unable to satisfy ourselves of the above-mentioned effect of alkaline salts or of borax solution. In the changes wrought by the application of thin solutions, we recognize the phenomena of endosmosis down to rupture of the sheath of the pus-cell ; in the changes wrought by the application of saturated solutions, a shrivelling thereof.

The pus-serum in which, when at rest, the pus-cells gravitate, has the composition of blood-serum, with some difference, however, in the relative proportions of its constituents ; fat, for example, predominating. With respect to pyin, to which we shall afterwards have to recur, our belief is that it is not a constituent of normal pus at all.

Pure pus we believe to be an albuminous exudate, out of which, like other elementary bodies, the pus-cell becomes developed by virtue of a specific conversion. This process, the so-called development of pus out of fluid blastema, occurs upon surfaces—upon mucous membranes, upon the external skin, upon open wounds, in abscesses. This pus exudate, however, enters frequently into combination with fibrinous exudates of different kinds.

Of these, we could first mention the combination with the fibrino-croup-ous exudate, because this furnishes the base of the so-called development of pus out of solid blastema. Whether, or how far, we participate in this view, will appear in the sequel.

Both upon surfaces and within parenchymata, a solidifying fibrinous blastema is frequently thrown out. A careful inspection will show, imbedded in the solid basement, as also floating in the sero-albuminous

fluid, molecular granules and genuine pus-nuclei and pus-cells. Together with these are always found a few nucleus and cell-forms, which vary in their relations, more especially to acetic acid. The solid basement manifests itself as croupous fibrin, which liquefies, incorporates the aforesaid elements, and is distinguished by its great abundance of the most minute molecules. This combination of pus with fibrino-croupous exudate, constitutes the so-called solid pus, or the pus-plug, and is that upon which the breaking down, the softening, of inflammatory induration, abscess, and the like depend. The pus-cell is always developed out of the sero-albuminous moisture pertaining to the fibrin-exudate, never out of or at the expense of the latter itself, the liquefaction of which implies a metamorphosis. This pus, in virtue of its constituent, croupous fibrin, contains pyin.

Another combination of the pus-exudate, is that with plastic fibrin exudate. This frequently, but not invariably, furnishes in pus the basis of new solid textures, of regeneration, of the cicatrix. The fibrin-exudate determines the pus-placenta.

Pus may, without impairment of its primitive character, present various anomalies, for example :

(*a.*) Watery pus ; the pus-cells having become bloated in the preternaturally thin medium.

(*b.*) Preternaturally saturated pus-serum, as containing more saline and albuminous matter. The pus-cells appear less turgescent, smaller, shrivelled, denticulated.

(*c.*) Pus which has become acid exhibits the nuclei more distinctly, more sharply defined, and, it may be, in a slight measure ruptured, within a transparent membranous sheath.

(*d.*) Various admixtures, as blood, mucus, epithelial, and other textural débris. It is to be remarked, however, that, by certain admixtures, for instance, of fæcal matter, of decomposed urine, and again by its acid conversion, pus may become transformed into a corrosive fluid, and its secreting texture goaded into the production of ichor.

The nearest approach to pus is found in broken-down fibrino-croupous exudates. It has been seen that these frequently enter into a combination with true pus-effusion, and the liquefaction of the fibrino-croupous elements in pus-effusion constitutes what is termed the development of pus out of a solid blastema. Broken-down, pus-like, fibrino-croupous exudate is always marked by its fluid parts holding in suspension a large proportion of the most delicate nebulous molecules, and is distinguished from pus by the relations of the coexistent nuclei and cells. These exudation elements, namely, manifest, as we have stated, on the one hand, an insensibility towards acetic acid, under the influence of which, by an evident condensation and shrinking, the cell-walls and contours of the nuclei are brought more distinctly into relief; or it may be that the cell-walls and cell-contents are rendered clearer, whilst the nucleus becomes condensed and more sharply defined.

On the other hand, these cells approximate to the character of the pus-cell, the nucleus exhibiting, with the disappearance of the cell-wall, to a various extent the phenomena of denticulation and splitting. It is, in our opinion, the broken-down fibrino-croupous exudate, either alone

or blended with true pus, that constitutes the pyin-holding pus form. It has been stated that these pus-like exudates frequently manifest a corrosive, deliquescent influence upon the textures. Not being organizable, they are extremely prone to further decomposition, and to assume the nature of ichor. They furnish forth the majority of cases of internal suppuration, of constitutional pus deposits, of abscesses.

Ichor, which, in broken-down croupous exudates, often closely resembles pus in appearance, is distinguished from the bland nature of true pus, by its corroding influence upon the textures, and upon the form-elements developed out of its protein substances. It is only under such a state of things that a fluid can be recognized as ichor. That met with after death varies infinitely. A chemical examination of it embodying what is essential, and simplifying what seems differential, is still wanting. Its degree of corrosiveness varies equally. Ichorous exudates are now thin, serous fluids; now albuminous, viscid, limpid or flocculent, emulsive and fatty, thickish, colorless, or yellowish, yellowish-green, puriform, whitish, creamy. Or, owing to the presence of blood-corpuscles and of blood-pigment, they are of variously shaded red, dingy brown, greenish-brown, chocolate-colored. Again, they are ammoniacal, hydro-sulphuretted, rancid or sour-smelling, acid or alkaline, and apt to produce upon the skin of the dissector a tingling or smarting sensation. These fluids, minutely examined, are found to contain variously sized elementary granules, down to the finest molecular mass, nuclei and cells, of the character of exudation and pus-cells, partly stunted in their development, partly owing to the saline, alkaline, or acid condition of the ichor, shrivelled, jagged, lax, diffluent, the pus-nuclei being in the act of denticulation and splitting.

They further contain fibrinous coagula of various kinds, in different grades of spontaneous reduction into pulpy masses, coagula out of casein and pyin substances.

Finally, they yield crystalline salts and textural débris in the act of breaking down, blood-corpuscles, animalcules, &c.

Even ichor enters into combination with fibrinous exudates, especially the fibrino-croupous; and, just as in the case of pus, there is, besides the fluid product ichor, another ichor developed out of consolidated blastema. Having now described both pus and ichor, this appears to us a proper place for the establishment of certain marks necessary for a due discrimination between the two.

The bland properties of normal pus are acknowledged; but how does this characteristic tally with the manifest destruction of tissues implicated in the formation of pus? The following remarks may tend to throw some light upon this point:

(*a.*) The destruction attendant upon pus-exudation is limited to necrosis of the textural elements involved. But this necrosis is due to the intercepted supply of blood and to forcible disjunction; not to that chemical corrosion and that resolution of the textures which result from ichorous discharge.

(*b.*) That the bland nature of pus is so frequently questioned, arises from products being so often regarded as pus, which are not so in reality, and which either originally possessed, or have acquired the pro-

perty of corrosiveness. Such products very commonly form the contents of shut abscesses. Normal pus very often acquires a corroding property through long seclusion within abscesses, which, when opened, forthwith secrete a normal, bland pus.

One of the most remarkable phenomena in the process of suppuration, is the formation of *flesh granulations*. These granulations present, with reference to their character and further development, two marked distinctions:

(*a.*) They consist, together with a small proportion of intercellular or bond substance, of primary cells. These cells emerge, together with the pus-cells, from a common albuminous blastema, and out of them are called forth, conformably with the laws of the cell theory, those elementary fibrils which ultimately compose the cicatrix.

(*b.*) They consist of a fibrinous blastema which, exuding conjointly with the pus, solidifies upon the suppurating surface, and yields, by immediate splitting, the fibrous texture of the cicatrix. Into both these scar-bases enters a new generation of bloodvessels, answering to those of abiding suppuration. They determine the healing of wounds by the second intention.

The formation of flesh-granules is not decisive evidence of a bland, benignant pus. It may accompany, and even luxuriate under, the production of ichor. In the one case, however, the granulations are marked by their durability, and by their further textural development, whilst in the other, they form but to be corroded and redissolved by contact with the ichorous fluid.

Flesh-granulations accompanying the production of pus in or upon heterologous growths, have, for the most part, the significance of textural elements of such growths.

The tendency of pus (and of ichor) to vent itself externally, is commonly overrated. Pus deposits are often deeply encysted within organs and a passage to without often needs to be made artificially, in order to prevent the fluid from burrowing.

The assertion that extensive exudates are especially liable to become converted into pus, is ill founded, if magnitude of the exudate be the assumed condition of such conversion. Doubtless what led to the opinion, was the liquefaction of the commonly very massive croupous exudates, together with the circumstance that pus-blastema, either pure or combined with croupous fibrin, very often becomes effused in great quantity. Apart, however, from quantity, the puriform quality of an exudate is invariably due to inflammatory stasis. This alone, and not the quantity, can determine the development of the elements of pus.

Purulent and ichorous exudates are met with not alone in the localities assigned to normal pus. They often occur in great quantity upon serous and synovial membranes, and in areolar tissue, more especially the subcutaneous and submucous, as also in certain of the more lax, deep-seated collections of this tissue, for example, in the mediastina, in the posterior circumference of the cæcum, around the rectum, and the like. Lastly, they take place upon mucous membranes, within soft parenchymata, and in bone.

The following are the metamorphoses which the said exudates, if not excreted, undergo:

(*a.*) *Transformation of texture*, appearing, after the process above described, in the form of so-called granulations.

(*b.*) *Dissolution* affects both the form-elements (cells, nuclei), and the fluid intercellular substance, in the shape of various unknown chemical transformations. Under this head may be brought the septic decompositions, suffered by these exudates under peculiar circumstances; for instance, through long stagnation, through contact with the atmosphere, through the effect of medicinal substances, and the like. These may cause the degradation of bland pus into ichor, and of ichor a step lower in the scale.

(*c.*) *Fatty conversion* in the formative process of granule-cells, frequently combined with the simultaneous deposition of the salts of lime, (cretefaction).

(*d.*) *Resorption.*

The manifold ways in which pus substantively, or as pus-serum, enters the circulation, and there occasions pyæmia, cannot at all concern us here. They were before adverted to, and will be further and more amply noticed under the head of Pyæmia.

Here we have to consider the resorption of pus in a more restricted sense, and irrespectively of pyæmia.

This resorption can apply only to the serum or plasma of pus. To pus or ichor in their totality, that is to say, their form-elements included, it can only apply after they have undergone liquefaction or fatty conversion. Upon the mode of liquefaction depends, in the case of pus, the nature of the consecutive phenomena. If it consist in a putrid decomposition, the noxious effects of septic poisoning of the bood may ensue.

Pus may either become completely reabsorbed, or leave within its former nidus a residue of fat in the shape of discrete or aggregate fat molecules and cholesterine crystals, within a glutinous fluid, or in the shape of chalk incrustation and concretion.

The depot of pus and of ichor (abscess) heals, through the subsidence of pus and ichor production, and through the organizable products of inflammation existing in the walls of the abscess, with new supplementary ones, undergoing transformation into vascularized textures. Pending this act, the contents of the depot are in the manner before specified, entirely or partially reabsorbed. According to the measure of resorption, the walls of the abscess shrink together and ultimately coalesce into a solid cicatrix, or else include an inspissated fatty or chalky, or lardo-cretaceous residue of the primitive contents.

Pus and ichor are the product, at one time of a mere local inflammatory stasis, at another time of a localized pre-existent crasis, namely, general pyæmia. In the latter case, the production is characterized by the rapidity of its occurrence.

Suppuration in open abscesses and upon granulating sores is peculiarly chronic in its course. This process manifests itself as a protracted stasis, communicated from the original textures to the embryonic new growths which the granulations with their newly acquired vascular apparatus have given rise to. Taken in another point of view, the granulations appear to stand to pus-formation in a relation which earlier pathologists signified by the terms *pyogenous membrane*, a pus-secreting apparatus.

Looking at the often slight intensity of the inflammatory symptons, the analogy between a granulating wound (abscess) and a secreting organ, —between pus and a secretion prepared by those elementary bodies, cells, appears so great, and the current comparison with a mucous membrane and its product so apt, as forcibly to recur to us, even at the present day. Thus, no sooner have the growths which presided over the secretion—in other words, the granulations—become exalted into textures, than the secretion itself fails.

Referring certain particulars connected with the anatomical doctrine of exudates to the head of crasis, we have still to consider, as kindred with ichorous exudates, *diffluent exudates*, and with them *hemorrhagic effusion*.

(*a.*) *Solvent exudates.* Akin to ichorous exudates, they are marked by their destructiveness to subjacent textures, by the obviously solvent character of their effects, and by the absence of any outward tokens to denote their mischievous character.

These are products which, owing to the corrosion of textures accompanying the very act of their exudation, are rarely to be met with in their simplicity, products which display immense variety in their physical properties. As the extreme limits of a long series, we find, on the one side, a coagulable, fibrinous exudate which has the effect of slightly corroding the subjacent membranous substance. On the other side we have a thin exudate, variously discolored, which reduces the textures, extensively, to a dingy brown, chocolate-colored, inky (hemorrhagic), or greenish, pulpy, tinderlike, fetid, slough. This last-mentioned exudate represents the processes which Boër described in the uterus as putrescence, a term quite deserving of application to the same condition in other parts. Midway between these two extremes, we encounter the most remarkable—however seemingly insignificant—thin, serous, sero-albuminous, tenacious, paste-like, sero-purulent, almost colorless, or again yellowish, reddish-yellow, exudates, in contact with which the textures are resolved according to their degree of injection, into a pale, or into a more or less deeply-reddened pulp.

Their seat, always diffuse, is most particularly the mucous membrane of the intestinal tract, and most commonly of the colon, not rarely of their follicles (in the shape of diarrhœa or dysentery), and, lastly, of the uterus, as puerperal affections following childbirth. Of the principle upon which this liquefiant destruction of the tissues depends, nothing is known beyond its frequent acid reaction, nor has any crasis corresponding to it been recognized.

(*b.*) The *hemorrhagic exudate.*

It is indispensable in the first place to discriminate between exudates reddened by blood-pigment only, and those which contain substantive blood, that is, blood-corpuscles.

The former are met with in all dyscrases, both acute and chronic, in which, owing to defibrination, to decomposition of the fibrin, or to diminished proportion of salts, blood-pigment is transferred from the blood-corpuscles to the blood-serum. Thus, exudates occurring during the progress of scurvy, of typhus, of gangrene, of the drunkard's dyscrasis, of putrid exanthemata, are stained with adherent blood-pigment. Those

containing blood in its totality, and the red color of which results from blood-corpuscles, are the true hemorrhagic exudates.

Holding fast this distinction, we shall be enabled partly to infer from physiological reasoning, partly to prove by the exact method, the origin and import of hemorrhagic effusion.

We have seen that, in every inflammation, at the stage of congestion and stasis, there occur extravasations of blood, proportionate in extent to the vascularity of the organ, to the magnitude of its congestion and stasis, and lastly, to the laxity and vulnerability of its texture. That this bleeding takes place out of lacerated, or in somewise opened vessels by extravasation, and not by transudation, is evident from the absence in the walls of bloodvessels of pores equal to the transudation of blood-corpuscles. This is the rule with respect to hemorrhage in textures like those of the brain or the lungs. The difficulty is, and always has been, to explain hemorrhagic effusion occurring upon serous membranes,—a formation so given to effusion in no way hemorrhagic. Upon this point it is to be observed :

(*a.*) A primitive genuine hemorrhagic exudate (not merely blood-stained), if it really ever occur upon serous membranes, occurs only as a rare exception.

(*b.*) Hemorrhagic exudates upon serous membranes are, almost without exception, the result of hemorrhage from the bloodvessels of a spurious membrane in the act of becoming organized ; in other words, from the product of a previous inflammation of the serous tunic. This hemorrhage may be an independent act, or it may be the concomitant of inflammation propagated from the serous coat to its pseudo-membranous duplicate. This is, in fact, usually the case. Such exudates are secondary ones.

The facility with which hemorrhage takes place from these new growths, is explicable on the ground of their imperfect organization, both as regards change of texture and the development of bloodvessels. An inflammation early set up in such a new formation, encounters a lax, soft, lacerable growth, involving an incomplete, soft, and delicately membraned vascular apparatus, with anastomoses as yet unclosed ; bloodvessels which, when urged into congestion and stasis, readiy give way, or possibly force a passage from their free and as yet unanastomosed ends into the substance, and through this into the cavity, of the serous membrane.

Accordingly, under these stereotype conditions, hemorrhagic exudation is precisely what extravasation is during the course of an inflammation in the laxer textures, namely, *exudation plus hemorrhage.*

Hemorrhage being, however, the consequence not alone of mechanical laceration, but also of a softening or a corrosion of the vessels, it is intelligible how ichorous exudates, and the solvent exudates generally, may put on a hemorrhagic character.

Hemorrhagic exudation seems to stand in an especial relation to tuberculosis, and is dreaded chiefly because the latter is assumed to be its source. It is important to be clear upon this point:

(*a.*) It is quite true that it is very frequently a partially tuberculized

new growth (pseudo-membrane) in which the hemorrhagic process occurs. Still there are very notable exceptions to this.

(*b.*) Tuberculized growths appear to be peculiarly liable to inflammation; tubercle being wont to set up reactive processes of inflammation everywhere in its circumference.

In this way the hemorrhagic inflammatory process often concurs with local tuberculosis, without directly depending upon the tuberculous crasis. That tuberculosis acts as the source of hemorrhagic effusion is rendered probable by experience, but it is by no means proved.

In like manner, hemorrhage and hemorrhagic exudation break forth in the midst of carcinomatous growths, or in pseudo-strata, of the same character, upon mucous membranes or within serous sacs.

In quantity, the extravasate mingling with the exudate varies considerably, and it may be either intimately blended both with the coagula and with the fluid portion of the exudate, or separated from it in the shape of pellet-like clots.

Apart from the aforesaid conditions of its appearance, and of its relation to tuberculosis, it is of evil omen only in proportion to the loss of blood entailed by it, and to the previously reduced vital strength of the patient.

The hemorrhagic exudate is not organizable, or only very tardily so. This refers more particularly to the exudate; the blood-corpuscles after long remaining unaltered at length become dissolved, leaving their pigment to undergo the changes elsewhere described. The exudate portion, answering to the character of its base, very commonly retains its rudimental condition, which in the majority of cases is tuberculous.

It would seem advantageous, before concluding, to revert to a few points already touched upon, relative to the habitudes of exudates in and upon diseased structures.

Exudates are deposited more or less uniformly between the elementary parts of textures. This is contingent upon the more or less uniform density and cohesion of the textures, as also upon the number of bloodvessels present, and their mode of distribution; for example, the striated exudates, following the linear arrangement of the bloodvessels in tendons and ligaments.

When copious effusion takes place suddenly and violently in the laxer structures, for example, in the brain, the exudate becomes established through the forcible separation and laceration of the natural textures.

In the inflammation of membranes the exudate is, as we have stated, for the most part thrown out upon the free surface. In the inflammation of glands similar effusion takes place into their respective cavities— the uriniferous tubules, Malpighian bodies, and the like.

Coagulable exudates solidify upon the surface of inflamed membranes, and are commonly termed spurious membranes. Upon serous membranes they occasion agglutination of the serous surfaces.

ISSUES OF INFLAMMATION.

The so-called issues of inflammation comprehend a variety of processes. They concern either the inflammation itself or its products and

the involved structures; that is to say, they embrace, in the latter relation, the changes which both the inflammatory products and the textures themselves undergo.

To the former category belong:

1. *Resolution or dispersion of the inflammation.*

To the second belong:

2. *Reliquefaction and resorption* of the inflammatory products.

3. *Abiding of the inflammatory products in various forms*, including, amongst others, the issue in induration, and in inflammatory hypertrophy.

4. *Suppuration*, ichorous, ulcerous destruction of textures.

We cannot ourselves regard suppuration as an issue of inflammation. Wherefore we have treated of pus and ichor as of products of inflammation, under the head of " exudates."

Of the issue of inflammation in gangrene, of so-called inflammatory gangrene, we cannot well treat separately. We therefore refer the consideration of this point to a subsequent chapter, to be devoted to the subject of gangrene.

1. *Resolution of the inflammation.*—Issue in *resolution* relates directly to the inflammatory process. It consists in a cessation of the latter, previously to any act of effusion; that is, in a reduction of the existing stasis.

As determining conditions, we may adduce:

(*a.*) Cessation of the efficient cause of inflammation; and, as a consequence, cessation of the palsy of the bloodvessels, and returning contractility of the latter.

(*b.*) Reinforced impulse from the arteries, brought about by the said contractility of the capillaries, one effect of which is a return of the phenomenon of oscillating motion in the arrested blood-column.

(*c.*) Liberation of the blood-corpuscles from a state of mutual cohesion, through endosmosis of the exuded blood-serum into the vessels charged with concentrated plasma. The blood-corpuscles lose the flattened condition and deep color which they had acquired in the stasis, swell out, and become spherical and at the same time paler.

(*d.*) The uninterrupted circulation in the capillaries surrounding an inflammation, plays its part likewise, portions of the stagnant blood-column being (according to Emmert) forcibly separated by the laving current, and a passage forced here and there through an entire capillary range.

Obstacles sometimes present themselves to the resolution of inflammation; and even when the process is accomplished, certain residua are left behind:

(1.) The blood-corpuscles are so firmly wedged in the dilated capillaries, as to resist both the contraction of the latter and the increased impulse from the arteries. This occasions a protracted stasis of a mechanical nature.

(2.) Even after removal of the stasis, a certain degree of palsy and dilatation of the capillaries may remain entailed. The part previously inflamed continues in a state of hyperæmia, and prone to relapse into inflammation. This tendency increases greatly upon repetition. The

resolution of local inflammations dependent on external causes, is common only where these causes are slight.

Inflammations dependent upon internal dyscrasial influences rarely terminate thus. An essential condition of their so doing is the extinction of the dyscrasis. If without this the inflammation take such an issue, it will localize itself in other organs standing in the relation of sympathy with that originally affected.

Even this favorable consummation of the inflammatory act is not a matter of indifference for the organism, seeing that plasma, altered by the previous stasis, is copiously received back again into the blood. The consequences are obvious, and directly commensurate with the extent of the inflammation.

2. *Reliquefaction and resorption of the inflammatory products.*—This issue of inflammation is contingent upon previous exudation, and consists in resorption of the products. It takes place with greater or less facility, according to the measure of the exudation, and to its degree of solidification. It succeeds either completely or incompletely, and hereupon it depends, whether in the sequel the diseased organ recovers its normal condition altogether, or only partially and imperfectly.

Fluid exudates are naturally susceptible of resorption.

Solid exudates become adapted for resorption by preliminary solution,—corrosion through blood-serum,—or else by disintegration, with various changes in their chemical composition.

Elementary bodies must be previously dissolved, in order to become adapted for resorption, which process takes place both through the bloodvessels and the lymphatics.

The consequences of resorption differ with the primitive quality of the exudate, with the mode of its preliminary solution and of its chemical transformation, and with the quantity reabsorbed. Finally they differ accordingly as the resorption takes place chiefly through the lymphatics or directly into the sanguineous current.

In this issue of inflammation is comprehended *wasting of the textures through inflammation.* It consists in the elements of the textures being themselves liable to become reabsorbed along with the products of inflammation. This is owing to the textural elements, within the range of inflammation, becoming functionally disabled by mechanical pressure, to the impediment to their nutrition offered by the effusion; the result being the dissolution and resorption of those elements. This termination is especially frequent in delicate, vulnerable textures, in very copious effusion, and where the latter, being solidified, is susceptible only of very tardy resorption. In this manner is the substance of the brain, of muscle, of kidney, and the like, destroyed within the range of inflammation, its place becoming occupied by a cavity, or by multilocular cavities bounded by scar texture. Where these are small and numerous, they beget a loosening, a rarefaction of the textures, as, for example, in the condition termed cell-infiltration in the brain. In hollow structures, for example the Graafian vesicles, the contents, altered by the exudate and its metamorphoses, are absorbed, and the organ becomes extinct.

3. *Abiding of the inflammatory product.*—The products of inflammation are retained bodily, or after imperfect resorption, partially, in their

original, or it may be in an altered shape and constitution. Conformably with what has been stated, the exudates continue—

1. In their original crude state, as entirely amorphous masses, or more commonly in a condition bordering upon this, of incipient textural development, in the shape of molecular granules, of nucleus and cell-formation, or in the case of consolidated blastemata, in the laminated and fibrous structure engendered by the coagulative process itself.

2. Or they break up sooner or later, and abide in a state of final cretaceous or fatty conversion.

3. Solid exudates waste away, and condense and shrivel into a horny substance.

4. They become organized, attaining thus to various grades of development. In fluid exudates this development follows the laws of the cell theory, whilst, in the organizable, solid, fibrin-exudates it consists in immediate fibrillation through dissilience.

The exudates enter, bodily, into a uniform textural change. This change may, however, conformably with the frequent primitive impurity of blastemata, be in many cases unequable. One portion of the exudate may attain a higher gradation and represent permanent textures, whilst another may be arrested at an embryonic stage, and there liquefy and become reabsorbed, or, like pus and ichor, qualify itself essentially for excretion.

A textural conversion of exudates may be of a nature very similar to, if not identical with, the normal structure, in its anatomical, chemical, and functional relations. Or, again, it may involve one or more heterologous formations, pus and cancer, for instance.

It is more particularly areolar tissue, and the various fibroid textures tending to the final composition of the latter; cartilage in the process of ossification; and bone with its penetrating vessels, that are here referred to as organized products of inflammation.

These often serve to compensate for lost parts, as also for the filling up of vacant spaces wrought by the retraction of normal textures after injuries, regeneration in muscles for instance.

The regeneration is complete; or it is imperfect, being accomplished by means of a texture not homologous with the lost one,—as in the cicatrix. This, again, may be permanent, or it may be provisional only, and about to disappear after becoming endowed with textural elements identical with the normal ones,—the nerve-cicatrix, for example.

Where permanent exudates do not serve for compensation, they occasion an increase of mass in the diseased organ. They are then—

(a.) Uniformly interposed between the elements of the normal texture. This determines an inflammatory hypertrophy, which, perhaps, occurs in a genuine form only in areolar tissue and in bone.

(b.) Or they form in a larger circumscribed mass, distinct from the normal texture,—a tumor. These local collections of cicatrix texture occur in muscle (the heart) and in all parenchymata. Upon serous membranes, they constitute the various organized pseudo-membranes and membranaceous adhesions. We have here further to remark:

1. In bulky, and particularly in solid, wasting exudates, or such as are in progress of transformation into dense, shrivelling (fibroid) struc-

tures, any textural elements which they have embraced become atrophied by pressure and tension, hindrance to their nutrition, and the suppression of their function. Even contiguous textures waste away, owing to arrested or impaired function—for instance, the muscular apparatus of respiration over thick, resistent, shrivelling, pleuritic effusion.

2. In hollow organs the abiding of exudates not rarely occasions a hypertrophic development to cystiform dilatations, with transformation of the texture of their walls and contents. As examples, we may refer to the degradation of glandular acini, and of the follicles into cysts. (See Cyst.)

3. Solid exudates determine, through increased consistency and density of the textures previously inflamed, the issue of the inflammation in so termed *induration*. On the other hand, the abiding of soft liquid exudates results in relaxation, softening, lacerability of the textures.

4. Exudates are often found to linger under several combined forms, with which, moreover, both resorption and suppuration may have concurred.

4. *Ulceration, Ichorous destruction.*—It consists in a wasting of the textures from the corrosive quality of the exudate. Herein ulcerous consumption of the textures differs from the loss of substance which inflamed textures undergo, within the best conditioned exudates, through necrosis and absorption.

To be productive of such a wasting process, the exudate must needs be fluid, whether originally so, or liquefied out of a solid blastema. Its corrosive influence upon the textures is sufficient to confirm its character as genuine ichor.

The mode in which textures in contact with ichor become destroyed, varies with the principle upon which its corrosive nature depends. The exulceration takes sometimes an acute, sometimes a chronic course. Large textural masses are not rarely destroyed within a short space of time. The destruction is marked, now by superficial extension, now by a burrowing propensity. In the former instance, it depends frequently upon a special relation of the inflammatory process to superficial textural expansion. In the other case, some heterologous formation, reproduced again and again, at the base of the ulcer, upholds the inflammation, and with it the ichorous discharge.

In the chronic form, the ulcer, like the pus-membrane, simulates, in the production of flesh-granules, a natural process of secretion.

All textures are not equally prone to ulcerative destruction. Under like circumstances, tender, young, budding growths are the most readily destroyed.

GANGRENE, NECROSIS.

Under *gangrene, necrosis*, is understood the death of an organ or part, as manifested by the more or less rapid breaking down and chemical decomposition of its texture. Gangrene may affect both soft and solid structures, the bones, for instance, or even fluids, as in necrosis or sepsis of the blood. The breaking down of solid structures, is generally a slow process, whilst in soft, juicy textures, and in fluids, it is rapidly con-

summated. Like normal textures, new formations of every kind,—tumors, exudates, pus,—are liable to become necrosed. Fluids degenerate through necrosis to *gangrenous ichor*, the most infectious and destructive of its tribe.

A general characteristic of gangrene is not easily given, so manifold are its forms, and so various its causes. Soft parenchymata commonly break down to a diffluent pulp, marked by a high degree of discoloration and of fetor. Exceptions are, however, numerously afforded in gangrene of the bones, mummifying, white gangrene.

Gangrene has the import sometimes of a local, sometimes of a symptom of general disease. The conditions necessary to the former case are nearly reducible to arrested afflux of blood, that is, stasis. It may begin by attacking fluid parts, and especially the blood, and extend from these to solid structures, or it may affect them all at once.

Gangrene is developed—

1. Out of *absolute blood-stasis*, which may occur under various circumstances :

(*a.*) Every hyperæmia in organs, or sections of organs, paralyzed or enfeebled, or obnoxious to debilitating influences, may degenerate into absolute stasis. This applies particularly to asthenic, hypostatic hyperæmia in torpid peripherous organs, vegetating, so to say, imperfectly under the embarrassment of continued pressure.

(*b.*) Mechanical hyperæmia frequently becomes absolute stasis, as observed in incarcerated, strangulated organs, and as a consequence of extensive plugging of the returning vessels in the lower extremities.

(*c.*) Every inflammatory stasis may degenerate into absolute stasis, more particularly those hypostatic and asthenic inflammations which occur in organs already diseased, paralyzed, or depressed by violent external influences, such as concussion, contusion, cold, or heat. An inflammation consequent upon influences directly or indirectly debilitating, may acquire, during its progress, a tendency to absolute stasis.

In *absolute* stasis, the blood undergoes gangrenous decomposition. Hence the blood is the portion originally necrosed and dissolved. It exudes in a state of gangrenous decomposition, and in the form of ichor, through the walls of bloodvessels, engendering the same gangrenous decomposition both in these and in the surrounding textures. This event gives rise to the most ordinary and most developed form of moist gangrene, in which the textures are, through the medium of the blood, broken down to a dark-colored, friable and lacerable, diffluent, and highly fetid pulp. The dark discoloration, however, of gangrene thus developed, is subject to various modifications due to certain elementary products, which the inflammatory stasis has generated both within the bloodvessels and without.

The progress of this gangrene is more or less acute, the gangrenous dissolution of tissues, already referred to under the term putrescence, being particularly marked by the rapidity of its course.

2. *Gangrene is determined by failure in the supply of blood :*

(*a.*) In impermeability of large arteries,—high degree of coarctation, and complete obstruction—consequent upon arteritis and ossification.

Here the gangrene, for the most part, takes the form of comparatively dry, black, mummifying gangrene.

(*b.*) As the result of the immediate compression and tension of a part; for instance, in incarcerated hernia.

(*c.*) As a consequence of the local destruction of bloodvessels, the denudation of parts of attaching and blood-supplying textures,—bones, for example, of their external and internal periosteum; the common integuments, of their supporting areolar tissue; the peritoneum, of its subjacent layers; isolation of the pulmonary pleura over cavities of the lung.

The gangrene appears as a white or yellowish-white slough.

To this category belongs the necrosis of smaller textural parts, loosened mechanically by exudation or by ulceration.

(*d.*) Extensive impermeability of the capillaries and minute vessels when plugged with coagula, or compressed by surrounding exudates.

In the last-mentioned case, the gangrene is dependent upon inflammation. To this kind of gangrene, textures poor in bloodvessels, such as compact bones, callosities, &c., are especially obnoxious. The color of the necrosed textures differs with the different nature of the coagulation, and of its exudate. Answering to the croupous character of bulky exudates, the textures involved in the necrosis commonly assume a yellow or yellowish-green hue.

3. *The gangrene is the expression, the localization of an anomaly in the blood-crase,* either directly ingrafted by infection (contagion), or developed out of other crases; a putrid decomposition of the circulating fluid. Blood so poisoned, especially if brought into stasis or into coagulation, possesses, in common with the exudates thrown out by it, an inherent tendency to gangrenous dissolution.

It has been already stated, that several varieties of gangrene are recognized:

1. *Gangrene* developed out of an internal cause is distinguished, by the designation of *primary gangrene,* from that arising out of a predominant external cause.

2. *Hot, acute, inflammatory gangrene.—True gangrene.* In what wise inflammation leads to gangrene, is sufficiently clear from the foregoing.

(*a.*) The inflammatory stasis, owing to its very intensity, to pre-existent debility of the diseased textures, or, lastly, to weakening influences exercised during its progress, degenerates into absolute stasis.

(*b.*) It occasions gangrene by the crushing effect of its products upon the capillaries, or by the mechanical or ulcerous isolation of textural parts.

In the first case, the necrosis affects more immediately the blood held in stasis; in the second, the textures. In the first case the gangrene is, as it were, an immediate issue of the inflammation, the opposite to resolution; in the second it is a remote consequence thereof.

In this way, gangrene may arise in tissues laboring under the sequelæ of inflammation, without being itself an issue of the latter.

3. *Cold gangrene, sphacelus,* is so-called, as being unconnected with inflammation.

4. *Moist gangrene* comprises the breaking down of fluid substances to gangrenous ichor, and of fibrin textures to a variously discolored, diffluent pulp, marked by its evolution of fetid gases. It is the gangrene developed out of absolute blood-stasis;—therefore, again, inflammatory gangrene. It may be compared to the decomposition of animal matter under the co-operating influence of water.

5. *Dry gangrene* is a consequence of deficient blood supply. It manifests itself in the perishing of the implicated textures, with shrivelling or withering thereof, to an incipiently tough, but eventually sloughing mass. Often, and particularly in the *gangrene* termed *senile*, which affects the extremities, especially the inferior, owing to impermeability of their arteries, the gangrenous textures blacken ; wherefore this species has been designated as *mummifying gangrene.* As such, it is comparable to the decaying of organic matter, that is, to decomposition with absence or insufficiency of moisture, and with the disengaging of pure carbon. Dry gangrene is frequently called *gangrenous slough.*

6. *Black gangrene, gangrenous slough.*

7. *White gangrene, gangrenous slough*, occurs, for the most part, as a consequence of pressure in incarceration ; of the denuding of membranous expansions of their subjacent textures, for example, as peritoneal sloughing at the base of intestinal ulcers. Again, it is generated by the necrosis or death of textures replete with fibrino-croupous exudates, or of such coagulate exudations themselves. This refers more particularly to the common integuments, the mucous membranes, fibrous and areolar tissue expansions upon wounded and ulcerated surfaces. To this head belongs *hospital gangrene.*

Of these different species of gangrene, several are often concurrently present. Beneath the common integument, often transformed into a swarthy parched rind, in *senile gangrene*, we frequently meet with patches in which the textures are reduced to a humid stinking pulp.

Just as gangrene of the solids, *gangrenous slough*, varies, so in like manner does *gangrenous ichor*, as necrosed blood or exudate vary, according to the crasis or constitution under which either has become attacked with gangrene. Thus the necrosis of typhous blood differs from that of pus-blood, or of fibrino-croupous blood.

Like normal textures,—diseased textures and new growths, fibroid, cancerous formations, for example, may become a prey to gangrene. Neither to ulceration nor to gangrene are all textures alike obnoxious. Bony, elastic, fibrous textures resist gangrene more ably than muscle, areolar tissue, or mucous membranes. Lax embryonic textures, as, for instance, certain kinds of cancer, are especially prone to gangrenous destruction.

The constituent elements of gangrenous texture-masses are, more or less well-preserved textural débris, larger or smaller black-contoured molecules down to a pulverulent granule mass, black and brown pigment granules, fat-drops and crystals, saline crystals.

Contact, reciprocity of action, with the atmosphere, is by no means indispensable to the generation of gangrene. It affects equally with the external parts, organs never in contact with the air, as the liver and the spleen.

A very important phenomenon involving a curative act, is the circumscription of gangrene by an inflammatory process of ulceration,—isolation of the gangrenous part through its own secretion. The ultimate healing is brought about by the same inflammatory process changing to one of pus-production, and of regeneration.

CHARACTERISTIC OF INFLAMMATORY TEXTURES AND DIAGNOSIS OF INFLAMMATION IN THE DEAD SUBJECT.

In the period preceding the real exudation, an organ is, within the range of the inflammation, reddened, injected; that is, more than ordinarily vascular, swollen, and at the same time relaxed, softer, lacerable. The redness must be that of injection, and is to be carefully discriminated from redness of imbibition. The swelling and relaxation result from infiltration of the texture with exuded blood-serum.

Generally speaking, the swelling is accompanied by increase of volume; to this, however, spongy textures, and in particular the lungs, are exceptions. In protracted, and especially in hypostatic stases, the swelling of the texture frequently occasions closure of the pulmonary cells, and renders them inaccessible to atmospherical air. The volume of the inflamed part falls short of the normal.

As, however, mere hyperæmia occasions similar appearances, although in a minor degree, the question arises: " What are the criteria in the dead body which justify us in pronouncing the inflammatory stasis to be attained ?"

The only true criterion in the dead subject is afforded in the changes suffered by the blood in the capillaries of the implicated texture, during the inflammatory stasis—changes cognizable, in part, by the naked eye.

Effusion having taken place, its product, *exudate*, affords incontestable evidence of inflammation, wherever its character is such as we know by experience to attach solely to the fruits of this process; examples are pus or croupous fibrin. The redness and injection cannot here commonly apply, having, for the most part, given way to the effusion. Even the swollen condition of the texture may in a great measure have subsided during a mortal collapse. The relaxation of the tissues still lasts, taking, however, in solid exudates, the form rather of fragility, as, for instance, in the hepatized lung.

Where, however, the exudate is a blastema, well known to exude both with and without inflammation, the question arises: what circumstances warrant us in assuming such blastema to be the product of inflammation? Such circumstances are—

(*a*.) Rapid and copious production of the blastema;

(*b*.) The concurrence of exudates, known to be exclusively generated by inflammatory stasis;

(*c*.) Analogy of textural destruction with that due to inflammatory exudation, more especially if coupled with analogy between the exudate and a product pertaining exclusively to inflammation, as between tuberculous infiltration of the lung and hepatization, and again between the hepatizing tubercle and the fibrino-croupous exudate.

Lastly, it is important to decide whether the appearances, in the

neighborhood of a product, are residue of the inflammation that called it forth, or the rudiments of a new consecutive inflammation caused by the said product. The discrimination, so far as it is feasible at all, may be deduced from what has been already stated.

COROLLARY.

(1.) The inflammatory process is especially fitted for displaying the primitive differences of blastemata, those inherent properties contingent upon internal (endogenous) formative processes.

(2.) It is equally adapted to demonstrate the commonly mixed character of blastemata, and the consequent variety of elementary constituents which enter into the composition of a new growth.

(3.) It is the last of a series of exudative processes, beginning with the exudation of plasma in the act of nutrition, which in degree and kind probably all bear more or less resemblance to it.

(4.) Scarcely a new growth exists, the blastema of which may not be produced by inflammation. On the other hand, inflammation yields products proper to itself alone. It is to be observed, at the same time, that where a corresponding crasis prevails, the stasis requisite for the formation of the product is in the inverse ratio of the intensity of the dyscrasial process in the general circulation. Such exudatory acts, together with other processes to be discussed in the next chapter, are wont, owing to the rapidity of the effusion and to the slightness of the accompanying stasis, to be characterized as *deposits*.

(5.) The homœoplastic textures produced by inflammation are areolar tissue, a fibre analogous to that of organic muscle, transition-cartilage, bone, blood, and bloodvessels. The regeneration of nerve-fibrils, after wounds attended with loss of substance, does not take place as a new formation out of the exudate, but as growth of the nerve from its cut ends into the exudate, constituting a provisional cicatrix.

(6.) Inflammation possesses now a local, now a general import. In the latter case it is the localization of an anomalous crasis which stands to it in the relation of cause.

(7.) Inflammation may concur with a crasis either accidentally or as its symptom. This has been to a certain extent recognized by the acceptation of an arthritic, a scrofulous, a scorbutic inflammation. Custom has stamped inflammation with fibrinous exudates and a kindred phlogistic crasis, as genuine inflammation.

(8.) How ought we to estimate that view which designates inflammation as augmented, vital, and formative energy—as increased vascular activity,—as reaction?

Neither in the sense of a neuro-pathological nor of an attraction-theory, can there be any question of increased vascular activity. A vigorous formative power no doubt *is* at work in the inflammatory process; still, in the formative efforts, the qualitative anomaly is predominant. Even adhesive inflammation, in which one might be most of all disposed to look for an augmentation of the normal process of nutrition, produces but few, and these simple, textures.

To define inflammation as a reaction of the organism against a mor-

bific influence, is simply begging the question. Inflammation is a morbid process, unconscious of its scope or object, evoked by a causal impulse, and sustained or repeated so long as this impulse remains in activity. Only in this general sense of cause and effect is the definition of reaction admissible.

DEPOSITS.—METASTASIS (SO-CALLED).

Together with those inflammations leading rapidly and insensibly to exudation, the term deposit (metastasis, capillary phlebitis, lobular process) applies aptly to certain processes which, considering the rapidity with which they become established, are, in many respects, of a very remarkable kind. These processes are founded in a sickening of the fibrin, with a tendency to coagulation. They consist in the blood, through spontaneous impairment or through infection [that is, the reception of various substances], acquiring the tendency to coagulate, and actually coagulating within the circulating system, under a more or less marked separation of fibrin.

This happens either in one of the larger vessels, or, what is far more common, in the capillaries of an organ. In the former case it is, consistently with the frequent reception of deleterious substances into the venous blood, the larger veins, usually in the vicinity of the point of infection. With respect to the capillary system, no point of it is exempt, although the more vascular organs, those in which the blood undergoes important changes, are most obnoxious to the affection ; for example, the lungs, the spleen, the kidneys.

In large vessels it is not difficult to interpret the appearances. In the absence of all evidence of local inflammation of the vessel's coats, the calibre is plugged with lengthy, cylindrical, or smaller, clod-like— in vascular trunks membranaceous,—in arteries, especially where the inner surface is rough or gibbous, adherent—coagula. The probability of these coagula resulting from inflammation of the vessels, increases in proportion as the fibrin which constitutes them is pure, and as its yellow color and general attributes approximate to those of croupous fibrin. The various coagula found within the heart's cavities—the valvular, the globular, vegetations, belong to the same class.

The cylindrical and clod-like coagula are often equably reddened by incorporated blood-corpuscles. At other times they contain layers of a lighter color than the rest, and which therefore have incorporated fewer blood-corpuscles. Some layers are even marked by a total absence of redness, and obviously consist of pure effused fibrin.

To trace the process in the capillaries is not an easy matter. It is conceivable that besides what happens within the vessels, and as a consequence thereof, exudation of blood-serum, and even of a portion of plasma with blood-pigment takes place. By means of this exudation, the vessels become obscured and uncognizable. Still, the simple fact of the process occurring in large vessels should remove all doubt as to its existence in the capillaries.

In the capillaries of an organ, the process originally manifests itself

by a dark red, sometimes reddish-white, spangled or striated, circumscribed, impacted substance, of a dense, fragile consistence.

These impactions possess the peculiarity of being seated for the most part in the periphery of organs,—the lungs, spleen, kidneys, for instance. They represent roundish tubercula or else wedges with their broad base directed towards the periphery and impinging upon the sheath,—their points towards the interior of the organ. They are always present in considerable number, and commonly associated with exudative processes. Their size is mostly inconsiderable, commonly ranging from that of a pea to that of a walnut. In organs of a lobulated structure, they are sometimes called *lobular metastases*.

The ulterior changes are various, corresponding with the nature of the coagulation, and therefore of the blood-(fibrin) disease.

They sometimes shrivel and condense, with obliteration of the canals of the vessels, and of the implicated textures, into a fibroid state,—still further shrivelling callus, towards which the neighboring parts are retracted in a scar-like manner, and which often involves a residue of blood-pigment in the shape of rusty brown, or rusty yellow coloration.

It is probable that, like the thrombus, they are often progressively redissolved, and again taken up into the circulation without detriment to the textures.

Sometimes they break down to a puriform, ichorous fluid, to gangrenous ichor, with diffluence and necrosis of the involved textures, resulting in a pus or ichor depôt, or a gangrenous slough. In greater coagula, this metamorphosis very commonly emanates from the central layer. Capillary impactions, at the periphery of an organ, often assume the aspect of a superficial boil.

The coagula often undergo fatty conversion.

Occasionally, the coagula have the character of tubercle or of cancer, especially of medullary cancer ; and it is probably through this process that the often very rapid development of cancer is produced in brutes, by the injection of cancer-blastema.

In conclusion, many an important crase-exhausting metastasis may have its source in the same processes, involving a great extent of capillaries, and issuing in gangrene. Such metastases occur as the sequel to typhus, and to the exanthemata.

With respect to the organs whose capillaries are especially obnoxious to these coagula resulting from infection of the blood, as it is in venous blood that the mischief commonly originates, and as the lungs are the first organs reached by the poison, their capillary tissue is commonly the first to suffer. In its arterial passage, the contaminated blood next attacks the capillaries of the spleen, and of the kidneys. Lastly, in a higher grade of the dyscrasis, all the other textures, mucous membranes, common integuments, and subcutaneous areolar tissue, even bone, become implicated in their turn.

In infection of the portal blood, the liver is the first assailed, otherwise it is only so in common with other organs, and by no means more frequently than the spleen and kidneys.

That, compared with the greater vessels, and especially the arteries, these processes occur pre-eminently in the capillary system, seems due

to the slackening of the circulation in the capillaries, which in itself promotes coagulation, and at the same time, leaves the deleterious matter longer in contact with the blood. In certain organs, moreover, their formation appears, as before stated, to be favored by the revulsion which the blood undergoes in them during the acts of nutrition and secretion.

Sensitive as is arterial blood in respect to infection, as in arteries, for instance, coagula are comparatively rare in arteries of the aortal system, with the exception of those at the point of arterial inflammation. This is probably owing to the great rapidity of the arterial current. In the arterial section of the heart, however, they are readily engendered by diminished action of their organ, far more readily than out of the venous blood in its right chambers.

The relation which these processes bear to inflammation and its terminations, is obvious. Determined by high grades of dyscrasial impairment of the blood, they represent product formation, endogenous exudation, within the vascular system.

B. ORGANIZED NEW GROWTHS SPECIALLY CONSIDERED.

Having now treated of organized new growths in general, of their blastemata, and of the several processes through which these are engendered, we come to the special consideration of new growths.

What order ought we to observe in this discussion?

(1.) An arrangement based upon morphological relations is inadmissible:

(*a.*) The elements being in themselves far too uniform, in their secondary arrangement too multiform, and generally too little distinctive of the nature of the new growth.

(*b.*) The elements being mutable, what is one day in the embryonic state, is found further advanced on the morrow; whilst various grades of development are found to co-exist in juxtaposition.

(2.) Similar objections obtain against a division of new growths according to their main organico-chemical constituents; these being not alone convertible substances, but also liable to enter into various combinations in the same growth.

(3.) With respect to the distinction of new growths into homœoplasiæ and heteroplasiæ, we have already once expressed an opinion. A systematic arrangement upon such a basis, irrespectively of its preternatural rigor, is open to the objection, that it breaks up new growths into two great series, in the first of which no doubt many points offer in the analogies for further subdivision. In the second, however, we are either driven to a ground of distinction alien to the system, er else compelled, in opposition to the principle itself, still to have recourse to comparison with normal textures.

(4.) How little the benign or malignant nature of new growths in general, affords a basis for a genuine classification is self-evident.

In order to avoid the difficulties alluded to, we shall here endeavor to observe a middle path, and treat of new growths in a series beginning

with those which bear the evident tokens of benignancy, and whose ulterior development is for the most part one of progress into fibre; passing next to those differently constituted in these particulars.

1. AREOLAR TISSUE FORMATIONS.

The new growth of areolar tissue is a very widely extended one. Newly generated areolar tissue occurs both pure, and also as a constituent of other new formations, for which it often furnishes a sort of stroma or framework. Generally speaking, it is not alone as to the constitution of, but also as to the arrangement of its fibres in new growths, a repetition of the normal. In the said stroma or framework, however, of many cancerous growths, there occur fibrils of extraordinary delicacy.

Its development often follows the laws of the cell-theory, that is nucleus, cell-formation, spindle-shaped, caudate cell, fibre with attached nucleus, fusion of several cells to a varicose fibre, breaking up of the fibres into fibrils. Areolar tissue is, however, much more frequently and more extensively shaped out of the immediate dissilience of a solid blastema into areolar tissue fibre, or else mediately, through a preliminary splitting into other coarser fibres.

Newly formed areolar tissue is often found blended in different proportions with elastic fibres, nucleus fibres in various degrees of development, from the oblong, caudate nucleus, the rodlike fibre stem, to the complete fibre.

Apart from its occurrence as mere increment of that previously existing (hypertrophy)—areolar tissue of new formation is met with:

(*a.*) In the shape of threaden, cord-like growths, of flocculent and velvety accumulations, of either free, bridle-like, or agglutinated and firmly seated layers and membranes upon serous tunics, even of entirely new-formed, movable, serous sacs. In many such cases it is, as membrane, invested with an epithelium on its inner free surface.

It determines those frequent adhesions of organs contained within serous sacs, both with each other, and with the parietes of the latter.

(*b.*) It constitutes the entire parietes of perfectly new anomalous serous and synovial sacs, or else it partially enters into their composition for the most part as the external layer, in connection with fibroid textures. To this class belong the anomalous bursæ mucosæ, the articular capsules of preternatural joints, the capsules investing foreign bodies or extravasate (the envelopes of apoplectic cysts), every variety of cyst-formation.

(*c.*) It forms the external vascular sheath of many new growths, both benign and malignant, fibroid and cancerous, or their stroma, for example in lipoma.

(*d.*) Of tumors it forms the condyloma, the hypertrophous lupus, the pedunculated wart. Commonly conjoined with fatty texture, it composes those appendices of the skin denominated mollusca, a species of so-called fibrous tumors and of fibrous polypi.

(*e.*) Finally it presents in various grades of development an essential constituent of scar-texture.

The growths composed of it contain a large proportion of gluten.

The blastema for the new growth of areolar tissue is sometimes fluid, and the development takes place according to the call-theory laws, but more often, especially when copious, it is solid and fibrinous. It exudes during the progress of protracted hyperæmiæ, and in the last-mentioned form, more especially, as a consequence of inflammatory stasis. Both modes of development concur with suppuration. Moreover, extravasated fibrin, as also endogenous fibrin-coagula within the vessels furnish, under certain conditions of the fibrin, the blastema for the new formation of areolar tissue.

The chemical changes which take place during this process of development are very remarkable. They consist in numerous modifications of quantitative type, as also in qualitative differences of reaction observable in the gluten-extracts.

The time requisite for the new growth of areolar tissue varies from one to several weeks. The process of dissilience into areolar tissue fibres and fibrils, more especially in the case of copious solid blastemata, is often a very slow one.

2. FIBROID TEXTURE.

In an extended sense, the collective term *fibroid texture* may be made to comprise all fibrous tissues, the development of which has been already delineated, and the occurrence of which as a more or less essential component of various new growths, it becomes our business to discuss.

Nowhere is the insufficiency of a mere anatomical principle more felt than here—a principle which would needs occasion us to class side by side, the most heterogeneous new growths, for example, fibro-carcinoma and the perfectly benign fibroid tumor.

In a more restricted sense we comprehend under fibroid textures those which consist either of the elements about to be described or of a blastema manifestly furnishing their groundwork, which yield gluten, are benign, and in external appearance, resemble the fibrous texture. In this sense the fibroid texture enters into the composition of various heterologous growths, constituting in them the benign ingredient which cornifies or ossifies by a spontaneous metamorphosis. Or it is woven in with normal textures, or, lastly, it represents sharply defined, often very voluminous masses, in a word, tumors. The fibroid (fibrous) tumor, besides the aforesaid attributes, is distinguished as consisting entirely or almost entirely of the elements of the fibroid texture. It is firm and elastic, or else tough, presenting a mere local evil, independent of dyscrasial taint and originating in local deposition, a fact denoted more especially by its selection of a particular organ, even where it occurs in great numbers.

The groundwork of fibroid new growths are firm, probably always fibrinous blastemata. Exudation or extravasation-fibrin, or fibrinous coagula within bloodvessels often constitute these.

Irrespectively of perfected areolar-tissue fibre, as the main, if not the sole constituent of growths called, from their dense texture and their resistance, *fibro-areolar textures, or fibrous tumors,*—the following elements are in particular deserving of notice.

(*a.*) Flat, smooth fibres resembling the organic muscle-fibre, here and there breaking up into fibrils, and thus engaged in the transition to areolar tissue. The nuclei present deport themselves as upon muscle-fibre, and the textures proper to it.

(*b.*) Flat, broad, band-like or roundish, shapeless, solid fibres, with rough, denticulatad or felt-like outline, which are held together by a solid blastema, and here and there break up into areolar-tissue fibrils. Nuclei often seem to stand in no developmental relation to them, and are frequently altogether absent.

(*c.*) The *fibroid blastema*, an embryonic, stratiform, fibro-laminated, solid, transparent, or opaque (brawn-like), formation, interspersed, or not interspersed, with elementary granules, nuclei, and cells.

The delicate fibre network of solidified fibrin is occasionally preserved in it.

The above-named fibre-elements originate directly out of their blastema through dissilience. The formations consisting of them occur both as superficial expansions, and interwoven in normal textures, as knotted, spherical, or irregularly ramified masses (callosities); and, lastly, as independent tumors.

These various formations demand a special inquiry. Besides the true fibrous tumors, of which we shall have to treat specially, we have here to mention:

Inflammatory products, fibroid exudates, representing within parenchymata, irregularly knotted, ramified masses, or, upon serous membranes, superficial expansions. The latter constitute pseudo-membranous calli of various dimensions, but frequently engrossing the entire superficies of a serous sac. They are of various thickness, which is considerable upon the parietal layer of serous sacs, of density and resistance equal to those of fibro-cartilage; in color white, or as a consequence of hemorrhagic exudation, varied with black, slate-gray, rust-brown, or a yeast-yellow. They often determine complete conglutination of the parietal and visceral layers of serous sacs. Or, again, they are smooth and even, or fenestrated, granular, stellate plates; or finally, they consist of granulations, scarcely surpassing in size, poppy, millet, and hemp seeds, for example, upon the cerebral arachnoid membrane, upon the hepatic and splenic peritoneum, &c.

The fibroid thickening of serous membranes in the shape of smooth or granulated plates, of granulations, to which last are to be reckoned the Pacchionian glands.

Most free bodies found within serous and synovial sacs.

Cicatrix-substance generally, as also the so-termed keloid of Alibert—arrested (ligamentous) callus.

The internal layers of numerous cysts and of receptacles and excretory ducts degenerated into dropsical capsules (dropsy of the gall-bladder; of the Fallopian tube).

Callosed extravasate-fibrin, in shape of central or peripheral (encysting) membrane separated out of extravasates.

Callosed coagulations of fibrin within the vascular system, the different so-called vegetations in the heart's cavities, the cylindrical coagula within veins and arteries.

The superimposed layers upon the internal surface of arteries; the soft groundwork of so-called phlebolites; &c.

These fibroid formations not unfrequently inclose within a nidus, a curd-like or puriform fibrin—the product of inflammation.

The secondary arrangement of the elements above treated of, is reducible to the following types:

1. *Parallel fibrillation*, superficial expansion predominating.

2. *Fibre-felt*, a multicrucial fibrillation, a section of which, in whatever direction made, always displays fibre-shoots and bundles, intersecting each other at various angles.

3. *Areolar disposition*, of very rare occurrence, at least in pure fibroid formations springing from a solid blastema.

The two following structures, determined by a primitive disposition of the coagulating process, are also rare.

4. A network of fibroid bridles (fibre bundles), of from $\frac{1}{80}$th to $\frac{1}{13}$th of a millimetre broad, crossing each other at various angles, and having their gaps filled up with embryonic elements—for the most part nucleus-formations in an amorphous blastema.

5. A web of similar fibroid cords emanating from a central mass, and anastomosing with other webs derived from other centres.

These structures seem to occur more especially in fibroid formations springing from extravasate-fibrin, as occasionally met with in stratified deposits upon the inner surface of arteries.

6. Finally, a kindred form is brought about by resorption, as a gap or fenestrate-formation, the gaps being round or oval. It occurs in fibroid tumors, in the fibroid thrombus, in the vegetations within the heart's cavities, but especially in the accumulated layers upon the internal arterial membrane. It is analogous with the fenestrated structure of the striated coat of bloodvessels.

All these formations have, even for the naked eye, the aspect of a porous, cancelled structure; the gaps are, however, widely different from the alveoli of areolar textures.

(*d.*) A further element of fibroid formations is a cylindrical, in its parietes structureless, striated fibre, with a simple, but sometimes a double contour, inclosing granules, nuclei and cells. We have encountered this sort of fibre in consolidated hemorrhagic effusion; in old vegetations about the heart's valves; in fibred cartilaginous investments of the joints; in the villous new growths upon synovial capsules.

(*e.*) Another element, again, of fibroid growths is the nucleus and the nucleus-fibre in the intermediate stages, as caudate nucleus, and varicose nucleus-fibre. These elements are to a certain extent found in conjunction with those hitherto discussed, with the concurrence, however, of an amorphous bond-substance—a membranous basement. They extensively furnish forth fibroid growths, but are not very common.

(*f.*) To conclude, not a few fibroid new growths consist in a fibre-felt, developed within a basement either solid or adapted for membranous expansion. It resembles that in the intercellular substance of reticular cartilage. This texture is often met with in the fibroid deposits within arteries.

A metamorphosis common to fibroid textures, is a so-called ossifica-

tion, and a cornification. The former is observed more especially in fibroid tumors, in fibroid exudates, upon serous membranes, in fibrinous coagula within the calibre of bloodvessels, in the deposits upon the inner membrane of arteries, and the like. The growth shrivels with obliteration of its vessels, loses its elasticity, becomes dry, of a dingy yellow color, and gradually bereft of its fibrous texture. Meanwhile a black, minute molecule, consisting of fat with the salts of lime, becomes imbedded in its substance.

Cornification is observed especially in the vegetations about the heart's valves, and in the layers accumulated within arteries. The growth becomes dry, denser, of horn-like toughness, and of dull transparency.

Vascularized fibroid growths occasionally take on inflammation, for the most part from the surrounding tissues, suppurate, and perish layer by layer. Nor is it rare for them to become loosened and cast away through the suppuration of adjoining textures.

Gluten yielding fibroid tumor, also denominated fibrous tumor, formerly designated as sarcoma, steatoma, or even as scirrhus.

They are distinguished from other fibroid tumors by their independence and circumscription, as being sheathed in a layer of vascular and areolar tissue, and thus, as it were, impacted in the texture of organs, from whence they may be fairly peeled out. They represent more or less perfectly spherical, for the most part tough, fibro-cartilage-like, resisting, distinctly fibrous, according to their degree of vascularity, whitish or reddish-white, new growths. In size they vary from that of a tumor just cognizable, up to, and beyond that of a man's head. They often coexist numerously in the same organ.

They probably occur in every organ, although they are, without doubt, frequently confounded, more especially in glands, with fibrous sarcoma, and with fibro-carcinoma. They are frequent in the submucous areolar tissue layers, more particularly in the intestine, stomach, and œsophagus, now and then in the larynx, again, in the subcutaneous areolar tissue, and very commonly in the uterus and its appendages, where their development is in every way very strongly marked. Their texture is usually made up of areolar-tissue fibre, or of the elements described at *a*, *b*, and *c*. The uterus fibroids, in particular, very often repeat the fibre of organic-muscle tissue.

With respect to the arrangement of the constituting fibres, fibre-layers and fasciculi, much variety is manifested, and this, again, is pre-eminently marked in the uterus fibroid. Thus:

(*a*.) The fibroid tumor, with *concentrical lamination of the fibre-layers*, is commonly quite spherical, very dense and tough, poor in bloodvessels, white, never attaining the magnitude of the species that follow.

(*b*.) Fibroid, with manifold *decussation of the fibres*. The fibration frequently springs from distinct centres marked by their density and whiteness. These tumors grow to a considerable magnitude, and have an irregular flattened tuberosity of surface.

The following is a variety of this fibroid:

The tumor consists of an *aggregation of dense fibroid tubercula or centres, about the size of peas or beans, united by means of a lax vascular texture*. These tumors have an uneven undulating surface, and

attain to a very considerable bulk. The loose interstitial texture sometimes becomes the seat of a serous infiltration, which, under dragging and eventual laceration of the said texture, may become exalted into dropsy within the tumor. The fibroid contains within its interior a cavity replete with serous fluid, fluctuates, and may thus present the appearances of a cyst—in the uterus, more particularly that of a hydrometra.

All these fibroids ossify.

(*c.*) There is one other variety of fibre-tumor, which, so far as its elements are concerned, ranks with the foregoing growths. In other respects, however, it differs from them, and offers a transition form to fibro-sarcoma.

The characteristic of this last form of fibrous tumors is that, varying in circumference, they are so rooted in the implicated organs as not to be removable from, without injury to, the latter; that they become lobulated in their growth, and yield little gluten; whilst, on the other hand, they contain albumen, and in their fibrillation are, at least in part, developed out of cells. Not rarely we find in them excavations lined with a smooth membrane, and filled with a sero-albuminous fluid, *alveoli, cysts.* They are further marked by considerable vascularity.

Like other kindred forms referable to the class of fibro-sarcoma, they are often rooted in the submucous, areolar, and muscular textures, in areolar tissue, in the periosteum subjacent to mucous membranes, and in the inner layers of the substance of the womb. Here, under the designation of *fibrous, sarcomatous flesh-polypi,* they grow into the muco-membranous cavities, with predominant longitudinal direction of their fibres, pushing forward the mucous membranes themselves in their advance, and representing cylindrical, spindle-like, pear-shaped, bulbous tumors, lobulated at their free extremity, and traversed, more especially in the uterus, by capacious bloodvessels (veins).

As so-called polypi, they are to be carefully distinguished from mucous, cellular, or vesicular polypi.

They do not ossify.

3. ELASTIC TISSUE AND TEXTURE OF THE ANNULO-FIBROUS MEMBRANE OF ARTERIES.

The elastic and nucleus-fibres enter, more or less, and sometimes in very considerable quantity, into the composition of the most varied new growths, although in no instance are the latter entirely composed of them.

We have, however, occasionally observed accumulations of elastic fibres, in the arrangement and form proper to the vocal chords, beneath the mucous membrane of the trachea, and close to the larynx.

With reference to the texture of the annulo-fibrous membrane, we have, in one instance, seen the muscular fibres of a hypertrophied, solidified, rigid, muscular tunic of the urinary bladder converted into dingy yellow, elastic bands, which presented a texture exactly like that of the annulo-fibrous membrane of arteries. A transformation of one texture-species, pertaining to a common genus, into another.

4. CARTILAGINOUS GROWTHS.

Wounds of cartilage are not reunited by means of cartilaginous substance, nor is this substance regenerated when destroyed. Nevertheless new growths of cartilage-texture are both frequent and voluminous. The structure of these growths or tumors was first ascertained, with the aid of the microscope, by Johannes Muller, who applied to them the term *enchondroma*. These excepted, not a single new growth, whether designated as cartilage-like, fibro-cartilage-like, or as cartilaginescence, chondroid, fibro-chondroid, has more than a seeming analogy with true cartilage texture.

Enchondroma repeats all the special physiological textures of cartilage. It occurs both as hyaline, genuine, as fibro-cartilage, and as reticular cartilage, imitating the articular investments, the laryngeal cartilages, and the septum narium on the one side, and the synchondroses of the vertebræ, the cartilages of the external ear, the epiglottis, &c., on the other.

Ordinarily, and especially in the enchondroma of soft parts, all these forms are often found in juxtaposition. The pure hyaline cartilage is, however, the least common, the intercellular substance displaying, for the most part, a fibrillation similar to that in the cartilages of the ribs.

The *enchondroma* forms spherical, or nearly spherical tumors, with an even, smooth, or else, which is more usual, a mammillated surface. Internally it either presents a continuous hyaline mass, or else, corresponding with its mammillated exterior, a lobulated structure, an aggregate of denser, hyaline knobs or spheres, either held together by a black-contoured, rough, inelastic fibre texture, resembling the intercellular fibrillation, or else imbedded in a loose texture imitating the fibre-layer of reticular cartilage.

Enchondroma chiefly occurs in bones, especially in the phalanges of the fingers and toes, in the sternum, in the ribs, more rarely in other bones, such as the long cylindrical bones, the ilium, the skull-bones. It is also met with in the mammary gland, in the parotis, in the testicle. We have ourselves seen it in the subcutaneous areolar tissue, and on several occasions in the lungs.

In magnitude, enchondroma varies from that of a tumor only just cognizable to that of a child's head, and beyond it. In bone, enchondroma exhibits two varieties, namely, enchondroma *with* and enchondroma *without* bony sheath. This osseous capsule is bone, whose texture has become distended and inflated by the enchondroma in the progress of its upward development. In this process it has, for the most part, increased in substance, so that the capsule far exceeds, in this respect, the original bone. Where the sheath ruptures at an early period, the enchondroma is devoid of bony investment. The capsular case of the enchondroma is unessential, and is common to many other heterologous growths developed out of the depth of bone, and more especially out of a medullary cavity. Many of the so-termed cases of spina ventosa of older observers, were probably of the nature of enchondroma.

Enchondroma is benign, provided it does not enter into any specific, infectious metamorphosis, and only undergoes ichorous destruction from irritation. A peculiar predisposition to its formation does, however,

exist, as shown by its occurring numerously in one individual (phalanges, ribs, &c.) It affects young persons more especially, although we have known examples of enchondromata first becoming developed at an advanced period of life. Here, however, they are usually concurrent with exostoses and bulky osteophyte forms. Echondroma generally imitates the permanent cartilages; with exceptions, however,—for it ossifies.

Not only have we seen in all enchondromata incipient ossification, but our museum contains specimens, for the most part, if not thoroughly ossified.

Ossified enchondroma is sometimes a white, extraordinarily dense, ivory-like, sometimes a yellowish-white, likewise very dense, although uncommonly brittle, bony substance, deviating in various degrees and various ways from the texture of normal bone. This difference of habit corresponds to a different process of ossification, and to a different elementary texture.

In the first place, we miss the laminated structure of true bone. The medullary canaliculi are present, the bone-corpuscles large, spherical, irregularly grouped, wanting in radiations.

In the next place, and this refers to the second form of bony substance adverted to, the process of ossification recedes still further from the normal. It resembles rather a process of involution, a wearing out of the cartilage, and, like the texture itself, it has its analogies in ossification of the larnyx, and above all, of the cartilages of the ribs. The intercellular substance of the hyaline enchondroma becomes dull, granulated, sallow, lardaceous, and fibred. The cells are centrally transformed into spherical bone-corpuscles without radii, or else the entire large cell-cavities are simultaneously, if not previously, filled with bone-earth. They are interspersed without order, the last mentioned forming comprehensive spherical or oval masses, which, with transmitted light, appear black, and have a diameter of $\frac{1}{15}$th of a millimetre. The lamellated structure is wanting. Medullary canals are wanting, or rudiments only of their structure are seen in the scattered grouping of the cartilage-cells.

Enchondroma commonly occurs in a simple form. We have, however, encountered it in the shape of little millet or hempseed-sized tubercula, interspersed through medullary carcinoma of the testicle, an occurrence allied to the frequent entering of true bone into the composition of cancers.

5. BONE-FORMATION.

Bone-formation comprises various new growths, which, in their developed stage, are readily divided into two classes, according to the analogy which their texture bears to that of normal bone. Still the line of demarcation is not sharply drawn, owing to the multiplicity of transition forms from the one to the other. The one category comprises new growths *identical, or nearly identical with,* the other, *a series of new growths less or more widely discrepant from the texture of normal bone.* On a closer scrutiny, however, this series again separates into the *osteoid,*

and into the *bony concretion*, which latter manifests itelf, more especially in fluid blastemata, as *cretefaction*.

It is, indeed, worthy of preliminary remark, that not alone solid blastemata and perfected textures, but also fluid blastemata, afford the basis of, and are liable to, so-called ossification.

The process which involves the conversion of the substances here alluded to into bone, is commonly termed *ossification*, and thus brought into kindred relation with the bony conversion of cartilage.

We have here to observe:

1. In the first place, amongst the pathological growths with which we are here more especially concerned, are ossifications for the most part not constructed upon a preformed cartilaginous base, whilst many of them deviate from the course and the results of the ossifying process of bone-cartilage. It will be seen that in these last referred to, there is an absence of the vascularization proper to the cartilage in its transition to bone, an absence of that lamellated structure-development, with that grouping of the cartilage-cells, and that resulting arrangement of the bone-cells, which both exhibit in common. In isolated cases, as, for instance, in the ossifying of enchondroma, it is not in the intercellular substance, but in the cartilage-cell, that the ossification first commences. That ossified enchondroma differs in essential points from the texture of true bone, we have already seen.

Where no preformed cartilage, but rather a rude, firm, sod-like, and fibrous, or a fluid blastema, or, again, an anomalous, mostly fibroid texture, constitutes the groundwork of the ossification, the result is a concretion more or less uniformly penetrated by bone-earths, and presenting scarcely any analogy with the texture of bone.

2. The characters of ossification differ according to certain differences in the implicated textures; or, where the textures are identical, according to certain peculiarities in the process itself.

The ossification of a cartilaginous base has frequently, although not always, an import coequivalent with that of ossified bone-cartilage—in other words, the import of a progressive metamorphosis into a complex vascularized texture. Genuine bone-texture, on the other hand, can, in the present state of our knowledge, be traced to a cartilaginous base alone, the pre-existence of which, if not obvious, must be taken for granted.

The ossification of other textural bases, on the contrary, has, if we take into account other collateral changes which these bases, and more especially the fibroid textures, undergo, the import of a retrogressive metamorphosis, of a decay, of a destruction of the base. With the display of lime-earths in the shape of black molecules, the textures lose their color, their succulence, and their elasticity, waste, shrivel, toughen, dry up, and become more or less lardaceous. Not alone do no new bloodvessels make their appearance, but old ones, if there be any, become obliterated. Fluid blastemata in the course of cretefaction become turbid, chalky, gritty to the feel. Under the development of fat, they form into a pap, and eventually thicken down to a mortar-like concrement. Even in cartilaginous bases, ossification is often so modified as

to represent rather a retrogressive than a progressive metamorphosis (compare enchondroma).

3. A most important and comprehensive question relative to ossification generally, and therefore to bone-cartilage inclusive, is: whence are the lime-earths which incrust and penetrate the various soft textural bases derived?

A narrow scrutiny of the ossifying process, especially in fibroid textures, and of the cretefaction in soft and fluid blastemata, will speedily convince us that the appearance of lime-earths is not essentially due to their deposition out of either a pre-existent or a new-formed and special system of bloodvessels within the ossifying growth. For, when we see growths ossify, which are almost, if not entirely devoid of blood-vessels, and which are, at the same time, remote from the vascular system of other formations (for instance, free bodies within serous sacs); when we see the process of ossification often attended, not with any new growth of bloodvessels, but with the obliteration of existing ones; when, again, we reflect upon the concomitant changes wrought in textures during their osseous conversion, their wasting and discoloration, the interlarding of their shrivelling substance with free fat, we are fain to look upon the entire process as the result of the total transformation of the chemical constituents; as, in fine, an elimination of pre-existent lime-earths out of their primitive connections.

Even in the normal ossifying of bone-cartilage, the process is the same at the commencement, the lime-earths appearing long before the development of any vascular system. This, then, offers at least one connecting link for all processes of ossification.

In the revolutions effected by the ossifying process, a most important part is without doubt assignable to the accession of fat. It is common to all processes of ossification, and probably results both from a release of pre-existing fat from its primitive combinations, and of a simultaneous conversion of protein substances into fat.

From these preliminary remarks, we may at once proceed to a muster of the new growths belonging to this category, premising, however, that much relating to them will have to be discussed more at large in later chapters of the present work.

1. Uniform or almost uniform with the normal bone, are:

(*a.*) Bone developed in permanent cartilages, and especially in those of the larynx, sometimes and in part also the ossifications of costal cartilages. In them, however, we usually miss the lamellated structure of normal bone.

(*b.*) Bone-structures which form as callus for the reunion of fractured, and for the regeneration of lost bone, hyperostosis, whether external or internal (sclerosis), exostoses, and all osteophytes, including such as in the shape of thorny, stellate, or scaly bone-growths and fabrics, enter into, and sometimes greatly surpass in volume, certain concurrent new growths, especially such as occur in bone.

Notwithstanding the all but identical relations of the texture of these formations with that of normal bone, they present not a few important discrepancies, cognizable both by a general comparison with normal

bones, and by a special comparison with those directly implicated. Thus, as examples, we may adduce the inferior vascularization, inferior number of medullary canals, less marked lamellated structure, anomalous amount and irregular disposition of the bone-corpuscles, in the new bone-growths.

As regards the process of ossification in the several blastemata, that produced by inflammation is the best adapted for investigation, as being at once the most frequent, and the most voluminous. The flaky or fibrous basis of the exudate furnishes the fundamental (intercellular) substance of the cartilage. Within this cells become developed, which, following the process of physiological bone-formation, change into bone-corpuscles.

(c.) The slowly developed bone-nuclei in callus, arrested at the stage of a ligamentous formation in bone-fractures, trephine-gaps, &c.

(d.) Osseous growths developed beyond contact with bone on the dura mater, as also upon the cerebral arachnoid, and upon the free visceral plate of the spinal arachnoid membrane; the so-called ossifications upon the intermuscular ligaments in the vicinity of hyperostosed articulations, and of the membrana obturatoria of the pelvic foramen ovale. The ossifications occurring in tendons are said by Henle to be of true bone-texture, as are also, in fine, the bony concrements found impacted within healthy muscular textures.

2. *Osteoid.*—Several of the growths adverted to as deviating in certain points from standard bone-texture, might be transferred to this section. To it, however, belong more especially ossifications of costal cartilages, and most of all ossifying enchondromata, both in bones, and in soft parts. In bone there occur independent texture-supplanting tumors, which consist of an ivory-like, dense, white bone-substance, and which are seen both in this, and in their general character to be ossified enchondromata.

Müller's osteoid is a bone-formation which enters redundantly into the parenchyma of cancer. Its constitution is identical with that of true bone, and it will be discussed under the head of Cancers.

3. *Concretions.*—Under certain, as yet unknown, conditions, the lime-salts in a soft basis are, by dint of a revulsive metamorphosis, set free so as to incrust and penetrate the said basis,—in a word, effect its ossification.

Such bases are the fibroid textures and blastemata occurring in the form of independent tumors, of membranous expansions, or of irregular masses lodged and entangled within various parenchymata. They may be the product of inflammation (exudation), or merely of an anomalous act of nutrition. The blastema may moreover be extravasate-fibrin, or spontaneous fibrin-coagulate within the vascular apparatus. Such bases are fibrous tumors, fibroid hardened exudates upon normal and anomalous serous membranes, within parenchymata, in the cutis as scar-texture, on the heart's valves, in muscles, in the heart's walls. Such, again, are the central and the peripherous fibrinous deposits in extravasates, after entering upon a fibroid transformation, hypertrophous thickenings of serous membranes and of the tunicæ albugineæ. Such are, in fine, the Pacchionic bodies, the different fibrin coagula in the heart's cavities

(so-called vegetations), the stratiform deposits within arteries, and the soft matrix of the phlebolite in veins. To sum up, therefore—all the so-termed ossifications of serous membranes, of the thyroid gland, of the heart's valves, of fleshy muscle, of arteries, and of veins.

Even the fibroid fabric which enters into the composition of malignant growths, for example of cancers in soft parenchymata, now and then ossifies into a bony skeleton, or shell-like framework. This is, however, not to be confounded with the thorn-like, stellate and scaly stroma of true bone-texture, accompanying numerous heterologous growths as developed in and upon the bones.

The ossification offers little or no analogy with normal bone, and its development. The bone-earth enters in a molecular form, accumulating, for the most part irregularly, in the soft basis, until the latter is converted into compact bone. The bone-earth is capable of being withdrawn by acids, with restoration of the soft basis. It has sometimes acquired the aspect of a stratiform deposit. Many a wide-spreading ossification,—of the arteries for example,—is concurrent with excessive fat-production in its vicinity.

4. *Cretefaction.* Finally, fluid blastemata are also liable to ossification. The process is perfectly identical with that which takes place in the fibroid blastemata just enumerated. It is in like manner conditional upon a metamorphosis of the fluid blastema, by virtue of which, the incorporated lime-earths being set free, predominate. Morphologically speaking, the blastema displays the development of free or of celled molecules (granule-cells). It is always accompanied by fat in a molecular form, and by cholesterine crystals. The ossification manifests itself as a lardaceous chalky pulp, as a cement-like and friable, ultimately, as a compact calculus-like growth.

The blastemata entering into this process are either originally fluid, or else originally solid, and subsequently liquefied (croupous fibrin).

These blastemata are either exudates external to the vascular system, or deposits internal to the latter, for instance, accumulated layers within the arteries, the basis of vein-stones in the veins, or coagula smaller or greater in extent (vegetations).

To this series belong the cretefactions of fibrinous, albuminous exudates, of pus, of tubercle, of atheroma in arteries, of vegetations on the heart's valves, of coagula in the veins. Just like ossification, cretefaction presents various grades from the aforesaid progressive pulp-like thickening, to the cement-like, and at length the compact, calculous concretion.

A very peculiar kind of ossification is represented in the *cell-incrustations*, which have their physiological analogues in the pineal concretions. They appear in a variety of shapes in the vascular plexuses, especially in the turbid, chalky, speedily condensing moisture of the cysts of the choroid plexus, as also in sarcomata and cancers, more especially within the brain. The cells both as primary and as parent-cells, together with their contents, fill with lime-salts, now in a molecular shape, now in that of concentrical layers.

6. GROWTH OF BLOODVESSELS.

Setting aside all dilatations of the smaller vessels and capillaries, constituting the so-called aneurysma anastomoticum and telangiectasis, we shall here treat of all that concerns the new growth of bloodvessels.

Upon a somewhat slender foundation of facts with which we have to commence, we must endeavor to build up a superstructure of well-considered hypothesis.

1. The common occurrence of this new growth in inflammatory products is incontestable, more especially in the adventitious membranes, affecting serous tunics. It is at the same time matter of certainty that such new vessels by no means originate through any prolongation of pre-existent vessels in the contiguous textures, but that the new process of development is altogether an independent one, and that only at a later epoch do the new-formed vessels enter into anastomosis with the older ones.

The process of the new growth of bloodvessels in inflammatory products of this kind is as follows:

In the first place, blood is seen to occupy little vacant spaces within the blastema or exudate thrown out in consequence of the inflammatory stasis. In other words, certain, for the most part irregularly spherical, ramified, to the naked eye point-like, areæ, unlined with any proper membrane, fill with blood; areæ obviously determined by the upspringing of blood out of the blastema. Both their remoteness from old vessels, and their ulterior development, controvert the idea of the contained blood consisting of little extravasates. This blood represents an aggregate of blood-corpuscles which the concurring testimony of Vogel and myself has shown to be of various magnitude, for the most part imperfectly round; not precisely disk-like, nor possessed of the intense redness of old blood-corpuscles. They are moreover soft, and adhere both to each other and to the parietes of the containing areæ. From these areæ there are gradually developed in all directions, sometimes, however, predominantly in a single one, blood-streamlets contained within chinks or canals in the blastema, and having no perceptible confining membrane. In minuteness of calibre, they excel the finest capillaries. Their next step is to become invested with a structureless confining membrane, the internal bloodvessel membrane, to which the outer layers associate themselves by and by. Finally they shoot out from various centres to anastomose with one another, and eventually with the old textural vessels.

In this process two material points remain unaccounted for, namely, first, the primitive development of the blood-corpuscles in those central areæ. The question is—their spontaneous and independent origin out of the common blastema being manifest, what portion of the latter is devoted to this purpose, and what chemical changes does the act involve?

Secondly: How do the vessels originate? In reference to the last question it may be asserted:

(a.) If we may judge from past observations, the vessels in these blastemata do not spring from primary blood-holding cells. The absence at first of any sharply defined contour in the said areæ disproves their

being primitive cells, or the blood-streamlets emanating from them, prolongations of cells. This leads us to the more probable assumption that:

(b.) The blood forms out of the blastema generally, and where the latter has wrought itself into cells, not within these, but in intercellular spaces between them. Where, however, blood and bloodvessel formation frequently precede cell-development, and more especially where it takes place in blastemata in which cell-development is either wanting altogether, or plays only a very subordinate part (for example, in fibroid blastemata), it can only be alleged that the formation takes place in chinks and chamberlets worked out of the blastema by the blood itself. The structureless membrane which subsequently confines the blood streamlets, is in all likelihood a secondary endogenous formation out of the blood, the remaining layers being built over it, so to speak, externally, out of the blastema.

As regards the relation of new bloodvessel development to the character of the blastema, the following points seem to present themselves for further inquiry.

(a.) In different products of inflammation, there is virtually a very different degree of proneness to the new growth of bloodvessels. In some it is excessive, in certain others, in the progress of development into precisely the same texture (the areolar), it is very faint indeed. Upon the parietal layer of the cerebral arachnoid, adventitious membranes occur, which along with an inconsiderable amount of nuclei, of nucleated cells, and of areolar-tissue fibrils, consist mainly of bloodvessels.

(b.) The new growth of bloodvessels is directly proportionate to the general capacity of the blastema for textural change. Blastemata which linger long in their primitive crude condition, and also which serve for the groundwork of indistinct fibroid textures, exhibit little, if any, new growth of bloodvessels.

The new formed vessels give token of a delicacy of structure, and a vulnerability in full accordance with their recent origin. To this are probably due the hemorrhagic products upon their becoming, when only just formed, the seat of inflammatory stasis. The fact is, moreover, to be inferred from the new-formed vessels originally consisting of the primitive vessel-membrane alone; and not until a later period attaining the perfected organization of old bloodvessels. In new-formed vessels of considerable calibre, we have frequently missed the layer of transverse-oval nuclei.

The vascular apparatus of new growth is marked by, for the most part, long, stretched vessels, by rare dichotomous ramification, with hardly perceptible decrease of calibre, or by a wide-meshed disposition.

A nervous system has not as yet been demonstrated in them.

The new bloodvessels both in the spurious membranes adverted to, and also in other new growths, are, as experience has again and again shown, susceptible of inflammation, and this in very early stages of their development. Here, for reasons both self-evident and already referred to, they are wont to yield essentially hemorrhagic products.

The manner in which their anastomosis with old vessels, or with par-

ticular arteries or veins is effected, has not been thoroughly made out by experiment. As regards the former case, it is probable that the anastomosis is brought about by an act of resorption in the wall of the old vessel, at the point where the *new* vessel rests.

With respect to the second point, Van der Kolk has distinctly shown that in the case of adhesions of the lung to the costal parietes, a connection becomes established, on the one side with the system of the pulmonary artery, on the other side with the aortic circulation. And although this fact may not warrant all Van der Kolk's deductions, it is nevertheless highly important, as offering a connecting link with other observations of his own upon anastomoses, to which we shall hereafter have to revert.

New-formed vessels doubtless undergo obliteration in spurious membranes, just in the same gradual manner as in the cicatrix, and probably for the most part in the progress and sequel of the retrogression,—the wasting—of the new textures themselves.

2. As in inflammatory products, so also in other blastemata, does a new growth of bloodvessels occur. An examination of these proves incontestably that there is a second mode in which a new growth of bloodvessels may take place. We have satisfied ourselves of the *development of new vessels out of parent-cells in cancerous structures*, composed, amongst other elements, of spherical or acinus-shaped parent-cells. Numerous cyst-like cells, with structureless parietes, contained in place of the brood cells of carcinoma, soft, adherent blood-corpuscles. Many of them bulged out in all directions into cœcal sacs, freighted with the same contents, and entering into anastomoses with others. Blastemata, therefore, entering into cell-development, are in reality capable of producing a new development of bloodvessels out of cells.

Certain new growths are remarkable for being rich, others for being, in various degrees, poor in bloodvessels. The former are mostly loose in texture, and consequently capable of considerable intumescence. Their hyperæmiæ are for obvious reasons, readily exaggerated into hemorrhage, and their inflammation is especially liable to determine hemorrhagic products. Amongst the malignant new growths, medullary cancer is notorious in this respect. Its highly developed vascular apparatus is doubtless the source of its excessive nutrition, of its rapid and often monstrous growth. It is pronounced by Van der Kolk to be of arterial nature, in other words, to anastomose with arteries only.

Very vascular new growths form the transition-link to nearly pure bloodvessel-formation. For blastemata are not wanting which present almost exclusively the groundwork of new bloodvessel-formation—in other words, of a new growth, consisting almost exclusively of new bloodvessels.

To this class belong the following new growths, concerning the development and import of which much obscurity still prevails.

(*a.*) *Cavernous textures, cavernous blood-tumors.*—These growths are of a cancellated structure, somewhat resembling that of the corpora cavernosa. They consist of areolar tissue fibres, constituting a multilocular stroma, the interspaces of which are invested with a structureless membrane, and contain blood. Numerous caudate cells liberated during

the investigation appear to be the débris of an epithelium. The intercellular spaces communicate amongst each other ; for, by pressure, the tumor may be completely emptied through a cut surface. They are surrounded by a tolerably dense capsule of areolar tissue, along with which they may be peeled out of the textures. They always communicate with a considerable vein, through which they will take up an injecting mass. No arterial ramification is demonstrable within their texture. They are very tumefiable, forming upon the surface of the body and at the periphery of organs, protuberant, compressible, but resilient dark-blue tumors, which supplant the original textures.

They are commonly regarded as telangiectases, and without doubt they pass, with many practitioners, for examples of fungus hæmatodes. By the French (Andral), they are termed "spurious spleens" (Aftermilzen), "placenta-like textures." In our opinion, they are by no means dilatations of bloodvessels, but to all intents and purposes new growths, and, as far as our experience goes, altogether benign. We have never seen them concurrent with a malignant growth.[1]

Their mode of development is not clearly made out. Numerous observations, however, render it probable that they originate as bloodvessel growths in their most extended sense, as blood-bearing depôts and canals, formed by absorption out of a solid blastema. Whether the contained blood be a primitive endogenous new growth, or become introduced into it subsequently, we know not. Thus considered, they bear a close analogy with the fenestrate and canal formation in certain blastemata pertaining to the vascular system, in the superincumbent layers upon the inner membrane of arteries, in the fibrinous coagula within the vascular system, in the thrombus, &c.

Within the cancelli small concretions occasionally form, corresponding to the phlebolites of veins.

We have seen these cavernous textures developed in the subcutaneous areolar tissue of the thigh, and communicating with the saphena vein, as also in the substance of the lips. We have seen them as tumors growing out of the diplöe of the skull-bones, and penetrating the compact outer skull-plate, and, again, in the texture of the pia mater. They are the most frequent of all, and the most various in dimensions, up to, and beyond those of a duck's egg, in the liver, where they communicate with branches of the portal vein.

(b.) *Fungus hæmatodes, blood fungus.*—What is commonly held to be *fungus hæmatodes*, is a luxuriant vascular growth, mischievous, both from its liability to occasional hemorrhage, and from its tendency to ex-

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[1] The author has since modified his opinions respecting these, so-called "cavernous textures." Abandoning the ground of their development out of a solid blastema through partial resorption, he now regards their stroma as nearly identical with that of cancerous structures, both in its elementary constitution, and in the fact of the same dendritic excrescences springing from its septa, and growing into its chamberlets. The affinity of these tumors to cancer, the author considers further established by their not unfrequent concurrence in the same organ (the liver, for instance) with cancerous tumors.

Incipient cavernous tumors do not, it is asserted, present any anastomosis whatever with the venous system. The anastomosis is established only at a later period through the mediation of very minute venous offshoots. It is not made clear, however, in what manner this communication is brought about. (See Rokitansky "über die Entwickelung der Krebsgerüste." Sitzungs-berichte der Kais. Akad. der Wissenschaften, März, 1852.)—ED.

haust the constitution by the habitual hemorrhage consequent upon its ulceration. Some deem it curable by removal with the knife, whilst others maintain that it invariably recurs either at the same spot or elsewhere, and that it is of cancerous nature. Some even fancy, that not only does it return in its primitive form, but that it may be replaced by another form of cancer, namely, the medullary or encephaloid.

Our own opinion respecting the occurrence of fungus hæmatodes, is to this effect:

1. It is incontestable that highly developed, bleeding, ulcerating *telangiectases*, as also *cavernous textures*, are frequently mistaken for *fungus hæmatodes*. This applies still more forcibly to a highly vascular, bleeding, ulcerating, blood-turgid medullary carcinoma. Nay, we have often seen a tumor, diagnosed as fungus hæmatodes, resolve itself in the dead subject, after collapse of the bloodvessels had taken place, into a medullary carcinoma.

2. We are convinced of the existence of new growths almost wholly made up of bloodvessels; we even regard it as likely that the so-termed telangiectases (both congenital and adventitious) are, for the most part, new growths of bloodvessels.

The question with respect to such luxuriations of bloodvessels is, what is it that determines their (innocent or malignant) character? We believe this to reside, not in the bloodvessel growth, but in the remaining, namely, the intervascular, portion of a common blastema; just as in the malignant osteoid, not the bone-luxuriation, but the adjacent, soft heterologous parenchyma, determines its cancerous nature. Scanty as is the proportion of this intervascular heterologous substance to the bloodvessel growth, the former merits, in this sense, the most ample consideration, and however important bloodvessel luxuriation in itself may be, the term fungus hæmatodes designates but a secondary feature of the entire new growth, a modification, through accidental excess of vascularity, of a new growth well-marked, only imperfectly examined. The designation *vascularized areolar tissue* or *vascularized cancerous formation*, would be both more philosophical and more practical.

Hence the benign or malignant character of a new growth associated with excessive vascularity would, in such cases, be primitive.

Can the benign new growth of this kind become malignant? It can hardly be doubted that vascular tumors may accidentally become the nidus of a malignant new growth, just as may any natural organ rich in bloodvessels.

Another kind of consecutive degradation to malignancy is also conceivable, although for the present little more than hypothetical.

To explain our meaning it will be necessary to enter upon a little further discussion.

Bloodvessel luxuriations represent, when the anastomoses with the old vessels are completed, a new vascular apparatus complete within itself, a repetition, so to speak, of the portal system.

The anastomoses might be of such a character as to attach solely either to the arterial or to the venous system. Van der Kolk considers this proved, maintaining that medullary carcinoma belongs exclusively

to the arterial, and another new growth, which he denominates fungus hæmatodes, to the venous system alone.

A vascular apparatus of this kind not being conceivable without interchange of matter, it might be not unreasonably inferred—

1. That the products of such a process would differ according to the arterial or to the venous nature of the blood circulating in the tumor, and that the products would be more especially anomalous where blood, previously rendered venous, has to permeate a secondary system of capillaries. In both cases the circulation must needs become torpid, and prone to undergo stases.

2. That the crasis of the general circulation must suffer a change, more especially in the second case.

In such wise, vascular luxuriations might indeed be imagined to pass into malignant new growths, the product, namely, of their interchange of matter constituting a malignant blastema.

The new formation of *lymphatics* has been demonstrated by Van der Kolk in adventitious membranes and in cancers.

7. FAT FORMATION, FATTY DEGENERATION.

The anomalous occurrence of fat is no less frequent than multiform. The subject is daily acquiring fresh interest, in proportion as the importance of fat in the animal economy, from the incipient stage of digestion through every process of assimilation, up to the formation of the elementary cell, renders itself more and more apparent. It is, however, quite within the compass of pathological anatomy to testify that protein substances, and in particular fibrin and albumen, are capable of undergoing conversion into fat.

FAT-TEXTURES.

1. *Normal.*—We have already adverted to the excessive formation of fat generally, and to its accidental unequable accumulation about certain organs, amid *general* wasting of this tissue, for example, in the omentum, the kidneys, the mediastina, on the pericardium, and on the heart. We have here to advert more particularly to fat collections still more marked by their locality and limitation. Of this kind are—

(*a.*) Those collections of fat important in various ways, which encircle diseased, and especially calculous kidneys, or such as have become atrophied through Bright's disease, or surrounding anomalous bone-formations (offering some analogy with the medullary system of bone), as in ossifying arteries, in the vicinity of cancers, &c.

(*b.*) *Lipoma* or fatty tumor, an accumulation of adipose tissue in the shape of a spherical, oval, lenticular, more or less lobulated tumor, invested with a delicate capsule of areolar tissue, and permeated by equally delicate and spare continuations of the latter. Its size is from that of a hemp-seed or a pea, to that of a man's head, or more.

It consists, as a mere repetition of the normal adipose tissue, of spherical fat-cells, wherein are discoverable stellate or radiating crystals of margarine or margaric acid. Where the areolar tissue capsule is more

strongly developed, the lipoma is an *encysted* one. We have met with such encysted portions in the midst of loosely lobulated lipomata.

The lipoma occurs chiefly in the subcutaneous areolar tissue, more especially in localities where fat is deposited in more than ordinary amount in the healthy state, and where accidental circumstances cause its still further accumulation, as at the glutæi, at the thighs, at the back and neck, about the shoulder. It is, however, also observed in parts less abounding in fat, as beneath the hairy scalp. Again, it occurs in the submucous areolar tissue of the stomach, of the intestine, even of the bronchia; in the subserous areolar tissue of the parietal, as well as of the visceral layer, although more commonly of the parietal, in serous and synovial sacs; for example, beneath the pleura and peritoneum, upon the inner surface of the dura mater, and upon the investing membrane of the ventricles. It has been fully described as it occurs in synovial sacs, more especially of the knee-joint, in that peculiar form which Joh. Müller has denominated *lipoma arborescens;* a form to which all lipomatous accumulations beneath serous sacs incline. We have also met with lipomata in glandular organs, more especially in the lungs, liver, and kidneys, in bone affected with osteoporosis and eccentrical atrophy.

Large lipomata in subcutaneous and in submucous areolar tissue, by dint of traction, acquire a pedicle, and lapse into the cavity, it may be of the intestine, in the semblance of a polypus.

Lipomata occur, for the most part, solitarily. Instances are not quite rare, however, of several, or even many coexisting beneath the subcutaneous areolar tissue. Such cases are the more remarkable, that they may affect individuals not otherwise at all prone to excessive fat formation.

In itself lipoma is innocent. By compression, traction, and hampering of space, it may be rendered nocuous. Moreover, its general integument may, through violent tension, through inflammation and suppuration, or through a sloughing process, occasion consecutive ulceration of the tumor, and exhaustion of the powers of life.

It is seldom traceable to palpable mechanical injury. In the majority of cases, more especially where several lipomata concur or succeed each other in growth, there is evidence of neither blow nor compression.

The term *steatoma,* so often misapplied of old, has been reserved by Johannes Müller to designate a peculiar species of lipoma, in which the fat texture is lobulated, as it were, through the intervention of a permeating membrane of areolar tissue, the latter forming a main constituent of the new growth, and imparting to it greater toughness.

2. *Abnormal (fat-texture).*—To this category belong, in the first place, cases in which the *contents* of the fat-cells vary from the natural character; in the second place, cases in which the cells themselves deviate from the normal type.

(*a.*) The fat-texture manifests, under certain conditions, anomalies for the most part referable to the nature of the contained fat;

(*a.*) The latter being sometimes preternaturally diffluent, like oil-fat (oleine). It is more than usually unctuous. On pressure or incision it gushes forth abundantly, and in big drops. In the dead subject, neigh-

boring parts are often found infiltrated with liquid fat. It is mostly tinged of a deep yellow, and resembles marrow.

These characteristics very commonly attach to the fat of the old and cachectic, laboring under osteoporosis (from atrophy), extensive ossification of the arteries, osteomalacia ; and of younger individuals a prey to cancer. Similar properties belong in an especial manner to fatty accumulations usurping the place of muscle or of pancreas-texture.

(β.) The fat contained within the cells is firmer, stearine-like, resembles mutton suet, and dulls the blade of the scalpel. The entire fat of the individual is of this character, more especially that of the subcutaneous adipose tissue, and it is commonly associated with strongly developed pigment formation in the rete mucosum, and a copious secretion from the sebaceous glands,—an oily skin. These properties mark, in an especial degree, the fat of younger dram-drinkers, and are almost without exception concurrent with lardaceous affection of the liver.

This variety may be caused by the fat containing a larger proportion of margarine ; perhaps, also, by the development of stearine. Spirit-drinking has a very marked tendency to produce it.

(b.) The fat-texture gives evidence of anomalies both as to the contents of the fat-cells, and as to the properties of the cell itself.

The *cholesteatoma* of Johannes Müller belongs to this class. It is invariably a local, circumscript new growth. The cholesterine holding stratiform fat-mass consists of thin, sometimes concentrically stratified, mother-of-pearl like, lustrous plates of scales, which, on a closer inspection, appear composed of partly spherical and oval, but for the most part polyedrical vegetable-like cells, one-eighth to one-sixteenth of a millimetre big. This texture accords with that of the tallowy, adipose tissue of the wether, only that the cells are smaller and more delicate. The majority of the cells do not appear nucleated; many others, however, more especially the younger spherical cells, show distinct nuclei.

Between the layers of this polyedrical cell-texture are visible crystalline deposits of fatty substances, mostly in the shape of rectangular tables. Barruel found them to contain cholesterine and a fat akin to stearine. The cholesteatoma commonly occurs encysted within a fibroid envelope, or within a cyst-membrane lined with a delicate epithelium. We have, in common with other pathologists, seen it thus in the subcutaneous areolar tissue, in bones,—those of the skull in particular,—in the pia mater, and in the brain. Johannes Müller met with it in cysto-sarcoma. It also occurs free, in the shape of a layer, as Cruveilhier has observed in urinary fistulæ, and Johannes Müller and myself upon the surface of an ulcerating mammary cancer. In our own case it was upon the sore surface of a fibrous cancer combined with epithelial cancer. We have met with it in the same combination upon the surface of a sloughing ulcer.

The cholesteatoma is in itself innocent. Inclosed within a cyst it usurps the place of surrounding textures, causing the forcible expansion of the osseous texture in bone, and occasionally perforating the common integument, when subjacent to it, becoming destroyed, and thrown off.

3. *Free Fats.*—The occurrence of free fat,—a condition, we think,

properly meriting the term of fatty disease—takes place under different circumstances :

1. It is immediately secreted as such. The seat of its deposition are normal textures and their elementary parts, or else pathological growths.

To the former category belong :

(*a.*) Tallowy infiltration of the liver, a condition in the one instance resulting from spirit drinking ; in the other, being the concomitant of tuberculous disease. In the former case it is often associated with a stearine-like character of the contents of the adipose tissue.

(*b.*) To the other mode of occurrence belong more especially the fatty contents of encysted tumors.

These fats differ in character. Thus, in ordinary fatty liver it is a normal fat, for the most part rich in elain,—in some varieties of fatty liver, the waxy liver, a more consistent fat, containing stearine and cholesterine.

In various cysts, again, we meet with a fat consisting in different proportions of elain, margaric, stearine-like fat and butyrine. Some cysts contain cholesterine alone ; the majority, other ingredients besides. In this mode of its occurrence, fat is very generally associated with epidermoidal and with osseous formation.

2. *Fat*, as such, is liberated owing to new combinations taking place amongst the ultimate constituents of a complex formation. Or, again, it is not set free as pre-existent fat, but is newly created, and this out of protein substances, by dint of an elementary transforming power.

This transformation or metamorphosis into fat is rendered highly probable, if not certain :

(*a.*) By the large proportion of fat found at various epochs in the place of parts, into whose composition fat assuredly did not originally enter in anything approaching to its subsequent amount.

(*b.*) By this appearance of excess of fat occurring under conditions which preclude its derivation from a vascular system. It takes place in growths destitute of bloodvessels and often remote from vascularized organs ; for example, in exudates, shut up within thick shrivelled sacs unfurnished with bloodvessels,—in stratiform deposits within arteries. It is even very commonly met with at the centre of the said formations, and therefore at the greatest distance from vascularized parts ; as, for instance, in the coagula, within the vascular system, in tubercule-granules, and in crude fibre in circumscript masses.

(*c.*) Lastly, by the appearance of fats in the said formations being the forerunner of their entire metamorphosis, and generally speaking of their destruction.

For this adventitious production of fat, more especially as it affects a blastema in the progress of development, or even perfected textures, we find the term *fatty disease* or *degeneration*, peculiarly appropriate ; and we consider it to be fraught with quite enough of interest for general pathology, to justify an attempt to enumerate here the various phases of its occurrence.

1. Appearance of fat in the blood [or fibrin] coagula in veins,—the

result of blood disease, either spontaneous, or consequent upon infection by inflammatory products.

2. The fatty conversion of coagula development through similar agency, in the capillary system, as so-called depôts—metastases.

3. In fibrinous and albuminous products of inflammation,—exudates, and especially pus. This is manifest in the exudates of serous membranes.

4. In the albuminous, fibrino-albuminous products of Bright's disease of the kidney. The spots involved in the process of fatty conversion are cognizable to the naked eye as dullish white, glistening, for the most part somewhat turgescent points.

5. In lardaceous infiltration of the liver. The lard-like blastema is seen here and there opaque,—of a dull white or whitish-yellow.

6. In tubercle, in incipient softening of the latter, and in like manner in crude fibrinous deposits.

7. In colloid, as met with frequently in the thyroid gland. In colloid of the more consistent kind, it is discernible with the naked eye as opaque, dull-white or whitish-yellow spots.

8. In cancers, where it enters into emulsion-like combinations with albumen, as also into saponaceous, glutinous conjunctions with bases—saponification of cancers. The points de départ are here the yellow, fibrinous masses which either traverse the cancer as a so-called reticulum, or else occur as circumscript accumulations.

9. In atheromatous disease, in the strata that form upon the inner surface of arteries, and in the soft matrix of phlebolites.

10. In the fibroid blastema and texture, more especially of fibroid tumors and exudates.

11. In the annulo-fibrous tunic of arteries, where it occurs, either pure or combined with, and dependent upon, stratiform deposits and their metamorphoses—atheroma and ossification.

12. In the muscles, especially the involuntary, and the heart in particular. Here we encounter an obvious conversion of the muscle-fibrils to molecular fat, with loss of the transverse striæ and inflation of the sheaths.

Further investigations are necessary to determine whether milky blood—the peculiar aspect of which is due to fat ; and whether the pellet-like excretion of fat from the intestine, should be classed along with the above.

This conversion into fat, affects, as we have seen, now crude fluid and solid blastemata, now such as have attained to various grades of textural development ; finally perfected textures.

The form or type under which the conversion takes place is very frequent, more especially in fluid blastemata. It has been described under the head of "metamorphoses of blastemata."

This process is in one sense to be regarded as a propitious event, as reducing certain growths to a condition readier for resorption and for reassimilation. Moreover, it determines a state of involution, isolation, extinction of the involved growths.

In this latter property, it is often allied with ossification and cretefaction of blastemata and textures,—processes offering many points of

analogy, and even of affinity with fatty disease from the disengagement of pre-existent fat. We need only advert to the cretefaction of crude blastemata in the coagula of the larger bloodvessels, and in the capillary system, in exudates, in pus, in tubercle,—to the ossification and crete-faction of stratiform deposits in the arteries, of the fibroid textures, &c., —and compare this process with the collateral one of fatty conversion.

Fats, the product of conversion, may present much variety of cha-racter. In most instances they are fluid fats in a state of minute molecular subdivision, in larger, lustrous, strongly refracting,—or else, in less bright, yellowish, tough globules. In exudates, in tubercle, in colloid substances, in cancers, and especially in the atheroma of arteries, cholesterine is frequently encountered in a crystalline state.

8. EPIDERMIDAL AND HAIR FORMATIONS.

The excessive production over expansive surfaces, both external and internal, of epidermis, with a normal form and aggregation of its elements, is often well exemplified, so far as the mucous membranes are concerned, in those of the œsophagus and vagina. There are, however, epidermidal luxuriations besides, marked by several peculiarities, such as site, circumscribed locality, unusual aggregation of elements. To these belong, also, the epithelial layers investing the various cysts.

(*a.*) As *epithelial* contents of encysted tumors. The form of the cells is most commonly that of tessellated epithelium cells.

Upon the external skin these tumors manifest themselves as luxuriant new growths, sometimes overspreading a wide surface, sometimes limited to a smaller space, occasionally as cyst-like developments of cutaneous follicles with their excretory ducts. These growths not rarely attain to a considerable circumference, and are distinguished by a peculiar anomalous arrangement of their elements, as also by, on the one side a retarded, on the other an excessive, horny character of the elementary cells.

(*b.*) *Clavus*, a local accumulation of epidermis-cells, of a conical shape with the apex pointing to the interior of the papillary body, with a superimposed disposition of the cells not deviating from the normal.

(*c.*) *Warts.*—Of these there are sundry varieties. The most ordinary consists of cornified epidermis forming a sheath-like receptacle of con-siderable thickness for the hypertrophied cutaneous papillæ. Others are marked by the elongated fibrous arrangement of very luxuriating cells, as polyedrical, edged cylinders in parallel array, some of which show imperfect cornification. They have a fibro-villous, appearance, are humid, and readily broken up by pressure into fibres and their elements. Their cells are devoid of nuclei, and in only a few instances cornified.

(*d.*) *Ichthyosis.*—The higher grades alone concern us here, the epi-dermis covering a papillary body, proportionately hypertrophied, luxu-riates into polyedrical tessellæ, cylinders, and disks. The disposition of the cells, at least in the cylinder form, is a fibrous one, parallel to its length. The degree of cornification is not in every case the same.

(*e.*) *Horn*—cornu cutaneum—a very common, for the most part dingy-

brown, longitudinally ribbed, more or less curved, cylindrical or conical horn-growth, springing from a cutaneous follicle of cyst-like development. It attains now and then to several inches in length. Its structure is seemingly fibrous. The cornification of the cells is very marked. It affects parts abounding in follicles, or hairy surfaces and their vicinity; for example, the forehead, the neighborhood of the pubes, and again, the back and the upper extremities.

All these growths are in their nature innocent.

Besides these, however, there occur upon the common integuments, as also upon the mucous membranes, growths which, although often extirpated with a favorable result, occasionally prove malignant and assimilate in all respects to cancer. Their elementary cells repeat the form of the non-cornified, nucleated, tessellated epithelium-cell, not rarely with a fibre-like prolongation, and whose secondary arrangement often displays the areolar type, or else represents in fibrils a moist velvety growth, similar to hypertrophied cutaneous papillæ.

Anomalous hair occurs in various shapes with reference to the form, color, length, and thickness of the hair-cylinder.

Besides their appearance at unusual points of the external integuments, especially upon pigment nævi, we have to advert to—

(a.) Hair contained within encysted tumors. This is commonly mingled with fat and epithelium. It is extremely frequent in the fatty cysts of the ovaries, but is also found in those of the omentum, of the cutis, of the subcutaneous areolar tissue, and even of the lungs. In these cysts it is often found to pervade the fat, as with a felt growing out of variously-sized patches, closely resembling the cutaneous texture, from the inner surface of the sac. Its development is here seen to be entirely identical with that of hair upon the common integuments.

(b.) Hair upon mucous membranes. It has been detected upon various mucous membranes, including even the conjunctiva of the eye.

Very small, partly microscopical, hairs are sometimes mixed up with the contents of encysted tumors,—with cholesteatoma. Those said to occur in the different secretions, the urine for example, are evidently derived from a mucous membrane.

9. PIGMENT FORMATION.

Irrespectively of all other anomalous coloration, but with a retrospect to that conversion of blood-pigment alluded to under the head of *hemorrhage*, we shall here treat of *granular pigment*. It appears under the various shades of jet, of Indian-ink black, of russet, or of a yellow-brown.

It occurs both free and enclosed within cells, in the shape of very small, spheroidal, scattered or clustered granules, together with rod-like molecules (with molecular motion), or else in the shape of larger spherical or spheroidal, in like manner either scattered or intimately grouped and blended corpuscles. The cells are for the most part spherical, but in certain conditions, as in cancer melanodes, spindle-shaped, caudate, rarely twigged.

These various modes of occurrence, together with the several shades

of the pigment enumerated, are, as we shall afterwards see, partly phases of development of the pigment, partly due to external conditions.

It hardly ever occurs quite pure between the elementary parts of a texture. Generally speaking, it adheres to a blastema at some period of textural development, for instance, to inflammatory products, adventitious membranes, colloid, malignant growths (melanosis).

When small in quantity and equably distributed, it determines slate-gray coloration of the textures, or a speckling or streaking with black dots. It may, however, manifest itself in larger knotty accumulations so as to consolidate and lay waste the textures, as for example in the lung.

It affects the fluids also.

Before speaking of its origin, it is requisite to pass in review, *as a simple series of facts*, the several cases of its occurrence.

In normal textures, the sequence of its amount and frequency is nearly as follows:

1. *In the lungs*, its seat is the true pulmonary texture, but also the interlobular areolar tissue. When inconsiderable in quantity, it lightly marbles the parenchyma. When abundant, it forms greater, branched accumulations, and along with these, distinct solitary masses of from a hempseed to a bean in size, and presenting a cut surface of dull metallic lustre. Or, again circumscribed patches of the parenchyma, especially at the apex, may be so replete with it as to display it as a coherent, black, hardened, impervious mass. It is particularly redundant around shrivelling, cretefying tubercles, healing cavities and cicatrices at the apices of the lung. It is for the most part found pure, free, in the shape of a minute molecule, unattached to any ostensible blastema, and certainly very seldom contained within cells.

In manhood and old age it is but a physiological product. In the earlier periods of life alone, as those of boyhood and youth, or when by its quantity it proves destructive to the pulmonary texture, is it to be regarded as a morbid phenomenon. The notion of its being peculiar to old age is correct only in so far as, in the aged, it is for explicable reasons hardly ever absent. It does not, however, by any means belong exclusively to this period of life. If it almost invariably abounds in the old, it is because in them there has been ample time for its accumulation. Still it is undeniable that the condition of the blood in advanced age, with its augmented proportion of blood-corpuscles, and its simultaneous diminution of fibrin, must essentially favor the deposition of this substance.

2. *In the bronchial glands*, from a speckling and extensive marbling up to the point of considerable increase of volume in the gland, and its conversion into a hard Indian-ink-colored tumor, in which the glandular parenchyma has perished. Its amount is here commonly proportionate to that in the lungs. Its form is that of free molecule, very rarely of molecule contained within cells.

3. *Gastric and intestinal mucous membrane.* In the shape of free molecules, often coherent in larger masses. It occurs thus both in the muco-membranous texture itself, more particularly at the great concourse of the solitary and of the aggregate glands, as also in the intestinal villi,

imparting to the mucous membrane an aspect, to the naked eye, of being lightly brushed over with black, or uniformly tinged of a slate-gray. Where the accumulation of pigment is considerable, the part appears of a blackish gray, or it may be of a deep black.

In rarer instances, the tracheal and bronchial mucous membrane is pigmented—that of the uterus frequently.

In the majority of cases it accompanies the more intense chronic catarrhs (blennorrhœæ) of the stomach and intestines. When affecting the intestinal glands, it points to antecedent hyperæmia, stasis, and exudation—typhus, for example,—at every age, even in delicate children, to a diarrhœa-like process in the follicles of the colon.

4. *In the mesenteric glands*, it is for the most part limited in amount, and concurrent with pigment in the intestinal mucous membrane. Here, again, it is a sequel to typhous hyperæmia and effusion.

In other lymphatic glands, the seat of hyperæmia, hemorrhage, inflammatory stasis, and exudation, it is less frequent.

5. In the *central ganglia* of the *abdominal sympathetic*, more especially the *ganglion* of the *solar plexus*, usually combined with wasting thereof, as a sequel to typhous hyperæmia. For the most part small in quantity, has a uniform, pale, slate-gray coloration, or is visible as blackish dots or striæ.

6. In the *common integument*, as the so-called *melasma* of the old or cachectic, as a diffuse suffusion of the common integument with pigment, in the lower extremities, and as knotty pigment accumulations in the face.

In new growths :

1. In the blood-coagula in arteries, veins, and capillaries (metastases), whether spontaneous or due to inflammation of their coats, and terminating in their transmutation to fibroid shrivelling cords and cicatrices.

2. In *atheroma* that has discharged itself into the canals of arteries, and especially in the depôts and cicatrices formed in the act of its excretion.

3. In the *membranaceous growths investing hemorrhagic depôts*, as also in the *contents of hemorrhagic cysts*, being here of a russet or yeast-color. The black pigment found in the shrivelled and extinct ovarian follicles, after elimination of their contents (during menstruation), is here deserving of mention.

4. In *inflammatory products* upon serous membranes, as a black pigment, upon the peritoneum, more commonly of a brown, or rust-color upon the tunica viginalis testis, upon the pleura, the pericardium, the arachnoid. It adheres to the exudate from the commencement, that is, from its crude condition, through all its phases of textural development, up to the areolar or the fibroid structure. According to its proportion, it manifests itself as spotted, striated, or uniformly slate-gray, bluish-black coloration,—always occurring as free pigment molecule.

It is more rare in the inflammatory products of parenchymata. Scar-textures are, however, not exempt from it, even in the common integuments.

To its appearance on serous membranes we have to add that detected upon the inner membrane of cysts, and of the cyst-like developments of

various hollow organs and canals—for instance, upon the inner surface of the dropsical Fallopian tube sac.

5. In *tubercle*, that is to say, the hemorrhagic tubercle, both in paren-chymata and upon serous membranes.

6. In colloid,—mostly as a brown tint.

7. In *cancer melanodes* (commonly called *melanosis, malignant mela-nosis*), a heterologous growth, consisting of medullary carcinoma with pigment. The brown and black pigment is here partly free, partly contained in cells, with the character of cancer cells. The medullary carcinoma is in various degrees spotted or striated with the pigment, or, in fine, so replete with it as to appear throughout dark brown or black. (See *Cancer melanodes*.)

In *fluids* :

1. In the fluid portion of the exudate in serous sacs.

2. Mingled with the contents of the larger cysts, and of hollow organs in process of cyst-like development; for example, the dropsical tube-sac.

Finally, it occurs under several *special conditions*, as :

1. In the black substance present in acute softening of the stomach; in the black contents of the stomach and intestines generally.

2. In the pulp constituting the rare black softening of the spleen.

3. In the detritus of necrosed textures, especially in dry gangrene, or mummification.

4. In the parietes of ill-conditioned abscesses—ichor depôts; but most of all at the margin and base of every variety of intestinal ulcer.

This preliminary will serve as a useful starting-point for an inquiry as to the groundwork of pigment, and the conditions upon which its appearance depends.

That the groundwork of pigment is the coloring matter of the blood appears to us proved, the cases in which pigment is obviously derived from hæmatin and blood-corpuscles being so numerous as to exclude all doubts on the subject. Still the circumstances by which the conversion is brought about, and still more the various shadings of the pigment, are unexplained.

The cases in which the metamorphosis of blood-pigment—that is of blood-corpuscles into pigment—is manifest, are of the most common occurrence. Such are the cases of hemorrhage, and of hemorrhagic exudates in serous sacs, more particularly the peritoneum, of hemor-rhage from intestinal ulcers, of pigment development in blood-coagula within vessels, of black or dark-colored softening of the stomach, of black contents of the stomach and intestines generally, &c.

But hemorrhage, whether simple or combined with inflammatory exudation, cannot, in all instances, be assumed, still less proved. In the other cases, therefore, where pigment occurs, for example, in the lungs, the lymphatics, &c., we must, whilst still holding on to the belief that hæmatin furnishes the groundwork of the dye, look around us for some further mode of elucidating the mystery.

The pigment may, independently of any development out of extra-vasated blood, be brought about through—

1. The obliteration of small bloodvessels or capillaries, with the con-

version to pigment of their contained blood, just as the stain is produced in plugging blood-clots within the greater vessels. As the vessel's parietes disappear through absorption, striated accumulations of pigment corresponding to the course of the vessel, are entailed in the textures. This takes place more particularly in membranaceous areolar tissue formed upon serous membranes previously vascularized. In these the opportunity sometimes offers of tracing the aforesaid process. Even the pigment in callosities entailed by so-called capillary phlebitis may be partly brought about by the same means, namely, by metamorphosis of the blood-coagulum in the vessels destroyed.

2. Through conversion to pigment of the blood in different blastemata, especially the products of inflammation and cancer melanodes.

3. By transformation of the blood-pigment along with other substances in the normal act of nutrition, or in consequence of hyperæmia and inflammation. The probability of this event will be relative to the predominance of the blood-corpuscles in the circulation generally, to the number of old and very highly colored globules present, and lastly, to the extent to which their coloring matter is taken up by the plasma when attenuated through the diminution of its salts, or the destruction of its fibrin. In this manner it is intelligible how pigment may become engendered without the extravasation of blood-corpuscles, how it so often becomes deposited in the lungs, as the central receptacles for venous blood, how the aged are peculiarly prone to its deposition, and lastly, how in certain crases, the typhous for example, it so frequently attaches, as the residue of hyperæmia and inflammation, to the follicular apparatus and the mesenteric glands.

It would seem to have arrived at certain organs partly through resorption—the bronchial glands, for instance.

Our own investigations concerning the morphological process of pigment formation have led to the following conclusions :

The pigment differs according as its basis consists of hæmatin alone in a state of solution, or of blood-corpuscles.

In effusions reddened by dissolved hæmatin, the pigment separates as a result both of the changes produced by resorption, by the accompanying menstrua, and by consolidation, and also of probable external agencies effecting coagulation or precipitation in the shape of a granular mass (of discrete or agglomerated molecular granules), which imparts a brown, a yeasty, or black coloration.

Where *blood-corpuscles are actually present*, either these become dissolved, and the development of pigment out of the coloring matter takes place as in the foregoing case ; or else the hæmatin becomes pigment within the blood-corpuscles, which thereby become transformed into mulberry-shaped corpuscles. These remain separate, or cohere in groups of two, three, or four, or they may even gather together into a lobulated mass. Earlier or later they break up into the molecular pigment-granules before adverted to. Under both forms and modes of development the pigment is, to a greater or less extent, contained in cells. Upon this point, experience has shown us that—

1. Pre-existent nucleated cells (of various forms) take up hæmatin, which, as the contents of the cells, becomes molecular pigment. This

is, perhaps, a repetition of the process that takes place in normal pigment formation.

2. One or more mutually coherent blood-corpuscles constitute, as it were, a nucleus-formation, around which a cell-wall developes itself. Even within this cell the nucleus-mass may break up into molecular pigment. The hæmatin frequently associates itself, dissolved, to the cell's contents, and there coagulates to molecular pigment, whilst the now colorless nucleus-mass (blood-globules) undergoes, probably in its protein contents (globulin), conversion into fat-globules.

3. A cell-wall forms around a conglomeration of molecular granules.

These are frequently all concurrent processes, just as happens with pigment formation external to cells. The two former processes are, however, both attested in colored exudates, and most particularly in *cancer melanodes*. In either way, a sort of pigment granule-cell is brought about.

The precise manner in which the conversion of hæmatin to pigment takes place, is obscure, if not altogether unknown. It cannot be doubted that the hæmatin undergoes various and considerable changes. Some indications in point are seemingly derived from the conversion of hæmatin into pigment, through the palpable influence of chemical agents, addressed, sometimes to the hæmatin itself, sometimes to the iron it contains. Strong mineral acids (sulphuric, for instance) introduced from without darken or blacken the blood with which they come in contact. Carbonic acid gas (evolved, along with carbonic oxide gas, out of glowing charcoal) acts in the same manner upon the capillaries when a stream of it traverses the fauces; and a similar influence is exercised by acid secretions generated in the organism itself, as we have seen in alluding to colored softening of the stomach.

Like the blood itself, the kindred spleen-pulp (spleen-corpuscles) suffers the same transformation of its elements. The change of color is here most probably determined through the combination of hæmatin with different acids, carburet, chloride of hæmatin, &c.

The very frequent conversion of red hemorrhagic exudates upon the peritoneum into black strata, is most probably founded upon the influence of the intestinal gases upon the hæmatin. In common with ammoniacal gas, it is principally the sulphuretted hydrogen of the bowel which, acting (by exosmosis) upon the iron of the hæmatin, enters with it into a black combination, namely, sulphuret of iron. A similar effect is wrought by phosphuretted hydrogen in abscess and gangrene.

Fertile in results as are the above anatomical data relative to the fundamental principle of pigment, they seem to throw very little light upon the chemical processes by which the conversion of hæmatin is regulated. The influences adverted to under which hæmatin blackens, admit of no ulterior application. We are still reduced to the entailed general view of defective decarbonization of the blood, to which the abundance of carbon detected by analysis in the various black substances, certainly adds weight.

But even should the pigment, as Guillot affirms, of the black pulmonary artery, consist of pure carbon, this would in nowise refute our theory, namely, that it is invariably developed out of hæmatin.

Although russet- and yeast-colored pigment are obviously derived from the same uniform base with black pigment, yet the conditions upon which the existence of this pigment depends are little known, and its composition still less. Thus much is certain, namely, that in color it is susceptible both of deepening into blackness, and of fading into paleness.

Generally speaking, an organ is liable to become the seat of pigment formation proportionately to its vascularity, to its proneness to hyperæmia, inflammation, and hemorrhage, and to the extent to which its blood-supply is marked by excess of coloring matter, that is, by the venous character (Venosität).

The resorption of granular pigment is a fact! How this takes place, —how and whereby it becomes adapted for the process, is not known.

In itself pigment is an innocent new growth.

It is still of some importance to inquire what is to be thought of the distinction of pigment into *true* and *false melanosis*. Seeing that pigment has, under all circumstances, one and the same fundamental principle (hæmatin), and that our knowledge of its workings is limited, the distinction seems supererogatory. Which *is* the true and which the false?

We deem it most advisable to abolish the word *melanosis* altogether, and to substitute for it the term *pigment*, designating all growths, normal or pathological, into whose composition pigment enters, as *pigment-holding* or *pigmental*, and what has been called malignant melanosis, as *pigmental cancer*, or cancer melanodes.

10. COLLOID.

Colloid, colloid substance, is a sufficiently common heterologous formation. It is requisite, however, to state, that under this term substances have been brought together which, in a physical and chemical respect, are not perfectly uniform; for instance, the colloid of the thyroid gland, the substance of collonema, on the one side, and on the other, gelatinous cancer. Further inquiry may, however, show such differences to be but modifications and gradations of the same substance.

Moreover, the occurrence of colloid abuts so closely upon the physiological, that it is difficult to define its pathological significance. Thus, it accompanies the often mere passing development of the thyroid gland, the secretion of certain follicles undergoing occasional cyst-like development, especially at the cervix uteri; and again it forms the contents of glandular growths in the progress of cyst-like dilatation. In other instances the appearance of colloid is too obviously of pathological import to admit of any doubt. It constitutes both innocent and malignant new growths.

Colloid is a semi-fluid adhesive substance, resembling a saturated solution of gum or glue, or a fruit jelly. It is seldom colorless, ordinarily of a honey or pale wine-color, but often brown, or green, sometimes black. With all these tints it is clear and pellucid, and only now and then turbid, flocculent. Microscopically examined, it displays, in smaller or greater number, elementary granules, nucleated forms, nucle-

ated and non-nucleated cells, together with parent-cells, in rare instances (even in colloid of the thyroid gland) the *pouch-like* formations mentioned under the head of metamorphosis of blastemata, and even caudate cells.

With respect to chemical composition, the reactions are those of various gradations of casein, of pyin, of certain kinds of mucus.

Colloid is, for the most part, found accumulated in hollow organs, in follicular, alveolar, cystoid spaces, and so seldom free within a texture, that the former mode of its occurrence has been regarded as pathognomonic of its true character.

1. It is most frequently met with in the *thyroid gland*, so frequently, indeed, that few thyroid glands are examined in which more or less of it is not here and there detected. It is accumulated in the cyst-like dilatations of already existing acini, as well as in others of new growth. The disease represents lymphatic, and in further development, cystic goître. It occurs, moreover—

2. In *simple cysts* (whether new growths or morbid developments of pre-existent hollow organs, for example, cysts of the kidneys consecutive to Bright's disease), and also in compound cystoids,—of the ovary, for instance.

3. In the *pituitary gland*, as a pale amber-colored layer interposed between its two lobes, believed by Wenzel to be the cause of epilepsy.

4. In *serous sacs*, as a remarkable transformation of a fibro-croupous exudate into colloid. Andral has witnessed this in a pleural, and we ourselves in peritoneal exudates.

5. A colloid substance constitutes *collonema*, and its kindred, benign, new growths. (See Sarcoma.)

6. A colloid resembling the vitreous secretion of mucous follicles forms the contents of alveoli, and of their endogenous cysts, in innocent and malignant new growths,—sarcomata and cancers, especially true alveolar cancer.

A question of great interest is, whether *colloid* is secreted as such.

(*a.*) Several facts, especially the appearance of colloid in the Malpighian bodies of the kidneys, but likewise the transformation of the aforesaid exudates into colloid, afford conclusive evidence that, under some unascertained conditions, albumen and fibrin become converted into colloid.

(*b.*) Other facts render it probable that it is the product of an altered function of secreting gland-cells, or of the action of anomalous cells, parent-cells, aveoli, and cysts.

(*c.*) The colloid of the thyroid gland in its voluminous occurrence, as endemic goître, merits an attentive consideration on account of its character of exclusiveness in relation to tuberculosis. The alienated habit of body acquired in endemic goître, may, indeed, be indicative of a change in the crasis; although as to the nature of such change, its relation to the function of the thyroid gland, and its character of antagonism with tuberculosis, we are altogether in the dark.

Colloid undergoes many spontaneous changes. Besides its resorption, as observed in colloid of the thyroid gland, it becomes, in cyst, diluted by the thin secretion from the cyst-wall, or else, under gradual extinc-

tion of the cyst, condensed, and eventually changed into a brittle sub-
stance resembling dried glue. Lastly, it achieves—

(*a.*) A remarkable conversion to molecular fat, becoming yellowish,
turbid, opaque, and unctuous (colloid of the thyroid gland, gelatin of
alveolar cancer).

(*b.*) In a few instances, cretefaction,—in colloid of the thyroid gland.
These cases are, however, quite distinct from the cretefaction and ossi-
fication of fibrinous exudates within the strumous thyroid gland.

11. CYST AND ALVEOLUS.

In asserting cyst to be a substantive new growth, with a distinctive
elementary groundwork, we exclude all accidental cyst formations, that
is, capsules and sheaths forming around foreign bodies, extravasate, or
entozoa (cysticercus, for example); as also cyst-like disease of hollow
organs consequent upon the closing and obliteration of their excretory
ducts and orifices,—for example, in the gall-bladder, the Fallopian tube
and uterus, the vermiform appendix, the sebaceous and muciparous
glands. Certain gland-elements, however, and in particular those of the
thyroid gland, demand an especial consideration, inasmuch as these
hollow bodies represent a certain stage in the process of cyst formation,
having the same elementary structure, and being susceptible of ulterior
cyst-like development.

Let us begin with the results of an examination with the naked eye of
perfect cysts, and in particular of the exquisite specimens so frequently
met with in the ovaries.

We have the simple (unicancellated) and the compound cyst (Müller's
compound cystoid). The first is sufficiently characterized by its name.
Compound cysts declare themselves by phenomena which induced
Hodgkin to distinguish them in two classes, although types of both
very commonly coexist in the same formation. The first comprehends
a cyst-formation with cysts of a secondary order in the parietes of a volu-
minous (parent) cyst; and these secondary cysts involve, in like manner,
cysts of a tertiary order within their parietes. These filial cysts project
upon the outer surface of the parent cyst, rather than upon its inner
surface, where they are in a degree flattened. The wall of the parent
cyst often appears separated, receiving the secondary cyst, as it were, in
a chink.

Such a formation is to be distinguished from a group of simple cysts
developed in mutual juxtaposition, some one of which predominating in
size, flattens the contiguous smaller ones. A group of smaller cysts in
an ovary may readily mislead; making it seem as if the fibrous capsule
of the ovary were but the wall of a cyst, and as if the smaller cysts
interposed between it and a contiguous larger one, were secondary
cysts.

The repetition of this process of secondary cyst-formation frequently
leads to a very complex cyst-formation, wherein, however, for the most
part, a cyst pre-eminent in size, reveals itself as the parent or primary
cyst in whose parietes the cysts of the second order become developed.
Sometimes the primary cyst is so prolific of this secondary cyst deve-

lopment within its parietes, as to endow the latter with a considerable thickness. It may even cause them here and there to degenerate into a tumor, consisting of an aggregate of cysts, collocated like facetted pouches in the breadth of the cyst-wall, and presenting a polyedrical cell-structure, in and upon the walls of which smaller cysts arise. Occasionally a cyst-wall, so constituted, further degenerates (owing to a rupture of the secondary cysts, and to their bursting into the cavity of the parent cysts) into a multilocular cell or network.

Every cyst is of course competent to represent a parent cyst in relation to its own ulterior cyst production.

The *second* category comprises cysts in which secondary cysts arise upon the internal surface of the parent cyst, and grow into its cavity. They are sessile upon a broad base, or more often upon a neck or pedicle; in which case they mostly represent pear- or wedge-shaped tumors. They often so luxuriate in number, and at the same time grow to such a size as nearly to fill a parent cyst of considerable magnitude. In rare instances, a solitary cyst of this kind so increases as singly to fill up the space of the parent cyst, causing the sac to consist, down to the base of the filial cyst, of two contiguous layers.

These secondary cysts become developed in the internal layer of the parietes of the parent cyst, and have a sheath derived from the internal membrane, from which the secondary cyst can, with care, be separated. In the pedunculated, pear-shaped cysts, it furnishes, in a state of involution, the pedicle into which the pouch, or wedge-like cyst, projects with a conical tapering end.

They are either simple or compound, according to one or other type. In their wall, namely, reside cysts of an ulterior, that is, a tertiary formation, which grow more or less outwardly or inwardly; the former acquiring a shallow-lobed, blackberry-shape, and appearing cellular within. The pear-shaped cysts commonly consist of several parallel pouches of various lengths. Along with these are found, on the inner surface of the parent cyst, in varying numbers, the smallest vesicles, just cognizable with the naked eye. In one instance these were found on the inner surface of an extensive ovarian cyst, mixed up with, for the most part, naked yellowish incrustations the size of a poppy- or a millet-seed.

The difference between these two types of the compound cyst is obviously not essential, but depends only upon the seat of development of the secondary cyst. Hence, the very common concurrence of the two types.

There also occur, on the inner surface of the cysts, both parent and secondary, ramified cauliflower excrescences, flattened, or fungoid, or pedunculated. These are scattered singly, or grouped together, or knotted in masses. Sometimes they luxuriate in and by the side of the said secondary cysts, to such an extent as to fill both these and the parent cyst, rupturing the latter, and, in the frequent cases of ovarian cyst, invading the peritoneal cavity. In the ruptured secondary cysts we often recognize their sheaths folded back, and reflected over the cauliflower vegetation.

Besides the variations alluded to, there is much that is worthy of note in and about these excrescences.

1. They consist of a very delicate membranaceous growth folded and rolled up in their pedicle, projecting about and especially above it in various ways, branching out into numerous villous and bulb-like processes, or into the semblance of a plaited frill. Upon the said processes are again seated delicate villous flocculi. They are highly vascular, and have a blood-loaded aspect.

2. Here and there they frequently carry, especially at the extremity of their branchlets, a just cognizable, poppy-seed-sized, limpid, or semi-opaque vesicle, or a hemp-seed-, or a pea- or bean-sized cyst.

3. More frequently still they bear upon their twigs solid, though soft, whitish, roundish, or, from mutual compression, indistinctly facetted corpuscles; or else tougher, white, opaque tubercula, mostly of the bigness of millet or hemp-seeds. Here the entire excrescence is commonly white, the small broad-based ones resembling delicate stellar horny warts, whilst the larger and more extensively clustered ones constitute an unyielding tumor, superficially stellate, studded with the aforesaid tubercula upon its peripheral villous structure, permeated throughout its cut surface by fibrous threads (the pedicles). In this tumor, the blood-vessels have become destroyed.

4. Besides the more extensive excrescences, smaller ones are commonly seen, resembling a nap of extremely delicate villi, or of finely pedunculated tubercula.

5. At the same time the internal investment of the cyst has, in expanded parts, often a very finely reticulated aspect; or it reveals very minute fissure-like grooves, not a few of which are surrounded by an elevated ridge-like brink. Out of these is here and there seen to rise a simple or branched excrescence. In other places, the internal layer of the cyst-wall is seen raised into a flattened vesicle, which, like the said grooves, often displays minute fissured openings. Internally is sometimes plainly discerned a convoluted mass of bulb-shaped excrescences, or else a very minute network.

With these are associated larger vesicles, rising into pedunculated wedge-shaped pouches, which contain a very delicate cancellated structure, and frequently exhibit roundish or angular chink-like openings, out of which delicate felt-like excrescences occasionally project. The size of the cysts varies greatly from that of a just cognizable vesicle to that of a sac of from 1 to 2 lines in diameter. The compound cysts may, of course, attain to a very considerable magnitude, a notable portion always appertaining to the parent cyst.

The free space of the cysts hitherto described, is commonly occupied by a serous synovia-like, or a thicker glutinous, or glutino-lardaceous, so termed, colloid moisture.

Examined under the microscope, the following additional light is thrown upon the above appearances.

The cyst-wall consists of densely-reticulated areolar tissue, the internal layer constituting an epithelium of cells or nuclei. In large cysts this is, for the most part, absent, and the internal layer generally presents a nucleated, structureless, or striated blastema, at the circumfe-

rence of which the oval nuclei are in the act of splitting into fibres, in the direction of their long axis. On examining a section of the internal layer of a cyst-wall, from a part furnished with vesicles (secondary cysts), we obtain a view like that presented by the cortical substance of a kidney affected with cyst-formation, to the consideration of which we shall shortly have to recur. The same nidus often contains concurrently incrustations, in some instances remarkable for their size and figure. On examination, the excrescence appears as a hollow growth, consisting of a transparent structureless membrane, studded with round or oval nuclei, often striated, especially at the pedicle, and breaking up into delicate fibrils, with numerous spheroid protuberances. These become developed into pouches, mostly bulb-shaped at the extremity, and by throwing out secondary protuberances and pouches, complete the branchlets and twigs of the excrescence. They may be invested with the epithelium of the cyst-wall, or even uninvested. They are furnished with conspicuous bloodvessels, which, running along the protuberances, describe extensive arches and anastomoses, and frequently become the seat of aneurismal dilatations, or the source of hemorrhagic effusion into the cysts. In their interior they contain nuclei in various numbers, and along with these, especially near the blind extremity of the branchlets, growths which turn out to be young cysts.

These young cysts dilate into those spoken of as cognizable with the naked eye.

The minutest excrescences appear as simple, smooth, or tuberous hollow bulbs. The internal layer of a cyst-wall, presenting the reticulated texture described (5), appears, when magnified, in the form of elongated, round, angular, distended meshes, through which the simple, smooth bulbs penetrate as they grow. The cancellated framework, contained within the described vesicles, consists of a hyaline, structureless membrane, studded with nuclei. It has unquestionably arisen out of the fusion of several bulbs.

The excrescence, as described at No. 3, arises through the development of areolar tissue out of a transparent amply nucleated blastema. In its cavity are lodged, sometimes in vast quantities, simple and laminated, semi-opaque, incrusted growths, from the size of an elementary granule to a diameter of $\frac{1}{2}5$th of a millimetre, the circumference, most common to incrusted cysts.

The excrescence simultaneously becomes fibrous, and shrivels, with condensation of fibrous parts, into the solid masses above described.

In these observations, two phenomena engross our attention, namely, the development of the secondary cyst and the hollow growths forming upon the internal wall of the cyst. We have made them the subject of an extended investigation, with a view to the solution of the double question as to the nature of elementary germ for cysts, both primary and secondary, and of its ulterior development,—and as to the import of the said hollow growths.

(a.) The cysts best adapted for the inquiry, are young, small, clustered cysts, just visible to the naked eye; others being probably present, still smaller, down to the germ itself, out of which they spring. The cysts so frequent in the kidneys, or on the broad ligaments, and on the

peritoneum of the tubes and ovaries, furnish ample materials for the purpose.

1. In the cortical substance of the kidneys, especially during the decline of Bright's disease, a luxuriating cyst-formation is not un-common.

In the dimpled depressions upon the surface of atrophied, gibbous kidneys, reside entire nests of parallel-clustered, just discernible, poppy-or millet-seed-sized vesicles, imbedded in a reddish-gray, or whitish nidus. Occasionally the kidney is found altogether degraded into an aggregate of various-sized cysts.

A small portion of such a nidus placed under the microscope, displays along with the débris of renal texture,—namely, uriniferous tubules and Malpighian tufts, in a state of collapse or involution; the former denuded of their epithelium, and here and there replete with fat molecules—a multitude of cysts invisible to the naked eye. The more marked have parietes, consisting of fibres beset with elongated oval nuclei, which, more particularly about the inner fibre layers, bend round towards the circumference of the cyst. These cysts are replete with granulated nuclei,—now and then with spherical or polyedrical cells, to which, in some few, is superadded a molecular mass, partially betraying by its brown coloration its character as pigment granules.

In some cases this occupies the centre of the cyst, where the nuclei become indistinct and disappear. In some cysts, the nuclei (or cells) are reduced to an epithelial formation investing the cyst. In others, again, even this is wanting, and the sterile cyst is entirely filled with a clear, or semi-opaque, viscid humor. They are of very various size, from a diameter of $\frac{1}{2}$ to $\frac{1}{20}$ of a millimetre, the former immediately preceding vesicles distinctly cognizable with the naked eye. Conjointly with these, are found cysts, which, with similar contents and parietes, consisting of a structureless transparent membrane, reside in an equally structureless stroma, interspersed with oval nuclei, and in progress of development into a fibrillation about to encircle the cyst.

We further discern, commonly within an aggregate of spherical, nucleus-like bodies, growths of various magnitude, down to that which only just surpasses the dimensions of the nucleus. These growths quite coincide with the aforesaid structureless vesicles. The smallest contain a clear moisture, or are faintly granular. In the larger ones, a central nucleus soon appears, joined by a second, a third, a fourth, and more, so as to fill the equably dilated vesicle. This description *comprises the history of the development of the cyst*, and may, under favorable cir-cumstances, be found exemplified in a single preparation. *It is obviously the nucleus that grows up into the cyst;* which, with reference to endogenous production, either generates brood nuclei or else proves sterile.

Besides the nuclei, there are seen smaller corpuscles of all sizes, from that of the nucleus down to that of the so-called elementary granule, and manifesting, in proportion to their magnitude, more and more of the character of the nucleus. It is, therefore, at once to be stated, that the *nucleus has arisen out of the elementary granule;* and this, through spontaneous germination,—not through the agglomeration of several.

Finally, we observe, in the preparation, concentrically stratified bodies, also, of different sizes, and consisting of incapsuled vesicles in varying number. These vesicles are themselves sterile, or the central vesicle may have its space occupied by one or more granulated nuclei. Sometimes it is itself represented by a nucleus. One or more of the external layers contain, in like manner, nuclei, oval in shape, and bent to a parallel with the layer. Again, the layers are in some cases slightly gibbous. Incrustations of these forms are also present,—nay, kidneys sometimes occur, in which the cortical substance, otherwise seemingly sound, is interspersed with them, looking like yellowish, transparent grains of sand.

These are the results to which I have referred, in describing the compound cystoid. A similar result is furnished by the inspection of a group of cysts *in the above-mentioned sexual attachments of the peritoneum.*

2. The renal preparation at first sight so much *resembles the texture of the thyroid gland, and more particularly the goîtred thyroid gland,* as to render it impossible to discriminate between the two. Simon has directed attention to this in vol. xxx. of the ' Transactions of the Medical and Chirurgical Society.' Not only is the normal gland-vesicle of the thyroid gland identical with a cyst of corresponding size, but the development of new gland-vesicles in a goître is identical with cyst development, and again the preternatural dilatation of the gland-vesicle—its so-called cyst-like degeneration—identical with a cyst outgrowing its microscopical proportions. Nay, the gland-vesicle betrays in its development the same anomalies as the cyst in its development as a sterile vesicle, or as a laminated cyst in its degeneration to a colloid sphere, and in its incrustation.

3. The same relations attach to *cyst-formation* in mucous membranes. In those of the stomach, the colon, the uterus, a morbid growth occurs, known by the term cell- or vesicle-polypus. It consists of an aggregation of from millet or hemp-seed to pea-sized cysts, broad-based, but mostly furnished with a neck, and commonly representing the head-like free extremity of a cylindrical prolongation of the mucous membrane. These cysts are developed in the texture of the mucous membrane, seldom exceed the aforesaid volume, but burst and evacuate their viscid, jelly-like contents upon the surface of the mucous membrane. Their fate, beyond this disruption and violence, I have been unable to ascertain. They probably give place to new ones. We may refer here to those bodies suspended by a pedicle of mucous membrane from the cervix uteri, and known as *ovula Nabothi.* These, though commonly received within the domain of physiology, in reality present a continuous cyst-formation, destroyed from time to time by disruption and evacuation. They occur, in like manner, on the mucous membrane of the renal pelves, and of the ureters.

But I have repeatedly observed millet, hemp, nay almost pea-sized cysts, in surpassing numbers in the mucous membrane of these urinary conduits. Some of them contained a flaky, inspissated, colloid moisture. In one instance, my attention was drawn to them, by the presence of little, roundish, naked, colloid pellets, in the urinary bladder.

Just as in physiological textures, so also in pathological parenchymata does cyst-formation occur. For example; in the textures of *sarcoma* and *carcinoma*,—giving rise to the family of *cysto-sarcomata* and *cysto-carcinomata*. Even the so-called *carcinoma alveolare* consists, mainly, in cyst-development.

Cancer-cyst varies in respect to size from the microscopic, to the circumference of the colossal cysts, in the compound cystoid. The alveoli of areolar cancer in particular—that is, the small cysts constituting alveolar-cancer,—and especially the peripheral-alveoli, grow into comprehensive cysts. The cancer-cyst contains a sero-albuminous fluid, a jelly-like (colloid) substance, and frequently cancer-parenchyma. It is not a rare thing to find—imbedded in a cancer, or independent, and remote from a heterologous growth, ascertained by its volume to be the primary seat of the cancer-production—tumors, obviously consisting of encysted-cancer parenchyma, or cancer-tubera, which, manifestly enveloped in an often stoutish fibrous capsule, are distinguishable at a glance from other uninvested accumulations of the cancerous substance. Within the encysted parenchyma, again, is sometimes lodged a smaller, filial cyst.

Upon this point and upon the development of the cancer-cyst microscopic inspection throws much light. The appropriate materials for examination are afforded more especially by cancer-masses, which, with or without the presence of voluminous cysts, exhibit to the unassisted eye, minute, limpid vesicles, or else an aciniform, glandular structure.

In such cancer-growths, besides the ordinary nucleated cells, often indeed distinguished by their eccentric forms, we discern:

1. Cells of notable diameter, up to $\frac{1}{30}$th of a millimetre, with a very large nucleus, dilated into a clear vesicle, which approximates to, if not touches, the cell-wall. In some of these bloated nuclei, a nucleus corpuscle has become developed into a second central nucleus, which, in its turn also, contains nucleus-corpuscle.

In certain cells we find two of these advanced globular or mutually flattened nuclei, as also several without a nucleus-corpuscle, or with one which, in like manner, expands into a nucleus. Other cells contain, along with a swollen nucleus, one or more ordinary granulated or transparent, spherical or oblong nuclei, besides.

2. *Divested nuclei*, which, like the cell-included nucleus (cell-nucleus), expand into larger, transparent, structureless vesicles. These—

(*a.*) Remain sterile cysts.

(*b.*) They give birth to numerous secondary nuclei, until the cyst is replete with these. Such cysts often entirely resemble the gland-cyst of the thyroid and supra-renal glands. Of these secondary nuclei, one or more occasionally grow into a vesicle, which remains sterile, or fills with brood-nuclei, or presents phenomena about to be described.

(*c.*) *A central nucleus-corpuscle appears in the vesicle and expands into a secondary nucleus.*—This nucleus, like the primitive one, dilates into a vesicle in which a nucleus formation of the third order takes place.

Out of the frequent repetition of this process originate growths concentrically laminated, or consisting of a series of endogenous vesicles,

which here again are distinguished by their proclivity to incrustation. Sometimes there are developed, in one of the secondary vesicles, more than a single nucleus—for example, two out of each, out of one only of which a laminated formation may become developed. In the latter case, ordinary brood-nuclei are generated in the other. The inner vesicle either remains sterile, or a central nucleus, or it may be several nuclei, engross its space. Here, again, there may be laminated structures, springing from extra-centrical nuclei.

All these structures are lodged within a parenchyma (differing in composition, and in the fecundity of its elements), of nuclei, cells, caudate-cells, fibres. These elements are so arranged as to inclose the said structures in a capsular fashion, the cells, and even the nuclei, lengthening into riband-like, caudate cells, and oblong nuclei, with a corresponding incurvation. (*Alveolar textural arrangement.*)

Still, the main condition for the growth of the vesicle is the presence of encircling fibres, and their appropriation to the fabric of a resisting fibrous cyst.

The simultaneous evolution of the brood-nuclei of the vesicle causes the cyst to become speedily furnished with a proper parenchyma corresponding with that which surrounds the cyst. In this a filial cyst may become developed.

The structureless vesicles alluded to offer many further points of interest:

(*a.*) There is, frequently, a marked difference in the contents both of the simple vesicles, and of the individual layers of the vesicles successively ingenerated. Thus, some appear clear and colorless, others of a reddish tint ; in others again, the contents are denser, pearly, or opaque, lightly granular. Some contain granules in various amount, which show themselves to be fat,—fatty conversion of the nuclei.

(*b.*) Of two intussuscepted vesicles the inner one is sometimes irregularly collapsed, wrinkled, or even pretty regularly indented. This probably results from a consecutive difference of density in the contents of the two, a condensation of the contents of the outer vesicle determining exosmotic effusion of the thinner contents of the inner vesicle.

(*c.*) Intussuscepted vesicles are, for the most part, sterile. Within their layers, however, are frequently impacted oblong, curved nuclei.

(*d.*) The layers are commonly smooth ; often, however, gibbous, wavy, and curled.

The development of these cysts out of the nucleus, through growth of the latter, is here demonstrable even in the naked nucleus; by growth of the celled nucleus, however, it is placed beyond all doubt. Here again, the elementary granule is cognizable, as the ultimate, fundamental form; the nucleus being obviously and simply developed by growth out of the so-called nucleolus, or elementary granule.

Difficulties might, however, still arise so long as that theory of cell-formation obtains which assigns to the cell a genesis and an import distinct from those of the nucleus. If there be parent cells, their resemblance with the expanding nuclei, both in form, and often in their relation to chemical agency, might render it no easy task to determine the precise nature of a vesicle, seeing that parent cells and parent nuclei are

met with concurrently. In a laminated structure, it would be peculiarly puzzling to have to decide, whether its external contour belonged to a cell, or to a nucleus-wall.

From what has been stated, however, the existence of a cancer-cyst certainly may be inferred, a cyst, namely, developed out of the elements constituting cancerous substance, and productive of cancer elements within itself. Not every cyst, however, concurrent with cancer, is necessarily of a cancerous nature, the malignant growth very possibly wearing but the character of an accidental complication.

In cysto-sarcoma the same relations obtain as to the [primitive] development of the cyst.

β. As regards the *excrescences occurring upon the inner surface of the cyst*, repeated observations have established the following facts:

1. Upon mucous membranes, and especially upon that of the urinary bladder, there occurs a cancerous growth which we have elsewhere termed *villous cancer*, as a structure pertaining to medullary carcinoma, and containing within a villo-membranous vascularized stroma, a medullary (encephaloid) cancer juice. Later investigations have led to results which induce me to reopen this subject.

It consists, as a walnut- or a fist-sized tumor, of a multitude of densely thronged excrescences, which, upon a cord-like, longer or shorter, pedicle unfold into delicate membranes, breaking up into numerous ramifications, and again into more and more tender branchlets, wholly overlaid with delicate villi. Many twigs have a grape-clustered appearance, their villi bearing poppy or millet-seed-like, clear or opaque, white vesicles. Larger cysts reside in the primary cotyledon and ramification. Many excrescences, again, represent hollow, shut, or at their free extremity, wide-mouthed pouches. In one instance, the entire growth consisted of polyedrical, at their free ends for the most part wide-mouthed, pouches, densely beset with villi at the brink of the aperture. The tumor is throughout surcharged with a whitish, creamy, medullary juice. It is frequently a more consistent medulla-like mass that fills up the cavities of the growth, which in this case acquires considerable density, and offers proportionate resistance.

The growth is in general highly vascular, and in its recent state, turgid, of a deep purple tint, and prone to hemorrhage. At the base, from whence the tumor commonly rises with a neck, we have found an extensive sinus of a venous kind, upon the inner surface of which are seen numerous pin-puncture and poppy-seed-sized orifices, leading to bloodvessels, which ascend within the pedicles of the excrescences, and accompany their ramifications.

In the vicinity of, or even remote from, the heterologous structure, are smaller groups, or solitary excrescences. These, when young, are very delicate, so as when under water to resemble a fine nap.

Microscopic Examination.—In the cream- or marrow-like juice that exudes on gentle pressure, are found variously-shaped cells, with one or several, in part turgid, vesicle-like nuclei, along with bare, middle-sized, and larger-sized nuclei, furnished with a considerable nucleus-corpuscle, of which one especially, was found large, and presenting internally a dull secondary nucleus contour. This juice resides in the before-mentioned

pouch-like chambers. The excrescences are externally clothed with an epithelial layer.

The membranous structure constituting the excrescence appears as a very delicate, transparent, structureless, here and there striated membrane, overstrewn with oblong nuclei, and breaking forth about the pedicle into slender wavy fibrils. It is invested with a simple layer of granulated nuclei for epithelium, which is, however, frequently wanting.

A *clustered twig* appears as a clavate, hollow structure upon a delicately fibred pedicle, young cysts, as structureless vesicles, occupying the interior of the protuberances. Here are seen two outlines, of which the outer one belongs to the protuberance, the inner one to the young cyst; elongated nuclei course along between the two. The cyst is replete with spherical nucleolated nuclei. A few cysts open towards the pedicle of the terminal bulb in which they are contained.

In some of these sacculi are besides found, in various numbers up to the point of repletion, fat-globules, some of largish dimensions. These lend to the cyst the white opaque aspect already referred to.

The larger millet-seed-sized vesicles, visible to the naked eye, contain a colorless, tenacious fluid in which the above-mentioned nuclei float.

A morsel of a membranous expansion of the excrescence appears, when magnified by 50 diameters, distinctly to consist of two layers, and is everywhere, but especially at the summit, overspread with numerous bulb-shaped protuberances, which themselves throw out secondary projections; whilst considerable bloodvessels ascend to all. In the inside are here and there seated groups of fat-globules.

Their bilaminated structure renders it more than probable that the layers, in consequence of the copious production and accumulation of the cancerous elements, separate into the pouches aforesaid, which, for the same reason, give way at this free extremity.

In 1842, a urinary bladder was shown to us by Hodgkin, at Guy's Hospital, upon the inner surface of which were seated numerous largish bulb-shaped cysts, filled with the excrescences referred to.

The extensive development of the cysts in a mucous membrane is in itself very remarkable; whether, with the excrescences, they be of a cancerous nature appears uncertain. Upon the *mucous membrane of the renal pelvis* we have seen, along with young cysts, some solitary, others grouped together in its parenchyma, awl-shaped and bulbous, smooth and villous, red, vascular excrescences. They were seated, in part singly, partly in collected groups, some bearing a just discernible transparent globule at their free extremity, which microscopic examination showed to be a young cyst.

2. Upon *serous membranes*, and the peritoneum in particular, we have, in connection with luxuriating medullary cystocarcinoma of the ovaries, met with medullary vegetations which, judging from their appearance and the arrangement of their vessels, would seem to belong to this category.

On the other hand, an examination of the preparations in the Pathological Museum of Vienna has taught us that the dentritic vegetations which often luxuriate upon synovial membranes, and in the capsule of

the knee-joint so numerously that it appears invested with them as with a felt, really appertain to this class. In the interior of the hollow growths which constitute them, a development of areolar tissue takes place, just as in the cysts of the vascular plexus, of which we shall hereafter speak, until they at length become replete with it. The largish terminal bulbs of their stems and branches are frequently so flattened as to resemble linseed or melon seeds (Majo).

The entire excrescences with these numerously seated upon them, and it may be in imbricated order, acquire the aspect of foliage. In one instance we found them to contain fat-cells, which explains what Johannes Müller meant by *lipoma arborescens*. In fine, we doubt not that they offer the original nidus for the production of those circular, smooth, or knotted, facetted cartilage or bone-plates, which occupy the inner surfaces of synovial sacs, often attain to a considerable volume, and, by spontaneous detachment, become free bodies within the capsule.

3. The encysted parenchyma is often contained free within the cyst space; sometimes, however, there is present a meshwork, issuing from the inner wall of the cyst, the spaces of which are filled up with the medullary mass. This framework consists of a transparent striated blastema, pervaded by numerous spherical and oblong nuclei, and breaking up into fibrils. Simple hollow bulbs shoot up from its trellises, and into its chamberlets. Respecting the development of this stroma, two theories might be propounded:

(*a.*) It might result from a continuous separation of the internal layer of the cyst-wall.

(*b.*) It is, however, far more probable that, like the framework of alveolar gelatinous cancer, or of the encysted new growth of thyroid gland parenchyma in goître, it results from the blending of the excrescences concentrically growing from the inner surface of the cyst.

Occasionally, sharply-defined, spheroid cancer tumors occur in which no cyst-wall is discoverable, but yet, in their interior, a stroma of this kind. It is very probable that these tumors were previously encysted cancer-masses, from which the cyst has, owing to a total disruption with excessive production of those excrescences, disappeared or become part and parcel of the stroma.

The repeated examination of so-called *alveolar cancer* (gelatine cancer in alveolar form) offers very interesting results, corroborative of the endogenous multiplication of its cysts. Besides the exogenous augmentation, there occurs likewise an endogenous one, the medium of which is offered by the often-mentioned excrescences; and in this process these become converted into the framework, in the alveoli of which the small cysts are subsequently lodged. From the inner surface of a thick-walled follicle, or alveolus, numerous simple bulbous pouches shoot up into it, so as to penetrate it. Another, contiguous, is replete with a young delicate alveolar parenchyma. In the interior of those hollow bulbs is seen one, or a pair of cysts. In such prolific cysts the development of the fibre-layer for the young cysts may often be seen proceeding from the base of the excrescences, near the inner wall of the parent follicle. Gluge has seen these bulbous pouches in a cancer of the rectum, and figured them. Owing, however, to his treatment of the pre-

paration, he recognized neither their relation to the alveoli nor their character in general. He regards them as altered and hypertrophied muciparous glands.

Thus, encysted alveolar cancer is accounted for and explained in the same manner as medullary carcinoma.

4. Upon the internal parietes of *cysto-sarcomata* there are known to occur (in the so-called *cysto-sarcoma proliferum*) warty, foliaceous, bulbous vegetations, as also pedunculate cysts. The former often attain to a considerable size, so as to fill up the space of the cyst, presenting a flesh-like aspect. [For an explanation of these phenomena, the reader is referred to an appendix to the separate section on *cysto-sarcoma*.]

5. In goître, besides others, there occur cysts filled with thyroid gland parenchyma of accessory growth. We have often the opportunity of witnessing the process of this endogenous production in its incipient stage. From the inner parietes of the cyst arise delicate, transparent, protuberant, vascularized excrescences, in the interior of which a new creation of gland-vesicles takes place.

6. Finally, in the domain of physiology, we encounter, upon the vascular plexuses in the lateral ventricles of the brain, a formation which, as comparative microscopical investigations teach us, fully coincides with the excrescences still before us. Upon the lateral plexuses of vessels are seated great multitudes of delicate, red, vascularized villi. These consist, beneath an epithelial covering, of a transparent, multifariously projecting, fretted, hollow growth, in the protuberances of which run arched bloodvessels of no inconsiderable size. Here we seldom fail, especially upon subjects advanced in years, to find numerous little cystlets, for the most part lodged in those protuberances. Generally speaking they do not outgrow a certain measure, a diameter of from $\frac{1}{25}$th of a millimetre up to a cyst discernible by the naked eye, and differing from what are usually called cysts of the vascular plexus. By dint of a repeated development of central nuclei, the majority are wrought into concentrically laminated growths, and undergo incrustation.

The very common so-called *cysts of the vascular plexus* are not genuine cysts. Numerous examinations have convinced us of their being dilatations of the hollow growth which constitutes the villus of the bloodvessel plexus. This dilatation pre-eminently affects the villi adjacent to the tortuous bloodvessels upon the convexity of the arch described by the vascular plexus. Accordingly, they are clusters of gibbous and indented vesicles, separated by the indentations into several loculi. Little remnants of villi are often to be detected upon them. We have frequently observed the dilatation of the villus at its commencement. In this manner, the so-called cysts of the vascular plexus answer to the pouches into which the excrescences constituting villous cancer widen. Besides other matters, they contain areolar tissue, sometimes to perfect repletion, so that, in this respect, they may be likened to the excrescences upon the internal membrane of the cysts, and especially to those which, through the development of areolar tissue, have become internally parenchymatous; as, for instance, in the cyst of sarcoma.

The contents of what are called *cysts of the vascular plexus* answer, in

all essential points, to the contents of cysts proper. They consist in a watery albuminous fluid, which, with a certain amount and quality of its accompanying organic elements, becomes turbid, thickish whey-like, and in great measure supplanted by the development of areolar tissue.

These elements are—

(a.) Minute, $\frac{1}{800}$ millimetre-sized, elementary granules, free, or numerously held together by a viscid interstitial substance.

(b.) Larger, up to nucleus-sized, spheroid, occasionally somewhat oblong vesicles, which speedily increase to $\frac{1}{25}$th of a millimetre. There are, besides, ordinary, granular, spherical, and oblong nuclei and granules.

(c.) The larger vesicles are simple, or successively enshrined. Some exhibit a double outline, that of the inner vesicle often lying so close to the outer one as to be easily overlooked. In other cases, the two are widely parted, the inner one being slightly wrinkled or curled. In others, again, there is, between the two contours, at some one spot, a space defined by a single outline, which sometimes resembles a shallow section of a sphere, and is particularly marked where the compressed contents of the external cyst are granular, and therefore contrast with the limpid contents of the inner vesicle. In some instances the inner vesicle is small, and either central to the outer one, or resting upon its wall. The latter kind probably engenders the form just adverted to.

Other vesicles, again, consist of three or of four, the inner, secondary, and tertiary ones being by turns central and extra-centrical. This species of complex and involved sheathing is met with in very small ($\frac{1}{266}$th millimetre-sized) vesicles. In some of the simple vesicles is found, —in place of a wall-inclined, secondary, spherical vesicle,—an oblong, wall-attached nucleus.

(d.) With these are associated many-layered, smooth, or slightly gibbous cysts, between the lamellæ of which oblong nuclei are often found inserted. The central vesicle occasionally holds a multitude of the most various primary and secondary forms,—elementary granules (nucleoli); spherical, oblong nuclei ; simple and compound vesicles, and incrustations of vesicles. These laminated growths, for the most part, undergo an incrustation proceeding from the central layers.

(e.) We also find the cysts of the vascular plexus to contain commonly in the shape of a *mucus-like* substance, deposited, as it were, from the fluid, and mostly infiltrated with fine sand-granules, a transparent blastema pervaded with round and oblong nuclei. This gradually forms into areolar tissue, displaces the moisture, and ultimately fills up the entire space of the cyst, in the meshes of which the aforesaid forms all lie imbedded. Within the structureless blastema we perceive the oblong nuclei bent in accommodating curves around, and closely attached to, the vesicles (*alveolar textural arrangement*). Such cysts gradually shrivel around these their contents, and finally become extinct.

The aforesaid cysts are very delicate, commonly clear and transparent; some refract the light with a whitish tone, their contents appearing somewhat denser; in others, the contents are of a reddish shade; in

others, again, finely granular, as though coagulated; in others, lastly, the contents consist of a multitude of sharply-defined granules. To this class belong, more especially—

(*f.*) Globular bodies, in which the outline of a cyst-wall is wanting, and which represent lightly granulated spheres.

(*g.*) Lastly, the vascular plexus cysts often contain a whitish, chalky fluid. This consists almost entirely of fat-globules, and gradually thickens into a lardaceo-cretaceous pap, around which the cyst speedily shrivels, and perishes.

The vesicles and granulated spheres, the incrustations, the areolar tissue developments and the fat-globules, render the contents of the vascular plexus-cyst more or less opaque.

γ. Finally, we have, in the well-founded expectation of throwing additional light upon cyst-development, examined their fluid contents. For this purpose we have found the contents of small (young) cysts, those, for example, which occur upon the broad ligaments of the womb, or within the cortical substance of the kidney, especially serviceable. They include a multiplicity of elementary forms, essentially identical with what are observed in the vascular plexus cyst. Nor is their occurrence limited to the cyst itself.

The fluid, semi-fluid contents of the sac, consist in an albumen-holding humor, which possibly presents various phases, the chief one being, however, that in which it constitutes colloid. In it are contained those elementary forms which are here fraught with peculiar interest. It is here adverted to irrespectively of what was before stated concerning its parenchymatous contents, as well as of whatsoever changes hemorrhage or exudation may give rise to within the cyst.

Besides the débris of an epithelial layer generally composed of granulated, nucleolated nuclei, there are found:

(*a.*) Similar free nuclei, some holding two, three, or four nucleoli, some visibly exceeding the usual size of nuclei. Along with them here and there, a form of which it is problematic whether it be a nucleated cell, or a full-grown nucleus, with a nucleolus.

(*b.*) Granules, the larger of which being $\frac{1}{700}$th of a millimetre in diameter, are obviously vesicles.

(*c.*) To both are associated vesicles and cysts, which grow from the size of the aforesaid granules to $\frac{1}{25}$th millimetre, and as will be seen, beyond this.

These cysts offer many points of interest:

(*a.*) They are simple, or else compound, incased within and within. As regards the latter, we meet in the first place with simple vesicles, within which a central granule or nucleus-corpuscle has become developed. This progressively enlarges into a cyst, in which the same process may be repeated. In a cyst are often contained two, three, four, and more secondary granules, nuclei, vesicles, in which the process of ingeneration is still further repeated.

(*β.*) Their shape is commonly spherical, but often flattened by mutual compression. Some, owing perhaps to the inspissation of their contents are wrinkled, bent inwards, more or less regularly indented. This applies to the simple cyst equally with the compound, affecting in the case of

the latter all the involved cysts in various degrees, or it may be only one, and that the innermost.

Some frequently throw out various projections, prolonged into cylindrical processes, which in turn display ulterior promontories, or it may be inlets. This applies both to the simple cyst, and also to the compound, affecting all the layers in unison. Occasionally, the projections are obviously determined by endogenous development out of multiplex nuclei and vesicles.

7. Their contents present marked differences :

The contents of simple and compound cysts properly consist of a viscid fluid, in some cases translucent and colorless, in others, of a reddish tint. In compound cysts, these two conditions often alternate one with the other.

Some contain in and along with the said fluid, granules in various numbers, even to thorough repletion ; some, together with these, clear, reddish-shaded vesicles, the size of a nucleus. Others contain granulated, spherical, oval nuclei.

The fluid contents of certain cysts appear denser, less transparent, opalescent ; of others, still denser, dully transparent, presenting at the same time a marked cloudiness. In others, again, the density is still further marked, and the cloud more sharply defined.

This cloud results from a parting of the contents into spherical corpuscles, and flaky pellets of various size. In a given cyst it seems tolerably uniform, or else it consists in a radius-like fissuring from the periphery towards the centre of the cyst. In compound cysts, the separation differs in degree and relative amount in the individual cysts, being often more developed, either in the outer or in the inner cysts, and in one or other not present at all.

The fissuring presents much that merits attention. In simple vesicles or cysts, it extends from the periphery towards the centre, where the radii or the points of the wedges of substance, bordered by the fissures, converge. In compound cysts, a fissure present in the external vesicle, borders either upon the contour of a nucleolus, of a nucleus, or of the secondary cyst, in which, if there be a fissuring, it is independently constituted. Or again, the fissure-lines of the cysts correspond with one another—in other words, the fissure affecting the external cyst is prolonged through the second, third, fourth inner cysts, &c., with a radius common to all. This is rendered possible by the metamorphoses of the cyst-wall about to be explained.

With the condensation of its contents, the wall of the cyst gradually disappears, seemingly blending with the contents to a uniform mass. As in the case of the so-called cysts of the vascular plexus, the entire cyst is transformed into a dull, opalescent, resilient, simple or compound, colloid globule, which splits under the covering glass plate,—a spherical, oval, cylindrical, or nodulated colloid mass. To this category, doubtless belong the unexplained bodies seen by Kohlrausch in a renal cyst. (Vogel, Path. Anat.)

This relation of the cyst-wall further determines the contents, reduced, after the completed process of separation, to an aggregate of stellate flocculi, which break up into roundish, opalescent fragments of various

size. There are always present globular débris from which numerous fragments of this kind have become separated.

This relation of the cyst-wall occasions the breaking up of the already fissured contents, either spontaneously, or from pressure, into stratiform, or wedge-like fragments, as is particularly frequent in incrusted specimens. The same cause produces the linear disruption, through pressure of the smooth compound colloid sphere, athwart many of its strata. These formations, together with the remaining amorphous colloid contents of the cyst, are sometimes tinged of a brownish or of a yellow hue, from imbibed pigment.

An ulterior change in these formations, which should here be noticed, is their incrustation with phosphate and carbonate of lime. It commonly affects the compound laminated cysts, but not unfrequently, the simple ones also; the comprehensive, equally with the small ones; the smooth, equally with the gibbous. It invariably emanates in the simple cysts from the centre, in the compound, from the central layer, whether this consist of a simple nucleus, or a group of nuclei in a vesicular nucleus-development. The secondary cysts lying side by side within a simple or a compound cyst, become incrusted independently of each other, and also of the parent cyst. The cysts affected with incrustation are those whose contents have undergone the condensation before referred to.

Lastly, many cysts contain granules and globules, which are shown to be of a fatty nature, the cyst resembling in some sort a colossal granule-cell.

(d.) There is in the primary and secondary formations within the cysts hitherto spoken of, frequently a colorless hyaline, or colored colloid substance, in the shape of roundish, oval, facetted, poppy-seed, millet-seed, or lentil-sized pellets and flakes, and of larger misshapen masses. It is uniform in character with the opalescent, self-separating contents of the aforesaid microscopic cysts.

The presence of all the semi-transparent and opaque formations renders the cyst-contents whitish, or turbidly yellow. This portion of the cyst's contents is frequently separated in the form of a deposit.

With this I conclude the catalogue of facts, relating to the cyst. I shall now proceed to recapitulate these facts, and endeavor to elicit from them such deductions as they appear to warrant and uphold.

1. The cyst is, with respect to its organization and secretory function, an independent hollow growth, essentially based upon a substantive element.

2. At the characteristic turning-point, between a primary (embryonic), and a secondary phase, overstepping the microscopic scale, the cyst consists of a structureless vesicle of from $\frac{1}{25}$th to $\frac{1}{10}$th of a millimetre in diameter, and an encircling fibrous layer, maintaining various grades of development. To these is added, as endogenous production, a nucleus or cell-formation, limited to an epithelial stamp. The cyst here completely resembles the glandular vesicle of the thyroid gland, and of the supra-renal capsules. The encircling fibrous layer furnishes the *alveolus* for the reception of the structureless cyst.

3. The elementary germ of the structureless vesicle, resides in the

nucleus,—nay, inasmuch as the nucleus is obviously generated out of an elementary granule, it resides in the elementary granule itself. The latter grows by intussusception into the nucleus, and the nucleus at once in the same wise into the structureless vesicle. The nucleus arising out of the elementary granule either retains the character of the latter, as a smooth, polished, sharply-pencilled vesicle, or acquires the well-known granulated character. It is obviously the former, in particular, that becomes developed into the structureless cyst, even the granulated nucleus, however, enters upon this development, its contents clearing up during the process, but resuming the granulated character afterwards.

This history of cyst-development is essentially corroborated by the expansion of the cell-nuclei, of so frequent, although by no means exclusive, occurrence in *cancer-cells;* an expansion first pointed out with precision by Virchow, but which, owing to the identity of development of the normal gland vesicle, and of the cyst, cannot be regarded as heteroplastic. It consists in the development of the cell-nucleus into a comprehensive cyst, identical with that evolved out of the naked nucleus.

This corroboration is rendered complete by the fact, that, in the inflated cell-nucleus, an elementary granule present as a nucleolus, expands into a nucleus, generates within itself another neucleolus, and forthwith becomes a second cyst. This in my opinion affords important evidence of the vesicular nature of the nucleus, and of its evolution out of the elementary granule, through simple intussusception-growth which Reinhardt has shown to be a property of the chyle-, the lymph-, and the pus-corpuscles. (*See Virchow's Archiv.*, vol. 1, fas. 3.)

4. To the cyst in its primitive condition, as a structureless cyst, there accedes from without, and blends with it, a more or less marked fibrous texture. The cyst in this secondary condition, consists of a wall of a definite texture, with an internal lining of epithelium, and is at once endowed with an enormous capacity of growth.

The structureless cyst is developed in a consolidated, structureless blastema, commonly studded with spherical and oblong nuclei, or else in a nidus of caudate and other cells. In the former case, the cyst is speedily surrounded with a fibrous formation following the course of the encircling oblong nuclei, the cells contiguous to the cyst assuming an elongated, ribbon-like, caudate shape, and arraying themselves in parallel order around it. A remarkable tendency to enter upon this relation to the cyst is shown by the caudate cells, which constitute so many cancerous tumors. They first associate themselves one by one to very small, young, vesicles, and shortly overlay them in quite disproportionate numbers. Even oblong nuclei will fasten upon young vesicles of the kind.

5. This arrangement of the elements of a texture, brought about by the presence of young cysts, and consisting in an essential portion of those elements uniting to form capsules, and alveoli for the reception of the cysts, I have termed *alveolar textural arrangement,* or the *alveolar textural type,* and assigned to it a range extending far beyond the limits of a special heterologous formation. This establishes the distinction

between the alveolar textural arrangement and many similar, but differently engendered, meshworks, cavernous structures, and the like.

6. The cyst, in its primitive state as a structureless, vesicle, and also in its development, fully corresponds with the simple gland vesicle—for example, *the thyroid gland, and its development*, as seen more especially in hypertrophy of the thyroid gland. Nay, the same anomalies of development, consisting in arrest and involution, are common to both. On the other side, the insensible progress of the gland-vesicle, when imbedded in its fibrous alveolus, from its normal standard to the morbid condition of a cyst, constitutes a process completely one with the secondary stage of cyst development.

7. Cysts form singly, or else collectively, in greater, often in redundant number. New cysts often arise within the fibrous wall of a parent cyst. There occurs also an endogenous multiplication of the cysts, new ones being developed in the fluid or parenchymatous contents of a cyst. In the former case, they do not in their development overstep the primitive condition, for lack of the adventitious element requisite to consolidate and advance the structureless vesicle into the true cyst.

8. The cyst, as appears under the circumstances discussed in § 6, possesses, under the same form, a different import. This it reveals more especially in the character of the textural elements frequently engendered upon its internal wall through the medium of the excrescences. These repeat, now a normal, now a heterologous parenchyma—for example, that of carcinoma. We are here reminded of Hodgskin's idea, which has, from time to time, been much too inconsiderately and hastily condemned.

9. Cysts are for the most part abiding growths, which often attain to a monstrous circumference. There are, however, cysts which never, or very rarely, exceed a certain volume, about that of a grain of millet, or of a pea, at which point they burst, void their glassy, mucoid, colloid contents, perish, and are substituted by fresh ones. *Exploding cysts*, to which belong the cysts constituting the so-called vesicular polypi, the ovula Nabothi. The cyst in its primitive state as a structureless vesicle appears to burst, in like manner, and eject the brood elements it contained. At all events, open vesicles now and then occur which do not appear to have become so by external means.

10. On the inner surface of the cysts are found *simple, bulbous or dendritic excrescences*, which represent a bulb-shaped, pouch-like, or a variously projecting, hollow-growth, branching out into secondary and tertiary pouches, and consisting in a hyaline, structureless membrane, studded with spherical and oval nuclei.

These bodies shoot from the inner surface of the larger cysts, isolated or collected in groups, sometimes from the innermost, sometimes, naked, from a deeper layer, and through slight fissures, or more spacious gaps, formed by the cyst-wall giving way. Or, coming from the deeper layer, they raise the internal structureless or striated lamina of the cyst-wall into a vesicle, which they afterwards perforate, and with which they coalesce, becoming blended together with them into a meshwork. In the smaller cysts they raise up the epithelium, and retain it as an investment.

They contain an albuminous fluid, or one possessing the germs both of physiological texture, and of heterologous parenchyma. Through its accumulation they become changed into protuberant or pouch-like sacs, which last are often patent at their free extremities. There is often developed within them a fibrous texture which imparts to them the character of considerable, fleshy, shallow-lobed (condyloma-like) tumors, as in the cyst of cysto-sarcoma; or they shrivel into fibroid growths and perish. Above all they frequently engender, in the terminal bulbs of their branches, young cysts, thus mediating the endogenous production of secondary cysts.

They frequently enter upon the final transformation into a stroma of fibrous texture, which receives into a meshwork or into alveoli the elements of parenchymata of various kinds.

They occur not only in cysts, but also upon serous and synovial membranes, upon mucous membranes, growing, in all cases, into the respective cavities. Their development frequently out of deep fissures in the cyst-wall, renders it likely that they spring at some depth out of heterologous parenchymata, and ultimately penetrate into larger spaces formed by the yielding of textures.

They appear everywhere as *germ-nidi, and as carriers of certain textural elements*. In the cyst itself their tendency is to fill up space by determining the production of physiological and pathological parenchymata, but in particular the endogenous multiplication of the cyst itself. In the vascular plexus, and in the so-called Haversian glands, they occur as physiological growths. They have sometimes the characters of a benign, sometimes of a malignant new growth.

The correlation between the chorion-villi and the cyst-formation occurring within them in the shape of *acephalo-cystis racemosa of Laennec*, and between the subject under discussion, is hardly to be doubted. I have, however, failed, of late, to obtain fresh materials for investigating this point.

The cyst, in its primitive state, is subject to various anomalies productive of arrest of its development,—of involution of the sac. These anomalies consist essentially in changes in the contents of the young cyst; it is therefore desirable, in the first place, to point out what is most remarkable in the contents of the cyst generally.

The first and most marked phenomenon in point, is the presence of delicate, diaphanous, for the most part simple, but also nucleated vesicles, the one kind including reddish glistening, the other kind, colorless limpid contents. In growths consisting of several cysts, encased one within the other (expanded nuclei), it is common for one or the other to include reddish contents, whilst the remainder exhibit a colorless, clear, or slightly opalescent fluid. Occasionally these different contents alternate several times. These vesicles vary from the size of a nucleolus, or a nucleus, to that of a vesicle $\frac{1}{25}$th of a millimetre in diameter, or more. The small ones occur in all physiological and pathological fluids containing plasma;—in the blood, in the humor Morgagni, in exudates, in the juice of various heterologous growths, in the grayish blastema supplying the place of atrophied nerve-medulla in the brain and spinal

tube. The larger ones occur more particularly in conjunction with cyst-growths, and as forming part of the contents of cysts.

The parietes of the vesicle, whatever may be the nature of the contents, are uniform with the sheath of the nucleus. They resist the influence of acetic acid, or else the latter occasions thickening of the membrane, and a sharper contour of the vesicle. An examination of the contents is essentially facilitated by the following occurrence: In the above summarily mentioned fluids, there probably is always, freely suspended, a reddish glistening fluid or viscid substance, both in little globules, and in larger masses, moulded into various forms, and, where the current is impeded, divided into smaller portions. This substance is rapidly dissolved by acetic acid. It forms the contents of nuclei, and also of cells,—very commonly of cells the recipients of the brood-nuclei of the thyroid gland vesicle, the brood-nuclei of young cysts,—sometimes the contents of pus-cells, and of young cells generally. Here, again, it is, together with the very delicate cell-wall, soluble in acetic acid. In like form we often observe, especially in the contents of cysts, a colorless, viscid substance, equally soluble in acetic acid, and probably identical with the ordinary colorless cell-contents.

These two substances are, to all appearance, unimportant modifications of the same material. Colorless elementary granules and nuclei, after evaporation of their surrounding watery medium, when placed under the influence of liquor potassæ, swell, and assume a reddish tint ; we might, therefore, fancy that a difference in density determined the modification, the reddish gleam being simply due to a diminution of density.

The colorless (denser) contents are coagulated by the addition of acetic acid, becoming still denser, bereft of transparency, opalescent.

Akin to this change is that of conversion into a colloid substance.

Occasionally a fatty transition is suffered.

Let us now proceed to pass in review those phases of the cyst, as a structureless vesicle, which we have set down as anomalies of its development.

(*a.*) In the first place, numerous cysts, like the brood-nuclei and cells they frequently contain, break up, in their primitive state, as structureless vesicles. This is occasionally preceded by a dehiscence and emptying of the vesicle. In other cases, the contents of the cyst are first converted into fat, a transformation of the cyst to a growth resembling the granule-cell.

(*b.*) Cysts developed within a fluid [the fluid contents of a cyst], for lack of attachments, and of certain textural elements accruing to the structureless membrane from without, do not overstep the primitive condition. They succumb to the processes of involution already described, or enter upon changes hereafter to be explained.

(*c.*) A remarkable arrest in the cyst's growth attaches to the *endogenous* development of *secondary* and *tertiary vesicles*, resulting in those laminated cyst-growths destined to undergo early incrustation. The lamination may be restricted to a system of many-sheathed or encased vesicles, developed out of either central or extra-centrical, parietal nuclei. Or else several distinct laminated systems occur within a com-

mon peripheral one, several nuclei, simultaneously or independently developed within a secondary or tertiary vesicle, having expanded into vesicles, and these, again, generated their own proper central or extra-centrical nuclei. This explains the frequent deviations from the spherical or oval forms. The size of these growths varies greatly. We may have nuclei in which a nucleus-corpuscle is inflated to a vesicle simply bordering upon the contour of the nucleus, or, again, of $\frac{1}{5}$th of a millimetre in diameter.

(*d.*) Another arrest of the development of a cyst, fraught with its eventual destruction, consists in the conversion of its contents into colloid. The cyst becomes reduced to a sheathless colloid mass, becomes fissured or furrowed, and so broken up into fragments. In laminated vesicles, this transformation may affect all the vesicles uniformly or unequally, often a single, and that generally the central one, which, in this case, generates no further nucleus, so that all ulterior lamination ceases.

The consolidation to colloid affects the contents, not alone of the cyst, but also of its basis, the nucleus, and the elementary granule of nucleolus. This we observe alike where cysts are involved in the said conversion, and also under circumstances where no cyst-development takes place beyond a slight extra-normal inflation of the nucleus. Such transformed elementary granules and nuclei occur in medullary cancers.

(*e.*) Next akin to colloid conversion is the *incrustation of the cyst*, and of its bases (the nucleus and elementary granule) with phosphate and carbonate of lime. It affects both simple and laminated cysts. These incrusted growths vary from the bigness of a nucleolus and a nucleus, to $\frac{1}{5}$th, or even $\frac{1}{2}$ of a millimetre in diameter. They are, however, most commonly of about $\frac{1}{25}$th to $\frac{1}{20}$th of a millimetre. They are identical in form with the simple and laminated cysts, namely, spherical, oval, smooth or nodulated, lobulated, spindle-shaped, or cylindrical, occasionally hourglass-shaped, trefoil-shaped, facetted.

On compression, they frequently split up into regular flaky or wedge-like sphere sections, a circumstance connected, no doubt, with the capabilities of colloid substances to split asunder according to determinate radial systems. At other times they break up into smaller roundish stellæ, or into wedge-shaped fragments.

In laminated cysts the incrustation is wont to commence with the innermost layers, where the colloid consolidation of the cyst's contents begins. From hence it proceeds to the external layers. Many incrustations are seen in which the outer layer is as yet free, the incrustation, as seen from above, appearing to surround it as with a light fringe. In vesicles with several collateral filial vesicles, the latter become incrusted—beginning in like manner with the inner layer—whilst the parent cyst and its layers remain free for some time longer.

It occurs originally either in molecular or in crystalline form, the completed incrustations displaying, now a stellate, now a crystalline, stratiform aggregation.

To sum up, it would appear that it is not the *cell* out of which the cyst becomes developed, but the *nucleus*, and that this latter again is evolved, through endogenous growth, out of the nucleolus or elementary granule.

Lastly, that the structureless cyst-wall is not, as we once supposed, an independent textural development.

12. SARCOMA AND CARCINOMA.

Kindred new growths, important from their frequency no less than from the question arising, in every concrete case, as to their innocency or malignancy. They constitute an extensive group, comprising most of the so-called heteroplases. They occur, now as independent tumors, which usurp the place of normal textures, now as throwing out a multiplicity of roots or branches through the textures, interlacing with their elements, destroying these, and assimilating them to their own proper substance.

Solidified, and more especially fluid blastemata enter very largely into their composition, both becoming developed after their own manner into the elements described under the heading, "Metamorphosis of Blastema." They become wrought into a stroma, various in extent and in design, which forms the receptacle for fluid blastema and its elements. Along with the fibrous stroma developed out of solidified blastemata, massive fibrous textures, evolved out of cells and nuclei, are derived also from fluid blastemata. Lastly, the structureless parietes of parent-cells, here so numerous, enter into an immediate fibrillation.

The ulterior development of the parent-cell determines that very common texture, the alveolar, as also the resemblance with certain gland-textures; for example, the salivary glands, the thyroid gland, the cortical substance of the kidneys, &c., resemblances which formerly and even recently gave rise to such designations as "sarcoma pancreaticum," &c. When these heterologous formations affect osseous structures, they often determine the extensive new growth of a texture simulating bone.

Their chemical composition involves, besides the gluten of the fibrous elements, several other gluten-like (pyin-like) substances, along with albumen, casein, and fats.

In size they vary from that of a just cognizable new growth to that of a human head, and upwards.

In form they occur as spherical, roundish, knotty, lobulated, stellate, irregular, ramified tumors. The form of infiltration belongs more especially to cancers.

Of consistency we meet with every gradation, from the fluid and gelatinous, to the consolidated, fibro-cartilaginous.

Bloodvessels enter into the fabric of all these new growths, although to a very different amount; some being, relatively to their volume, sparingly vascularized, whilst others are distinguished by their redundant vascularity. In those of the latter kind, the collapse of the bloodvessels after death occasions a marked change in color, consistence, and volume.

These tumors have a pathology of their own, being especially subject to hyperæmia, hemorrhage, and inflammation with its sequelæ, more especially ulceration and necrosis.

Many undergo a process of involution and extinction, either spontaneous, or evoked by preliminary disease, especially by inflammation.

They originate at all periods of extra-uterine life, and are occasionally found of considerable size in the new-born infant.

Upon the whole, no texture or organ is exempt from them. There is, however, something peculiar and remarkable in the affinity evinced by certain types for particular organs.

We would distinguish these new growths, as shadowed forth in the heading, into two series, the one comprising *innocent*, the other *malignant* formations, for the grounds of which distinction we must refer the reader to the section on " Organized New Growths."

We have selected the term *sarcoma* to designate the benign growths, not because of any especial analogy with muscle-flesh, but in order to fix and define a name familiarized by long usage, and also by no little abuse. The malignant we shall leave in possession of their ancient characteristic appellation *cancer,—carcinoma*.

To catalogue the species of these new growths in a strictly philosophical order, is not feasible at this day. We shall best respond to the requirements of practice by determining the species upon the grounds of predominant structural and chemical relations, a due regard being had to the general habitudes of a growth. The varieties, numerous in the instance of certain species, are for the most part concerned with elementary texture, by which the general habitudes are not essentially influenced.

SARCOMATA.

These, as already stated, represent benign new growths. They are always purely local affections, and therefore exist almost always as a solitary growth. They are most commonly due to mechanical influences; accordingly their seat is generally in organs or parts near the surface, and obnoxious to such influences. They are curable by complete extirpation; that is they do not recur at the same spot, and still less do they multiply in other localities.

They generally constitute circumscribed, spherical, stellate, and clavate, superficially lobulated tumors. Often enough they ramify throughout the texture, nestling and luxuriating through the elementary parts, so that these perish, degenerating, so to say, into the heterologous growth.

They often increase to a very considerable volume, and this within a brief period.

They affect the areolar tissue, the fibrous membranes,—especially the submucous,—the muscles, inter-muscular tissue, and submucous muscular textures (uterus), the bones (osteo-sarcoma), particularly the facial bones, glandular organs, the mammary, the parotid glands, even the testicle; and, in rare instances, the brain.

The osteo-sarcoma is often inclosed within a skeleton sheath formed by the distended bone, which eventually becomes perforated. In rare instances a new growth of bone enters, in the shape of an inner skeleton or framework, into its composition.

Generally speaking, sarcomata are more frequent than carcinomata in the early periods of life—those of childhood and of boyhood.

They seldom lapse into a process of ichorous ulceration spontaneously,

although frequently through inflammation brought on by the membranous expansions which covered them, namely, the general integument or the mucous membrane, inflaming and sloughing away, so as to leave them denuded. This ichorous ulceration may even lead to cachexia and exhaustion; the inflammation itself, however, never gives rise to a specific infection, and to a multiplied production of the heterologous growth.

Sarcomata lend themselves naturally to a division into three species.

1. *Gelatinous Sarcoma.*—A very frequent heterologous formation, of which there are several varieties. It consists, besides some albumen, almost wholly of a gluten-, chondrin-, or pyin-like substance.

The varieties are principally referable to consistence, which varies from that of gelatin to that of fibro-cartilage, conforming itself partly to the amount of water held by the glutinous basement, partly to that of the textural, and especially the fibrous elements developed within it.

(*a.*) The first variety is a very soft, jelly-like, nearly limpid, tremulous, yellowish-gray new growth, sparingly vascularized. It is the *gelatinous tumor*—the collonema of Johannus Müller.

J. Müller describes the parenchyma of collonema as made up of spherules, some of which are much larger than blood-globules, interspersed with crystalline needles.

We have met with the gelatinous sarcoma in different organs; more commonly, however, in the brain, and in the mammary gland, bearing, if not all the attributes of Müller's collonema, at least so close a resemblance to its texture as to remove all doubt as to their identity. Our specimens presented, on the one side, a perfectly embryonic form of collonema, on the other side one more highly developed, and bordering upon the following varieties:

A roundish, goose-egg-sized, gelatinous tumor, from the mammary gland, consists of a very soft, amorphous blastema, interspersed with, for the most part, very minute elementary granules, and delicate little twig-like fibre rudiments. This blastema is permeated by whitish membranous septa, differing from the basement in nothing save in their greater consistency.

A very bulky, lobulated, gelatinous tumor from the brain, displays branched fibres, resembling the elastic, with very numerously imbedded nucleated cells, mostly larger than the pus-cell.

One extirpated along with a portion of the inferior maxilla, exhibited a stroma, consisting of elastic, branched fibres, shooting forth twig-like, out of a stem.

Lastly, a fourth, from a spermatic cord, showed single spiral, elastic-like, but transparent fibres, in an otherwise amorphous, tenacious blastema.

(*b.*) The second variety comprises a series of kindred new growths, marked by a progressively increasing density and resistance, and mostly by a very pronounced stellate and lobulated structure. A white, areolar tissue-like fibrillation, cognizable with the naked eye, and bearing in its interstices nuclei and cells, caudate nuclei and cells, and nucleus-fibres, enters largely into its composition.

There is here often both a microscopical and also a ruder alveolar

texture and cyst-formation, which lend to the heterologous growths the semblance of a glandular structure; the alveoli and cysts being the especial holders of the gelatinous moisture.

This variety of sarcoma is generally endowed with considerable vascularity. During life, the new growth exhibits various shades of redness, and offers to the feel either a woolly resiliency or a greater degree of elastic firmness. In the after-death collapse, it is of a grayish-red or reddish-white, flabby, and in various degrees resistant.

(c.) The third variety consists of a firmish, amorphous basement, broken up into solid fibres, after the manner of the intercellular substance of hyaline cartilage, and teeming with cells more or less resembling those of cartilage. This variety approximates in its elementary structure to the cartilaginous new growths, to the *enchondroma*, with which the gelatinous sarcoma is manifestly and essentially cognate.

These varieties, more especially the last two, are often found combined in one and the same new growth.

The gelatinous sarcoma, besides the rarer localities specified in the instance of collonema, namely, the brain and the mammary gland, affects the parotis, the subcutaneous areolar tissue, the intermuscular parts, and with great frequency the periosteum and the bones, more especially of the face. The second variety is particularly marked by the immense circumference to which the growth becomes developed, often within a brief space of time, through redundant lobulation and ramification.

2. *The albumino-fibrous tumor; fibrous sarcoma.*—This tumor is of a fibrous texture, and is distinguished from other and especially the pure, gluten-yielding fibroids, by its albuminous contingent.

The genesis of the fibres and their relation to the fluid albuminous blastema vary.

(a.) The fibre-texture becomes developed out of the fluid, that is the albuminous, blastema, according to the laws of the cell-theory.

(b.) The fibrous texture originates out of consolidated blastema, forming in this case a stroma, in whose interstices the fluid blastema is contained and becomes developed.

The fibre varies in all the forms derived from solid and fluid blastema. In particular, all the forms described under "Metamorphosis of Blastema," recur here as the stroma. Again, we have recognized in one of the forms specified in the section on "Gluten-yielding Fibroids," the transition from the latter to fibrous sarcoma.

Varieties of this sarcoma, dependent upon the form and arrangement of the fibres, might, accordingly, be numerously adduced. A frequent appearance in the albumino-fibrous sarcoma, is the formation of the cystic and alveolar texture.

Just as, in the gelatinous sarcoma it is the gluten-like blastema,—so it is a predominantly albuminous blastema with which the fibrous parenchyma becomes drenched, or, with which the gaps of the fibrous stroma, the cystic and alveolar spaces, become occupied.

The albumino-fibrous tumor, like the gelatinous sarcoma, occurs in areolar tissue, in the periosteum, especially the submucous periosteum; and in submucous and muscular strata of areolar tissue, as fibrous, pharyngeal, nasal, uterine polypi, &c. Moreover, it affects bones, especially

those of the face, and, more rarely, glandular organs, the parotis, the mammary gland, the testicle. It is spherical, elliptic, single, or more often lobulated and ramified, more or less resilient, vascularized, turgescent new growth, with a texture fibrous to the naked eye.

3. *The albumen-like fibrous tumor.* (Johannes Müller.)—The albumen-like sarcoma is a gibbous, tenacious, albumen-like tumor, sparely and but partially vascularized where its texture is slightly reddened and less firm. It consists, generally, of a white or yellowish-white, solid, fragile mass. Here and there it exhibits clefts or fissures, which contain a synovia-like fluid. It consists, in part, of a uniform, almost structureless or indistinctly fibrous mass. According to Müller, it is made up of a basement of multifariously interwoven, microscopic fibres, amongst which are interspersed a vast multitude of globules. The tumor is said to have yielded no gluten by boiling; the scanty extracts to have been thrown down by the reagents of casein, and the insoluble main mass to have represented an albuminous body.

We have only once encountered this new growth, namely, in the bone, within a loopholed, bony sheath.

A very interesting variety of sarcoma is the

CYSTO-SARCOMA.

The combination of the heterologous parenchyma with cyst-formation, the groundwork of which is here, as elsewhere, the parent cyst and the alveolus, together imparting to the new texture the glandular aspect.

Just as the basis of the cyst in sarcoma is identical with that of the pure cyst, so, in like manner, do the types of both simple and compound cyst-formation here recur. In point of fact, therefore, the forms designated by Müller as varieties of cysto-sarcoma, offer a mere repetition of those types.

These varieties are :

1. *Cysto-sarcoma simplex.*—The cysts imbedded in the parenchyma of the sarcoma, are on their inner surface smooth, or simply speckled with little, isolated, injected elevations of parenchymatous texture.

2. *Cysto-sarcoma proliferum.*—In the same parenchyma are found numerous intra-cystic cysts, some flattened along, others attached by a pedicle to, the parietes of the parent-cyst. With A. Cooper, we have seen some of these secondary cysts as hollow appendices, or even free within the parent cyst. In like manner parenchymatous masses of a fibrous, or of an acino-glandular structure, grow as pedunculate offshoots into the cavity of the parent cyst. This offers a transition-link to the following variety.

3. *Cysto-sarcoma phyllodes.*—A large clavate tumor, consisting of a firm mass, and presenting a fibrous torn surface. Within is one large cavity, or there are several cavities, unprovided with any cognizable proper membrane, into which firm, sarcomatous, red, vascularized, foliaceous, or warty, tufted, broad-based, or pedunculate, bulb-shaped, sometimes cauliflower-like, or fringed and villous excrescences, germinate and grow. These consist of the same substance as in the former case, but are mostly less dense and more succulent, readily drawn out length-wise

into fibres, or spread out to a membrane, showing that they exist in a folded and rolled-up condition. Along with them the cavity contains a viscid humor.

In this description of the cysts in sarcoma, we easily recognize the types of pure cyst-formation. Whatever interest, however, attaches to this repetition, its description is inconclusive as regards the real nature of the new growths. The main feature is still the heterologous parenchyma, which becomes the cysto-sarcoma without doffing its primitive character. This parenchyma is that of the gelatinous and albuminous sarcoma. A peculiarity, in point of form, consists in the membranous basement of sarcoma phyllodes, in its above-described exquisite form. Cysto-sarcomata, and the species phyllodes most particularly, often attain to a very considerable magnitude. They are frequent in the genital organs, of females more especially, in the mammary gland, in the ovary, less often in the testicles. In a word, they occur in all those organs specified as liable to the development of sarcoma, in its several species.

APPENDIX.

[Since the publication of Rokitansky's treatise on cyst and on goître, he has devoted much attention to the subject of *cysto-sarcoma*, the *chronic mammary tumor* of the late Sir A. Cooper, the *imperfect hypertrophy of the mammary gland* of Mr. John Birkett, *the mammary glandular tumor* of Mr. Paget. Rokitansky, in an essay, read before the Imperial Academy of Sciences, at Vienna, in Jan. 1853, on " The new growths of the mammary gland texture, and its relation to cysto-sarcoma," gives the details of several minute examinations of these tumors, the results of which are thus summed up : " In the one case are found imbedded within a small transparent, succulent tumor, acinus-like formations, consisting of a structureless membrane, and replete with nuclei. In the second case are found, in the texture of nodules, of which the tumor is composed, numerous delicate fissures bordered by a fringe, in which the matrix substance is, with the naked eye, seen to shoot inwards, in the form of almond- or bulb-like projections. There are also larger cavities present, into which stellate masses project. A magnifying power shows that these projections themselves begin to become lobulated at their terminal points. In other cases an extreme, lobulated, textural mass is thrown forward into a fibrous cyst ; some of its lobes being again enveloped by a cyst. Upon the surface of others are found furrows, whose margins spring up into conical and bulbous excrescences, and, together with these, larger, open, cyst-like chinks. In the texture of those lobules are, moreover, channels and acinus formations, as also upon the cut surface, chinklets cognizable with the naked eye, which, under a magnifying power of 90 diameters, appear as considerable cavities branching out in all directions, whilst between their emissaries the neighboring texture shoots inwardly in the shape of conical and bulbous excrescences."

It results from this that—

1. The acinus-like cavity with its emissaries resides in a layer consisting partly of embryonic, partly of fibro-cellular tissue.

2. The cavity enlarging, coalesces with the matrix substance, which grows into its space in the shape of conical and bulbous excrescences, whilst these, at their free extremity, throw out lesser projections.

3. As these multiply, the number of the fissure-like emissaries increases.

4. They shoot out from all points around the dilating hollow growth, or from individual points, or from one point only, into the excavation. Where they are wanting, the dilatation of the cavity is uniformly that of a cyst with smooth parietes.

5. This dilatation is often very considerable, and the excrescences may also attain to a vast size.

6. In these excrescences, a secondary formation of acinus-like growths takes place, in which the events which occurred in the primary one are severally repeated. This occasions certain excrescences to appear encysted whilst others remain bare.

7. At the surface of the excrescences are observed furrows or open cyst-like clefts, into which excrescences intrude in like manner.

Dissenting from the view to which Mr. Paget inclines, namely, that these tumors originate as cysts, subsequently lose their cyst form, and continue to grow as solid masses, differing, moreover, with Mr. J. Birkett, who attributes them to a blastema effused into the areolar tissue of the mammary gland, Rokitansky contends that an acinus-like hollow growth which determines this tumor, and repeats one element of the mammary gland, becomes developed within a matrix of new-formed connective tissue, which primarily constitutes the tumor. And this tumor is by no means encysted, unless an adventitious fascia-like sheath of areolar tissue be called a cyst. A true cyst is only subsequently developed within it through the dilatation of the said gland-structure, the connective tissue layer being expended upon the construction of the fibrous cyst-wall. This, growing into the space of the cyst, and carrying before it the primitive structureless cyst-membrane, works out an encysted textural mass, an encysted tumor, in which the gland elements are reproduced.

The dendritic excrescences, therefore, which vegetate in the cyst of cysto-carcoma, are not, in Rokitansky's opinion, and, as he once held, outgrowths from the internal cyst-membrane, but intrusions into the cyst of its own cradle mass, or matrix, still invested with the primitive cyst-membrane and its epithelium.

Cysto-sarcoma simplex, in which the cradle mass does not intrude at all into the cavity of the cyst, is of the rarest occurrence.

C. proliferum is engendered by the development, within the terminal excrescence-bulbs, of the acinus-like cavities into filial cysts, and the ingrowing of the cradle mass is here repeated.

The cysto-sarcoma phyllodes of Johannes Müller, with its amply developed, warty, cauliflower- and foliated- or cock's-comb-like ingrowths, has nothing to mark it beyond the size and development of the excrescences. The cyst-membrane is here no longer demonstrable, having coalesced with the cradle mass of the cyst.

It has been stated that the dendritic intrusions into the cyst may occur at one point only of the cyst, at several points, or, lastly, at all

points simultaneously. In the last case they converge, coalesce, and eventually fill the entire cyst, determining thus its aggregate, lobulated structure.

With two exceptions, the one mentioned by Johannes Müller, the other by Mr. Paget, of these tumors occurring in the breast of the male, Rokitansky knows them only as affecting the female breast, where they are generally seated at the inner and upper part of the mammary gland.

Their figure approximates to the spherical. Small tumors are commonly even and smooth, greater ones irregularly nodulated, knobbed, lobulated, at the same time tolerably resistant, elastic, often generally or partially presenting the feel of a cyst tense with fluid.

The skin often presents a livid aspect, and is traversed by dilated veins. It is sometimes found coherent with the tumor, but not degenerated. The mammary gland becomes displaced by large tumors, and wastes away.

These tumors have an enormous capacity of enlargement, growing, now slowly, now rapidly, often with lengthened periods of arrest. Occasionally they disappear spontaneously. In a few instances several small tumors are concurrently present.

They are usually painless; there are cases, however, in which the pain is excessive. These represent Cooper's *irritable tumor of the breast*, a fibrous tumor (a neuroma) for the most part associated with the present new growth.

The individuals affected are often unwedded, or childless females. The married and child-bearing are however not exempt.

The tumors are innocent, and, although often recurrent at the same spot after extirpation (Mr. Birkett relates a case of their reproduction five times in succession), not so beyond the range of the mammary gland. In many respects they are analogous to the fibrous tumor of the uterus, and to enchondroma.

In rare instances they undergo ulceration, which involves the superimposed cutaneous textures.]

β. CANCER—CARCINOMA.

Heterologous growths not distinguishable from sarcomata by definite generic marks, and, like these, to be dealt with only as species, but contrasting with sarcomata in the single feature, common to them all, of malignancy. Carcinomata originate and subsist not rarely as local evils. Far more commonly, however, they are associated with a dyscrasis, which, in point of fact, often precedes and engenders the cancer. Hence the multiple appearance of carcinoma as the sequel to a single one, as the sequel to the extirpation of a voluminous and hitherto solitary one. Hence, in other cases, the original appearance of cancer in several organs simultaneously, or in rapid succession.

Conformably herewith, carcinomata can rarely with adequate reason be attributed to external local causes, whilst it is very common for them to luxuriate in internal organs beyond the reach of palpable influence from without.

The crasis which gives rise to the production of cancer, consists mainly

in a preponderance of albumen, a defibrination (hypinosis), for the particulars of which we must refer to the doctrine of crasis. Concurrently with this we have, more especially in the medullary crasis, an excess of fat in the circulating fluid, which determines a complication of cancer to be discussed in a more appropriate place; and, again, that remarkable relation of exclusiveness towards ordinary, fibrinous tubercle.

This crasis is essentially the same for all cancers, only exquisitely developed in the medullary form. This may be inferred, at least, from the frequent concurrence of various cancer species, in primitive or consecutive combination, either in the same locality, or in different organs. It may also be inferred from the circumstance that, after extirpation, the one is replaced by the other under the same contingencies, and that, conforming with an augmentation of the crasis, the medullary cancer is generally the consecutive one, more especially where the substitution takes place rapidly.

The highest grades of cancer-crasis originate through infection, that is, through the reception into the lymphatics, or more especially into the bloodvessels, of cancer-cells, or of cancer-blastema, of a lax, soft, semi-fluid character. The blastema is carried thither by imbibition, partly in the mere act of nutrition, partly, with or without the cancer-cells, through the lymphatics or veins laid open by ulceration of the tumor, or lastly, by the cancer penetrating into the canals of bloodvessels. Infection thus brought about, occasions locally, or it may be remotely, both in large bloodvessels and in the capillaries, coagulations of blood. In the former case, these are cylindrical, branched, plug-like, or clavate coagula, adhering to the internal bloodvessel membrane, or to the endocardium (vegetations). They reveal their cancerous nature by their external medullary characters, as well as by their vigorous growth. In the capillaries the coagulation assumes the form of the cancerous depot—so called metastasis (capillary phlebitis).

Cancer-formation assumes both a *chronic* and an *acute* course, the former being the more ordinary mode of occurrence for primitive cancer; whilst secondary cancer production is brought about with more and more rapidity in proportion as the cancers multiply. Ulceration and extirpation of carcinoma are especially apt to determine its very acute secondary formation. Still there are instances of highly acute, primitive, general cancer production. Moreover, the individual species of cancer manifest marked differences in this respect, both the first development and the ulterior growth, for example, of fibrous cancer, being slow, whilst in the case of medullary cancer they are incomparably more rapid.

In primitive cancers, the blastema is, in the great majority, insensibly produced. In acute cancer-formation it is thrown out under the symptoms of hyperæmia, and occasionally of inflammation. In the latter case, it often covers serous membranes with a stratiform cancer exudate, or infiltrates and hepatizes the lungs with cancerous tubercles. From what has been said, our opinion may be inferred respecting the seat of cancer, in opposition to that of Carswell and Cruveilheir, who refer its origin to the capillary system. But, although in the ordinary process of cancer-formation we look upon the blastema as an exudate in its broadest sense, we by no means question the origin of cancer from

coagulation within the bloodvessels after the type of depôt-formation in general (see Metastasis). It is indeed to this mode of development that we would ascribe the rapid cancer-formation engendered, in brutes, by the injection of cancer-blastema.

We are further disposed—although from isolated facts only—to believe in cancer-formation, through a conversion of certain physiological elements into those of cancer. In the liver, namely, we occasionally light upon a process, limited to circumscribed patches, of pallescence and alteration of the parenchyma, with some augmentation of its volume. Upon further examination, the portion of liver so affected is found to consist indubitably of hepatic cells, more or less bereft of their biliary and coloring matter, and of an intermediate, whitish, albuminous blastema,—as though the hepatic cell had become transformed into the cell of medullary carcinoma.

Cancers present sometimes well-defined, easily removable, spherical, irregularly knobbed, lobulated, branched tumors, which may lose their circumscribed character, only during their progress, by insinuating themselves betwixt the elements of textures hitherto merely displaced. Or they may appear, from the first, as infiltrated heterologous masses, involving the textures without definite limits. When an established cancerous mass stretches forth from one organ to seize upon a second, the latter is forcibly drawn in the direction of the first. Membranous formations, in particular, become attached to it with umbilical flattening, waste away, and become perforated by the heterologous mass. This is especially the case with fibro-carcinoma.

The size of carcinomata greatly varies. As tumors, some, and in particular the gelatinous and medullary forms, attain to a very considerable magnitude. A special notice is due to the occurrence of cancer in the shape of little millet- or hemp-seed-sized tubercle-like granules, as detected upon serous membranes. They have the import of medullary or of gelatinous cancer,—sometimes, however, of the alveoli, or follicles of alveolar cancer. In membranous formations, the magnitude is often represented in the superficial extension of the mischief wrought by the infiltrated malignant matter,—as in cutaneous cancer, in cancerous degeneration of the dura mater, &c. The number of cancers present in an individual differs materially, varying from the solitary tumor to almost general cancer-production.

With reference to the occurrence of cancer in the different organs, it may be generally stated, that no organ or texture,—not even cartilage —is exempt from it, with the solitary exception of horny textures. Certain organs, however, are hardly ever primarily affected with cancer, being attacked only, either under the conditions of general cancer-production, or through contiguity with, and by propagation from some other organ previously a prey to the affection. Thus, *primitive* cancer of the salivary glands, or of the small intestine, very seldom occurs, of the lungs or of the spleen scarcely ever.

As regards the preference of cancer for different organs, the following average scale of frequency might be established. First, the uterus, the female breast, the stomach, the large intestine, and especially the rectum; next comes the cancer of lymphatic glands, especially as retro-peritoneal

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; of the œsophagus; of the salivary glands and parotis. Again we meet occasionally with cancers in large serous sacs, as in the peritoneum, for instance, adherent only by very inconsiderable portions of areolar tissue, or vegetating at large, and sometimes attaining to an enormous volume. Bone-cancer has now and then a sheath-like skeleton; far more frequently, however, it scatters the bony substance piecemeal, advancing at the same time, with redundant bone-formation, in the shape of a laminated, stellate, thorny stroma.

Both in the primitive, and still more in the secondary appearance of cancers, we may,—apart from all disease from contiguity—discern certain relations of sympathy; for example, between uterus- and ovarium-cancer, between testicle- and kidney-cancer, between stomach- and liver-cancer, between stomach- and intestine-cancer;—cancer of the spleen is probably always associated with liver-cancer.

Generally speaking, cancer is more common in the middle and advanced periods of life. This relates, however, only to the cancer of certain organs, more especially of the mammary glands, of the uterus, of the stomach and intestines. All other cancers, especially those of the lymphatic glands, of the brain, of the eyeball, of bone, &c., occur even in early youth. Of the individual forms, the medullary is that most common at the earlier periods of life. In rare instances it is met with even in the fœtus.

Cancers are themselves subject to not a few diseases, amongst others, to hyperæmia with intumescence, and to hemorrhage. The most important amongst them is, however, inflammation. Besides this, they are liable to metamorphoses, inductive of an involution of the cancer; and these are developed, apart from external causes, in due proportion to the vascularity of the organ, and to the looseness of its texture.

Inflammation may become kindled spontaneously in the interior, the depths, of the heterologous growth. Or it may be the obvious consequence of external influences,—of irritating medication; of exposure of the tumor, after perforation of the investing external skin or mucous membrane, to contact with the external air or with passing secretions and excretions.

Its distinctive signs are identical with those which reveal the inflammation of normal structures, and they are developed in the direct ratio of the vascularity and looseness of texture of the heterologous growth. The disease has a marked tendency to open up, and, by hemorrhage and its results, to destroy such texture. It takes either an acute or a chronic course.

Its products are sometimes organizable; more frequently, however, and more voluminously, they are coagulable, yellow, fibrinous, or yellowish-white albuminous, deliquescent, pus-like, purulent, and ichorous, —very often hemorrhagic—exudates.

Its terminations, besides discussion of the inflammatory stasis, and resolution, are:

1. *Abiding of the products* in their primitive crude condition, or else *disruption*, wasting, textural conversion.

We have here, first, to advert to the textural conversion of consolidated fibrinous exudates, to gluten-yielding fibroid textures which may spring up as a reinforcement of similar stromata already in existence. Secondly, we have to mention the development of embryonic cancer elements out of a fluid exudate, as represented in inflammatory hypertrophy,—increase of volume of the new growth. Both may concur with deliquescent, pus-like, ichorous exudates, and, emphatically, with genuine pus-exudate.

2. *Suppuration, ichorous production*,—the most ordinary termination. It runs either an acute or a chronic course, with or without simultaneous granulations, possessing the character of a rapidly developed new growth—representing certain lax, bleeding, easily suppurating, sloughing, fungus-like vegetations upon the cancerous ulcer. This process takes place either in the depths of the growth, in a shut space, as so-termed *occult cancer;* or upon the free surface of the body or of a mucous cavity, as so-called *apert or open cancer.* This last is, for the most part, marked by a funnel- or crater-like, deeply-extending base, with an elevated, mammillated brink.

Ichorous destruction of a cancer is very commonly followed by fugitive reproduction of the fungus upon the base of the ulcer, by the accumulation of cancerous matter in its vicinity, by cancerous degeneration of the implicated lymphatic glands, and, lastly, by the translation of cancer to other organs.

Even independently of suppuration, and without its concurrence, the necrosis of cancerous growths, both small and great, is not unfrequent.

Ulcerating and necrosing cancer—cancer-ichor—besides its corroding property, is marked by a very disgusting, penetrating fetor. This is, no doubt, essentially due to the sulphur and phosphorus of the broken-up protein and fatty constituents of the tumor, especially when exposed to the air.

We have here still to advert to one other important phenomenon. In cancers of the most different structure—in all cancers—we meet, not rarely, with a yellow substance, sometimes scattered in points, sometimes permeating the texture as straightened or serpentine, ramified striæ, interlaced to form a mesh- or network, or, on the other hand, imbedded in considerable masses. It is a yellow, brittle, consistent,—or a soft, friable, unctuous, glutinous substance, which, if closely examined, is found to consist either of an amorphous blastema, dotted here and there with minute molecules, and interspersed with misshapen nuclei and with more or fewer of the elementary cells of cancer,—or else chiefly scattered or grouped elementary granules (or fat-molecules), of the elementary cells of cancer replete with the same elementary granules, and lastly of fat-drops.

This substance constitutes, in the aforesaid mesh- or network, the so-called *reticulum* of Johannes Müller, who, regarding it as essentially prolific of cancer-cells, founded upon its presence a new species, under the denomination of *cancer reticulatum.*

On this point we cannot quite agree with Johannes Müller, the said

reticulum not being confined to a single species of cancer, but occasionally met with in every form of the disease.

We hold it to be, generally speaking, a solidified product of inflammation destined, earlier or later, to break up, its protein-substances along with the contained cancer-cells undergoing fatty conversion. We look upon this process as both interesting and important, inasmuch as, from its original foyer in the said substance, it gradually evokes a similar process throughout the cancerous growth. This is particularly the case where the substance possesses the reticular form, so as to master the cancerous parenchyma at all points. It is certain, however, that the cancer-blastema itself undergoes the very same transformation, and that spontaneously.

Cancers for the most part prove fatal, sooner or later, by their exhausting effects. The anæmia, emaciation, and eventual exhaustion are the result of the luxurious growth of a single tumor, or of the development of a multitude of smaller tumors, or of hemorrhage or ulceration. Moreover, cancer, like other heterologous growths, kills through mechanical hindrance to the function of vital organs which it may have made its abode—for example, the brain. Acute, violent cancer-production rapidly destroys life, through the prefatory and attendant hyperæmia of important organs. Suppurating cancers become deadly through infection of the blood, and pyæmia.

However seldom the extirpation of cancer proves successful, its spontaneous cure is a still greater rarity. So favorable a result can only be brought about either by the progressive destruction, necrosis, and partial rejection of the tumor, or else by its more rapid death and expulsion ; a circumscribing suppuration isolating it from the healthy textures—(mammary, uterine cancer).

Other processes of cure present, however, greater interest, bearing the character of an *involution*, a *decadency* of the cancer. Such are :

1. *Saponification of cancer*, a metamorphosis usually evoked by the conversion before alluded to of the substance constituting the reticulum. It partly consists in the liberation of fats, or in the conversion of protein substances into fat, with consecutive emulsive and saponaceous blending. This process, the above designation of which is warranted by a series of minute examinations, attaches chiefly to the encephaloid, medullary carcinoma, so remarkable for its proportion of fats and of mutable crude albumen, and occurs more especially in the liver and the womb.

2. *Decadency, wasting of the tumor*, with condensation, solidification of its blastema, liberation of salts of lime in the shape of free molecule, and cell-incrustation. It affects in particular the denser cancers provided with a solid blastema (intercellular substance),—the firmer medullary and the fibrous cancers. The ossification and cretefaction of inflammatory products in cancer often gives the first impulse to this. Here again ossification and cretefaction (of the reticulum) are combined with fatty conversion.

Although carcinomata are, generally speaking, pre-eminently malignant new growths, still the degree of their malignancy is not the same in all, medullary carcinoma occupying the extreme point of malignancy,

whilst colloid and the epithelial cancers are in this respect the mildest of all.

1. COLLOID, GELATINOUS CANCER.

Alveolar Cancer (C. Aréolaire).

In the array of cancers we again encounter a gelatinous, colloid new growth, namely, *gelatinous cancer*, better known under the epithet *alveolar*, derived from its very frequent *alveolar fabric*. This texture cannot, it is true, alone mark the character of a species. It occurs, however, in gelatinous cancer so commonly, and at the same time in so exquisite a form, that under alveolar cancer nothing else is understood than gelatinous cancer.

This species occurs under two forms:

1. It presents a yellowish-gray, yellowish-red, here and there colorless, firm, jelly-like, transparent, tremulous, sizy, and when voluminous, irregularly clavate, lobulated mass. This consists of embryonic elements —for the most part a cell analogous in appearance to the pus-cell—in an amorphous colloid blastema, and of a very scanty, very delicate, fibrous texture, mostly investing the interlobular clefts. Bloodvessels enter into its composition only in very subordinate number.

2. *So-called* alveolar cancer—cited by Otho, in 1816, as a peculiar kind of stomach-scirrhus—cancer glélatiniforme, aréolaire of Cruveilhier.

The growth consists mainly and characteristically of follicles (alveoli) of either very delicate, pellucid, or of more compact and massive, white, satin-like, shining fibre-texture, containing a colorless or a pale yellow, grayish, diaphanous gelatine. Sometimes the growth consists entirely of these follicles, with inconsiderable columns of fibres intervening between them. Then, again, in the deeper layers, towards the base of the new growth, we shall find the follicles separated by a copious, firm, white fibrous stroma of new parenchyma. Its quantity stands obviously in an inverse ratio to the quantity and the grade of development of the follicles. This is shown most especially from the examination of cancers of the stomach and intestines. The walls are here found considerably thickened, hard, clavate, the inner layer presenting a multitude of collateral and superimposed hemp-seed- or pea-sized follicles, the innermost of which open, collapse, and cast their gelatine upon the inner surface of the stomach or intestine. In proportion to their depth, the follicles are, with exceptional patches, smaller, whilst the densely fibroid stroma before referred to gains the ascendency. This character of alveolar cancer is frequent both in the stomach and intestines, and may be designated as a combination of the alveolar with the fibrous form.

A closer investigation of alveolar cancer presents a fibrous texture of the parietes of the alveoli, and of the inter-alveolar substance. Along with areolar tissue-like fibrils, black-contoured granule- and nucleus-fibres, with similar fibre elements arising out of solidified blastema and uninfluenced by acetic acid, play here a great part. In the gelatine are found, besides elementary granules and nuclei, non-nucleated and nucle-

ated cells, cells with one or with multiple cells, parent cells. Besides these, there are present spindle-shaped, caudate cells,—under certain conditions granule-cells, together with a large proportion of fat-molecule. Johannes Müller obtained out of this gelatine, by boiling, no trace of gluten. An alcoholic extract, boiled with water, contained but an inconsiderable quantity of a substance somewhat akin to ptyalin.

According to Mulder, the main constituent of alveolar cancer does not occur at all in the healthy body. We regard it as the same gluten-like substance which furnishes colloid (see Colloid), and which, as we see, constitutes numerous new growths, both benign and malignant.

Cruveilhier further distinguishes a "cancer aréolaire pultacée," the follicles of which instead of transparent gelatine, contain opaque pultaceous matter, in which Boutin Limousineau has detected casein. We hold this cancer to represent a transition state of cancerous gelatine to fat, with its ulterior saponaceous and emulsive combinations, partly in the act of granule-cell formation. The same transformation is witnessed in like manner in the first form of gelatinous cancer, and often affecting considerable portions of it. It is in its nature analogous with the so-called reticulum of fibro-cancerous textures.

Alveolar cancer displays, in a consummate form, all the characters assigned, under the head of "Cyst," to the alveolar texture. In its most pronounced, that is, its most fully developed state, it offers the following varieties:

(a.) The gelatine accumulates in the follicles in so excessive a degree that the walls of the alveoli, owing to the distension, become thinner and thinner until reduced to a mere residue. The heterologous mass degenerates into an almost uniform tremulous jelly, traversed by delicate membranous septa,—the residua of the alveolar walls. It is, in point of fact, scarcely to be distinguished from the first form of gelatinous cancer.

(b.) The follicles dilate with increase of substance of their walls (hypertrophy) into cysts, attaining thus to an enormous magnitude. This excessive development affects the follicles more and more, in proportion as they are more peripheral. The new growth presents an aggregate of collateral and superimposed cysts, one or more of which, at the periphery, are of immense circumference. This growth resembles the compound cystoid, inasmuch as a redundant alveolar type is common to both, a circumstance readily ascertained in the instance of alveolar gelatinous cancer from the more and more dense fibro-alveolar structure exhibited on its cut surface, in proportion as its base is neared.

The contents of the enormously developed follicles include all the variations observed in cystoids, just as their parietes are subject to the same class of diseases.

The parts most liable to alveolar cancer are the stomach and the large intestine, the serous membranes and the peritoneum in particular, the omentum (less often independently than in association with cancer of the stomach and colon), the ovary, the bones, in rare instances the kidney, the uterus, and the liver.

Wherever situate, gelatinous cancer generally enlarges, and that often in a short time, to enormous masses. Upon serous membranes, espe-

cially the peritoneum, it occurs in scattered gelatinous accumulations, in little millet- or pea-sized tubercula, or in larger masses,—occasionally as a continuous bulky growth, which vegetates from a few points of adhesion only, if not almost free within the peritoneum.

Upon the peritoneum the first form is predominant, but not to the exclusion of the alveolar, the scattered tubercula having the character of isolated alveoli. In the ovary the cyst-like alveolar cancer is prevalent, very often as encysted dropsy of the areolar cancer-form. This form occurs also now and then in the bones.

Gelatinous cancer, and especially the alveolar, is sometimes associated with other cancers. This combination, however, with alveolar cancer, must be considered apart from the alveolar type. This type constitutes every cancer an alveolar, but not a combination with alveolar gelatinous cancer. Every alveolar cancer may be regarded as associated with fibrous cancer, by virtue of a notable fibrous inter-alveolar substance. A combination with medullary cancer is generally brought about by the medullary cancer supervening upon the alveolar, the peripherous follicles of which fill with, and are eventually overlapped by the looser encephaloid mass; or else, the medullary cancer grows into the cavities of the alveoli. More rarely, the gelatinous cancer supervenes upon the medullary, in the cystic and alveolar form.

Pure gelatinous cancer is the least malignant of any, and, unless it prove exhausting by dint of surpassing volume, a dependent cachexia is less pronounced than in other cancers. It is very rarely the seat of inflammation and ulceration. It is for the most part solitary, although somewhat prone to extend to contiguous organs, and to scatter itself over serous surfaces, in the manner already adverted to.

2. FIBRO-CARCINOMA.

Simple Carcinoma.

The *schirrhus* of older pathologists, the only new growth designated by them as cancer; other equally and still more malignant formations being by them divided into sarcoma and fungus. It is upon the whole the most compact in texture, and therefore the hardest of cancer-growths. Hence, the expression scirrhous hardness, formerly employed to denote in an organized product a resistance analogous to that of fibro-cartilage.

In a parenchyma like that of the mammary gland, scirrhus commonly appears as a clavate, gibbous, indistinctly lobulated, somewhat branched, not sharply defined, very hard, grayish, or bluish-gray new growth, which has the faculty of dragging down surrounding textures upon itself, is of moderate size, of from a walnut to a duck's egg, is heavy in proportion to its density of texture, and creaks under the knife.

Several deviations, to be hereafter specified, here present themselves. We shall, however, limit ourselves in this place to the statement that the density of the fibrous texture, sufficiently cognizable with the naked eye, as also the hardness, do not always attain the presumed high grade. Under certain conditions, scirrhus becomes tolerably lax and succulent.

On a more minute examination, the principal mass appears to consist

of a *fibrous texture*, imbedded in which are *embryonic elements*, in the shape of nucleus and cell. The former gives it the impress of fibrous cancer, and determines its density and hardness. The greater the number of the embryonic elements, the more does its fibrous texture serve as a stroma for a constituent, whose preponderating mass alters and determines its characters.

The fibrous groundwork presents manifold differences with respect to the form and the arrangement of the fibres.

1. Very commonly it is a fibre resembling an areolar tissue-fibre or fibril, or that of the organic muscles.

2. Sometimes it is a consolidated, tolerably transparent blastema, in the act of splitting into fibres and fibrils, and presenting a fibrous torn surface. In both instances there is an accession of granule- and nucleus-fibres in various numbers.

3. In a case of stomach-cancer it was a dense felt of black, branched, anastomosing fibrils, similar to the fibrils of fibrin.

With respect to arrangement:

1. The fibres for the most part point in one direction parallel to each other.

2. They radiate from different centres.

3. Considerable columns of fibres traverse each other at various angles, so that upon a parallel-fibred section we find displayed, here and there, the stumps of transversely and obliquely divided fasciculi.

4. The alveolar fibre arrangement is very frequent.

The embryonic elements consist of nucleus and cell. The former are often very numerous, as spherical, shining nuclei, furnished with black contours. Not rarely, indeed, the transparent nature of crude, cancer blastema makes it appear as if the cancer consisted exclusively of these nuclei.

The cells present many points of difference.

They are round, or angular, ganglion-globule-like, or again wedge-shaped, caudate, &c.

There are often present parent-cells, which become developed into alveoli; and upon a cut surface we meet with these, visible to the naked eye, in the shape of prominent, transparent vesicles, imparting to fibro-carcinoma the aspect of a glandular structure. Bloodvessels are not wanting in scirrhus, although their abundance is not very great. Lobstein is wrong in asserting these growths to be non-vascular.

Although the mass of fibro-carcinoma is not altogether dissolved by boiling, it yields, nevertheless, a notable amount of gluten. Compared with medullary cancer, it contains a smaller proportion of fat (according to Martigny, a soft fat; according to Breschet, cholesterine).

Acetic acid certainly does (although denied by Müller) render the cells more limpid, throwing nuclei and nucleus corpuscles with black contours and some little shrivelling, more into relief.

No other cancer possesses, in so high a degree as the fibrous, the tendency to condense and corrugate the textures, in which it has taken up its seat, or to drag down upon itself contiguous, especially if they be membranous, parts. The invariable consequence is the wasting of the cancerous organs, and the shortening, with consolidation, of implicated

membranous formations. Fibro-carcinoma is slow of growth, and slower in proportion as the fibrous character predominates in its fabric. It will thus vegetate long, without producing any visible cachexia, provided it do not interfere with the function of any vital organ, and provided it remain solitary. A more rapid growth is always conditional upon an overpowering development of embryonic elements out of fluid blastema ; which latter, in the inverse ratio of its plasticity, relaxes the texture of the scirrhus, drenches it, and causes it to swell. It is often of a medullary (encephaloid) character, giving rise to a combination of fibrous with medullary cancer, in the shape of a more or less intimate blending of the two. With this, there is always a simultaneous increase of vascularity in the cancer-parenchyma, hyperæmious tumefaction, and inflammation ; frequently, also, the development of a reticulum.

The presence of this reticulum changes fibrous cancer to that form which Johannes Müller has designated carcinoma reticulare or reticulatum. That is to say, we conceive ourselves to be warranted by experiment in assuming the latter to be fibrous cancer, plus the reticulum,—fibrous cancer in the aforesaid progress of rapid and redundant growth, and incontinently passing into congestion and inflammation. Its form-elements are identical with those of pure, fibrous cancer ; although the embryonic elements and bloodvessels predominate. The capacious cells and membrane-clad cavities met with occasionally in carcinoma reticulatum by Johannes Müller, are probably nothing more than the follicles of an alveolar texture that has invaded the fibro-carcinoma ; such follicles being replete with the substance of the reticulum as a product of inflammation. This so modified fibro-carcinoma frequently occurs in the mammary gland, attaining, for the reasons stated, a greater volume than pure, fibrous cancer.

Fibrous cancer occurs (primitively and in a developed form), in the mammary gland ; in the stomach, perhaps, still more frequently ; in the colon ; in the submucous areolar tissue ;—more rarely in the vaginal portion of the uterus, upon serous membranes, and in the subserous areolar tissue. Again, as an expansive degeneration of the omentum and of the mesentery ; in the salivary glands ; in the fibrous tunic of the bronchia. In several of these, as well as in other structures,—for example, the ovaries, the brain,—there occur cancerous growths of embryonic composition, and in all likelihood of fibro-cancerous nature.

With respect to shape, fibrous cancer in and upon membranous structures deviates from the clavate form before described. In the stomach, for instance, it represents degenerations, spreading along the course of the submucous, areolar stratum, and only here and there swelling into knobbed projections ; whilst in the intestine it assumes the annular shape. Upon serous membranes, the pleura for example, it sometimes occurs as a fibroid exudate, that is, as a densely fibrous, whitish, shagreened mass of unequal thickness, branching, as if outpoured, or dropped here and there, over the surface.

In the bones it appears in the shape of roundish knobs, imbedded in the diploe of the cylindrical bones, over which the compact covering plate becomes wasted by compression, giving occasion to spontaneous bone fractures.

In the frequent cases of cancer of the stomach we have the best opportunities for studying the character of the cancerous degeneration of muscular tissues. It consists in a development of white interfascicular striæ, imparting to the fleshy tunic a white-celled aspect. The white striæ consists of accumulations of nuclei, cells, and lastly, fibres, which receive and so to speak encapsule the swollen, reddish, or yellowish red, exsanguine muscle-substance. The formation of these septa multiplies, and they increase in volume until the muscle has entirely given way and perished.

Fibro-carcinoma is, for the most part, the primitive cancer in the organism, and very rarely indeed the secondary. The cancer-growths consecutive to it have, in proportion as they multiply, more and more the character of the medullary form. Even the occasionally more rapid development of fibro-carcinoma takes place under the supervention of medullary carcinoma, and the affection of the implicated lymphatic glands occurring in the consecutive series is of the same medullary character. In like manner the extirpation of fibrous cancer is generally followed by medullary growth.

In conclusion, we would advert to certain malignant accumulations, proved by antecedent circumstances to be undoubtedly cancerous. These infest bone, the ovaries, again the mediastina, the retro-peritoneal space, lastly, the intermuscular areolar tissue; and they are distinguished for the great bulk to which they attain. As regards their elementary fabric, they are almost always embryonic structures, that is to say, they consist of nuclei and spindle-shaped or caudate cells, which last, by their arrangement, impart to the whole the semblance of fibrillation. The intercellular substance (blastema) is very scant; and the heterologous mass is consequently very dense and firm. They are to be regarded on the one hand as embryonic fibre-cancers; on the other, as kindred with the firmer varieties of medullary cancer.

3. MEDULLARY CARCINOMA.

In every way the most malignant heterologous growth, described by Burns as *spongioid inflammation;* by Hey, and afterwards by Wardrop, as *fungus hæmatodes;* by Abernethy as *medullary sarcoma;* by Monro as *fish-testicle-like* (soft roe-like) *tumor;* by Laennec as *encéphaloide;* by Maunoir as *fongue médullaire.* All these appellations serve well to designate the external characters of this new growth; that of fungus hæmatodes being, however, applicable to a combination of this malignant growth with redundant vascularity. (See New Growth of Bloodvessels.)

If, for the sake of unity and clearness, we select for our principal delineation, medullary carcinoma in its most marked form, and with all the attributes of the most malignant cancer, we must preface the description by admitting that in the instance of no other cancer are more variations from this cardinal character cognizable.

In this, its exquisite form, medullary carcinoma certainly does offer a striking resemblance with the brain-medulla of younger individuals, or with the testicle of fishes; namely, a soft, semi-fluid, when present in large quantity, fluctuating, white, or under certain conditions, reddish-

white or gray, yellowish-white, red or russet, or even in various degrees blackened, heterologous mass.

As an independent tumor, its cut surface exhibits either a perfectly homogeneous or else a variously cancellated, lobulated, more or less distinctly fibrous structure. When pressed or scraped, the cut-surface also yields a perfectly homogeneous substance out of a parenchyma which mingles with water to a uniform mass. Or, again, the entire mass separates into a looser medullary constituent, and into another more consistent, which furnishes a sort of stroma for the former, and appears as a more or less fibrous or villo-membranous framework. The relative quantity of both varies considerably.

These relations are subject to great variations, determined for the most part, by the degree of consistency of the heterologous growth as cognizable with the naked eye. There are some growths of this kind which recede so far from the medullary character, as hardly at all to tally with the description above given of medullary carcinoma. Still, the occasional blending or interlacing of such deviating structures with exquisite medullary carcinoma, in one and the same organ, the embryonic condition of their elements, their rapid growth, and their voluminous character, seem to justify their mention in this place.

Thus there are, on the one side, medullary carcinomata of almost cream-like fluidity, or which, infiltrated into the textures, into the medullary system of the bones, or into the sheaths of organs after the destruction of their parenchyma,—for example, the neurilemma of the pituitary gland, the capsule of the spleen, &c.,—resemble a milky juice. No stroma enters into their composition. On the other side, there are congenerous growths—heterologous masses, very commonly regarded as medullary carcinoma in a crude state, that is, in a primitive stage of the true medullary encephaloid—which, in point of consistency, do not yield to the fibroids, to fibro-cartilage. Amongst these denser masses there is one particularly remarkable—namely, an often very voluminous, in appearance, and also in reality, unevenly-lobulated, homogeneous, whitish, or yellowish-white, heterologous mass, which offers a striking analogy with the virgin mammary gland, especially in point of firmness and of elasticity. It is probably to this that Abernethy applied the term *mammary sarcoma*. Others present the aspect of a glandular structure; for example, of the texture of the salivary glands, or of the cortical substance of the kidney.

Lastly, the vast difference in bloodvessel-formation, referable to the structure of medullary carcinoma, is perceptible even to the naked eye. In no other parenchyma does it appear so frequently in redundance as in medullary carcinoma. Conformably herewith none is so susceptible of hyperæmia, of tumefaction, and of rapid growth; in none do hemorrhage (apoplexy) and inflammation so readily occur—processes, upon which the anomalous coloration of genuine white medullary carcinoma obviously depends.

The differences, however, discoverable with the naked eye in carcinoma, are slight compared with those revealed in the elementary texture of medullary carcinoma, with the aid of a magnifying power.

They are divisible into those recognized by the naked eye as compo-

nents of medullary matter, and into those which, at the same time, present an intercellular substance,—a stroma.

With reference to the former, there are medullary carcinomata.

(*a.*) Consisting of granulated cells with a more or less distinct nucleus, and resembling pus-globules.

(*b.*) Consisting of smaller and greater, granulated, round, or angular, protuberant cells, more or less resembling the cells of tessellated epithelium, the hepatic cells, the ganglion globules, and provided with one or several nuclei.

(*c.*) Consisting of spindle-shaped and caudate, nucleated cells, fibrecells, amongst which are many others, both spherical and oval.

(*d.*) Consisting of elliptical corpuscles, of $\frac{1}{100}$ to $\frac{1}{30}$ of a millimetre in circumference, and furnished with one or two nucleoli. They have the significance of a (heteroplastic) transcendent development of cell nuclei.

(*e.*) Consisting of spherical or oval corpuscles corresponding in size and tendency with the cell-nucleus.

(*f.*) Consisting of elementary granules down to the finest moleculemass, with scanty nucleus formations in progress of development.

(*g.*) A further element concurrent with those specified at *b*, are pouchlike formations (see Metamorphosis of Blastema), and chiefly the parentcell, which often constitutes a prominent element in medullary cancers. It forms here again the groundwork for the alveolar textural type of medullary cancer.

These elements occur predominantly, it may be, in the one or the other form, but intermingled with others. Viewed with the naked eye, the elementary composition of a texture is, even to the well initiated, a matter rather of conjecture than of any certainty. The consistency and density of a texture may vary infinitely, being dependent upon the character of the intercellular substance. It is only where there is the appearance of fibrillation that we may perhaps infer a composition of spindle-shaped or caudate cells.

Differences more important affect the character of the intercellular substance, and of a stroma in which the elements adverted to lie imbedded. This stroma is developed either out of those elements themselves, which, according to the laws of the cell theory, form into a fibrous skeleton work; or else it springs immediately out of a consolidated, amorphous, intercellular substance. Both together occasion, in medullary carcinoma, a special structure manifest to the naked eye, in the shape of a variously disposed fibrillation and lobulation, &c., the character of which so greatly modifies the consistency of the heterologous growth.

In this regard, we have the following forms, some more or less cognizable with the naked eye.

(*a.*) A medullary carcinoma, with an amorphous fluid, or semi-fluid, intercellular substance. The aforesaid elements vegetate in a thin or a thickish medullary juice. It is represented in the very lax, milky or cream-like *encephaloid* cancer.

(*b.*) A medullary carcinoma, with a solidified, amorphous, or else striated, indefinitely fibrous, intercellular substance, interspersed with roundish and fibro-elongated nuclei.

(c.) Medullary carcinoma, with a stroma consisting of fibre-cells (spindle-shaped, caudate) arising out of the development of the elements of the medullary substance itself, with consumption of the intercellular substance, and condensation of the heterologous growth.

(d.) Medullary cancer with a delicate hyaline, structureless, or else an opaque, striated, membranous stroma, studded with elementary granules and nucleus formations, or fibrillated like areolar tissue ; which stroma, at the same time, forms the groundwork for the vascularization of the alien growth. Its interspaces are filled with a loose, fluid medullary matter, and it is easily thrown into relief if the tumor be scraped, pressed, or simply steeped in water. In villous cancer this stroma appears developed into a main constituent.

(e.) Medullary carcinoma with a more or less developed fibrous stroma, whose fibre-elements, upspringing from a solidified blastema, now resemble fibro-cellular tissue, now organic muscle-fibre. It represents either a scaffold-work or a stellate structure, the gaps being filled up with embryonic elements. Even with the naked eye it is discernible as denser striæ, disposed as aforesaid, and remarkable for their whiteness and their tendon-like lustre. This stroma has frequently the significance of fibrous cancer blended with medullary. It is, however, often enough an innocent fibroid growth, which may very possibly become the seat of so-called ossification (bony concretion). Hence the extraordinary phenomenon of medullary cancer becoming traversed by a concrete skeleton-work, in the midst even of soft parts.

This seems the proper place to take into consideration another combination with a benign new growth in the shape of a stroma, namely, that with *normal bone-texture.*

A normal bone-texture occurs very frequently in medullary cancer affecting bones, as a thorny or stellate skeleton or stroma. This is, however, generally limited to the base of the alien growth. Greater interest attaches to a medullary carcinoma, possessing throughout a firm bony stroma, which, as a finely cancellated diploë, receives into its cancelli the soft parenchyma of the medullary cancer, to which it bears a relation similar to that of bone to its normal medulla. This growth certainly affects bones and their vicinity, although not exclusively. It is what Johannes Müller termed *malignant osteoid.* The bony texture entering so largely into its composition, is a very remarkable phenomenon, but its nature is simply that of a benign stroma for the reception of a cancerous, soft parenchyma.

An important part is assigned, in medullary carcinoma, to the parent-cell, and to the alveolar textural type resulting from it. We have often examined medullary carcinomata which mainly consisted of parent-cells. One consisted entirely of parent-cells, and being in the progress of fatty conversion, it presented a very peculiar aspect. Numerous liver cancers were found to consist of a fish-roe-like accumulation of yellow, poppy-grain-sized granules—parent-cells, replete with fat-containing filial cells —loosely connected together by a liquid, lardo-glutinous, yellowish-brown, intercellular substance.

Both forms of the alveolar texture occur in medullary cancer, the true alveolus, and also the aciniform, excavated body. Both, more especially,

however, the latter, determine the likeness of many medullary cancers with gland-textures. Both may coexist independently of each other, or the second vegetate as an endogenous growth within the alveolus. Medullary carcinoma occurs no less frequently as cysto-carcinoma.

Upon the dura mater, heterologous formations are not unfrequent, which, closely resembling granular cortical substance of the kidney, consist of spherical or roundish rolls of caudate cells, imbedded in a layer composed of the same elements. They are gorged with a white medullary juice, are for the most part considerably vascular, and of a turgid, soft consistence.

Medullary cancer consists mainly of albumen, with fat, according to Wiggers a phosphorus-holding fat (brain-fat), according to Gugert cholesterine, and, as Eichholtz contends, with pyin.

Medullary carcinoma ordinarily assumes the form of roundish tumors; not rarely, however, both primarily and consecutively, that of infiltration into the parenchyma of every variety of organ. To the naked eye, the tumors often seem sharply sundered from the surrounding textures. Nevertheless, the impossibility of dissecting them out, without injury to those textures, and a narrower scrutiny, teach us that they penetrate into neighboring textures, and moreover, that they grow in suchwise as to infiltrate and destroy the textures in their immediate circumference. In other cases, however, they are capable of being shelled out of an organ, having a very delicate, areolar tissue-like, vascular sheath. Such growths are generally furnished with a membranous stroma, are more or less distinctly lobulated, grow independently, and simply jostle the textures out of their place.

Medullary cancer in the one case grows to an enormous volume, in the other case is remarkable for its numerical dissemination. Its increase in volume, especially when rapid, takes place through the accession of embryonic elements. Hence the circumstance that old medullary carcinomata which suddenly undergo great augmentation of volume, have, at their base only, a solid and textural stroma, or it may be a bony skeleton. Medullary carcinoma is, both in its development and in its subsequent course, the most acute of all cancers. As a solitary growth in the organism, it arrives very rapidly at its full volume, and throws out a multitude of secondary tumors with the same celerity, not unfrequently under the accompaniment of very acute typhoid fever. The more hurried its development, the more does the embryonic form (elementary granule, nucleus, fluid intercellular substance) of its elementary composition, that of genuine encephaloid, predominate. Wherever cancer-production is acute, its form is the medullary.

Conformably with this, every other cancer, goaded into redundant growth, degenerates into the medullary, that is, enters into combination with the latter, the new accession being the medullary. The fungus upspringing from the ulcerating base of a cancer, is in its nature medullary. Every consecutive, every general, cancer-production is invariably medullary, nor is there any organ in which medullary carcinoma does not occur, either primitively or consecutively, as part and parcel of general cancer production.

In point of fact, medullary carcinoma occurs in organs in which no

other cancer, least of all fibrous cancer, ever occurs; as in the liver, the kidneys, the lungs, the testicles, the lymphatic glands.

In the bones, medullary cancer is frequently distinguished by a lamina-stellate, thorny bone-skeleton, the form often obviously depending upon the nature and arrangement of the stroma. Sometimes it causes the bone to rise up into a bone-capsule; more frequently, however, it dissipates it into a voluminous honeycomb mass.

In medullary carcinoma the cancer-crasis has attained its *highest grade*. It experiences a further augmentation through infection,—through reception of this, the most readily absorbed cancer blastema, into the lymphatics and bloodvessels. The products of inflammation placed under its influence are eminently albuminous, white, opaque exudates, and these become developed into medullary cancer, upon serous membranes, or as cancerous lung hepatization, and the like. The same thing happens with respect to coagula within the vascular system, both in the greater vessels and in the capillary system; by dint of an alienation of the fibrin, they bear evident marks of the cancerous character—cancerous phlebitis, capillary phlebitis (deposit).

We cannot subscribe to the assumption of regular stages of medullary cancer, of a stage of crudity, of softening, of ulceration, &c.; these being conditions not correlated by any necessary causal links.

That which is regarded as crude medullary cancer, is the variety furnished with a consolidated intercellular substance. Softening obviously characterizes the form of medullary cancer luxuriating as the true encephaloid, and it attaches equally to that which originates at once as such. Lastly, the ichorous and ulcerous destruction of the structure is a consequence of its inflammation, that is, of accidental disease of the tumor.

Medullary carcinoma frequently destroys life as a consequence of its surpassing growth, either as a solitary alien formation, or as one distributed over several organs,—through cachexia and exhaustion, through hindrance to the function of important organs; for example, of the digestion, of the larger veins,—the vena cava, by its closure; again through hemorrhage; finally, through inflammation and ulceration, often under the symptoms of cancerous infection of the blood.

The substance constituting the reticulum occurs, especially in the softer forms of medullary carcinoma, in large accumulated masses. In the forms furnished with a fibrous or membranous stroma it follows for the most part the distribution of the latter, and therefore of its bloodvessels.

Contradictory as it may seem, after what has been stated, in no cancer is a spontaneous or natural process of cure brought about so frequently as in the medullary.

Such a process is the sudden and rapid destruction of the cancer by *ulceration* and *necrosis*, as observed not unfrequently in the dead subject, in medullary cancer of the womb. Such a process, again, is the metamorphosis described, under the general heading of " Cancer," as *saponification* and *incrustation*. Moreover, it is known as a fact, that medullary carcinoma in the subcutaneous fat-layer will disappear through resorption, and return again.

With reference to the fungus hæmatodes of Wardrop, and the medullary carcinoma of Abernethy, we feel compelled to subscribe to Walter's verdict, namely, that they are identical. For we have always found the former to resolve itself, when closely examined, into medullary carcinoma with luxuriating vascularity. Assuming, therefore, the term "fungus hæmatodes" to designate a mere accidental condition of medullary carcinoma, there might be no impropriety in abandoning it, or in understanding by it only a highly vascularized medullary carcinoma.

On the other hand, it is requisite to bear in mind that which we have stated under the head of bloodvessel formation, namely, that assuredly there are alien growths, which, although primitively mere bloodvessel luxuriations, may subsequently combine with cancer, and this possibly without any concurrent anomaly of the general crasis, through mere impairment of the blood held within their own capillary system. We must here once more refer to the results of Van der Kolk's injections of the growths in question, which induced him to discriminate between fungus hæmatodes and medullary carcinoma.

That medullary carcinoma has some sort of affinity to the medulla of the nervous system, appears, not alone from its general aspect and chemical composition, but also from the fact that, in medullary cancer of the eyeball, the tumor springs from either the retina or the optic nerve, and that nerves speedily perish within the range of medullary tumors.

To medullary carcinoma we shall annex, *as varieties*, certain growths which bear an affinity to it.

(a.) CANCER MELANODES.

The entrance of pigment into the composition of any cancer converts it into cancer melanodes. Nowhere, however, does this substance occur in so marked a degree as in a cancer closely resembling the medullary. It may indeed be said, that *cancer melanodes (so-called malignant melanosis) is but a medullary carcinoma modified by pigment*, an idea promulgated by Meckel, von Walther, and others, in their day.

Cancer melanodes, as an independent tumor, presents most of the physical aspects of medullary carcinoma. Its cut surface appears to the naked eye either homogeneous, or fibrous, or lobulated, and of a more or less firm and brain-like consistence. A closer inspection of it reveals elementary granules, nuclei, cells of spherical or oval, caudate, elongated, angular shape, and along with these the most varied intercellular substances and stromata. Melanotic cancer imitates most commonly the encephaloid variety of medullary carcinoma, with round and caudate cells, and a membranous—a villo-membranous—stroma.

These alien growths are chiefly marked by their black or brown-black, brown, bronze-green, or rust-brown coloration. The first glance at these often numerous tumors generally suffices to show that the color is merely accessory. For, amongst thoroughly tinged, we meet also with perfectly colorless, white, heterologous growths; and again between the two extremes others pigmented in the most various forms, in dotted or

stellate patches, or in ramifying anastomosing striæ. The white growths are recognized at once as genuine, ordinary, encephaloid cancer.

A minute examination detects, according to circumstances, a greater or lesser proportion of pigment, and, even in the blackest, elements enough—cells and intercellular substance—free from pigment.

Pigment occurs free or inclosed in cells, in all the forms enumerated under that heading. Its basis is, as there taught, and especially as the examination of acutely produced or redundantly growing cancer melanodes incontestably proves, hæmatin in a free and dissolved state, or else blood-globules, with their pigment, in substance. In the latter case, the alien growth resembles a hemorrhagic effusion, in which are found along with the blastema the elements of medullary cancer in various phases of coloration and of conversion into pigment.

Chemical analysis must needs detect the constituents of medullary carcinoma, and the pigment with its base. Barruel and Henry have discovered, in the melanosis in man, hæmatin, fibrin, three kinds of fat, a considerable amount of phosphate of lime, and iron.

Like medullary carcinoma, cancer melanodes is found to infiltrate the textures of parenchymata, as also of membranous parts, the dura mater for instance.

By reason of its pigment, melanotic cancer may be studied at its outset in very small point-like portions, which, under a magnifying power, appear minutely ramified.

Like genuine medullary cancer, the melanotic often attains to an extraordinary circumference. Its simultaneous occurrence in many, if not in most organs, is, however, still more usual. Its multiplication is often very rapidly brought about, with the concurrence, it may be, of acute typhoid fever. No organ is exempt from the disease. Even when attacking all, or several, organs simultaneously, it may grow inordinately in a single one or more than one, in which case the liver is almost always found to be the organ of predilection. We have seen it in the brain and about the nerves, at the eyeball, in the lungs, in the thyroid gland, in the liver, spleen, kidneys, bones, lymphatic glands, ovaries, in and beneath the intestinal mucous membrane, between the mesenteric layers, in the skin and subcutaneous areolar tissue, upon serous membranes, in the dura mater, upon and within the heart.

In the majority of cases, cancer melanodes is found to affect middle-aged or still older individuals. Both we ourselves and others have however observed it with little less of frequency even in youth.

The crasis upon which cancer melanodes is based, is without doubt essentially the medullary. The pigment has, however, still to be accounted for. A special dyscrasial character of the hæmatin and of the blood-globules might here suggest itself, a crasis analogous to the constitution of the portal blood with a continuous excess of aged and spent blood-globules which have reached their climax of coloration in a defibrinated plasma, the ready suscipient of hæmatin. Such a view would find support in the cachexia so often concurrent with melanosis, and so characteristic of a predominant venous constitution, with a vivid, brownish coloration of the common integuments. And to this might be added the fact, that cancer melanodes is more than ordinarily rich in

pigment when occurring in the liver and the choroid plexus, in which, for various ends, pigment is thrown out from the spent blood-globules even in the physiological state.

But, apart from numerous exceptions in this last respect, we must guard against overlooking very important local processes in cancer melanodes, where the base of the pigment is furnished, not by the general circulation, not by hæmatin, but by substantive blood-globules. Here the question is, first, whence is derived the blood as the basis of pigment? and, secondly, what causes the transmutation of the blood to pigment? The latter question is the more pertinent that in *medullary* carcinoma hemorrhage is common enough without any entailment of the pigment of cancer *melanodes*. In reply to the first query, we have to express a well-substantiated conviction that the blood furnishing the base of the pigment in cancer melanodes is not—at least not mainly—an extravasate out of a perfected system of bloodvessels ; but blood newly formed in parent-cells, and transformed into pigment either within these cells or upon their breaking up.

This metamorphosis within parent-cells engaged in a process of radiation and ramification into a capillary system, explains the circumstance that the pigment, in its first manifestation in the parenchyma of a genuine white medullary carcinoma, appears in the form of finely branched and stellate points and patches.

Cancer melanodes generally proves fatal in its excessive, multiple production, through the exhaustion and wasting corresponding to such redundant alien growth. In rare instances cancer melanodes enters upon a process of ulceration, and kills through hemorrhage or simple exhaustion.

TYPHOUS SUBSTANCE.

The product of *typhous blood-stasis* deposited, in intestinal typhus in the follicular apparatus of the bowel, in broncho-typhus in the bronchial glands, and probably in plague-typhus in different superficial lymphatic glands, appears to us so analogous in many points with medullary carcinoma that we do not hesitate, in accordance with an opinion long entertained, to award it a place here.

Typhous substance appears, in extreme cases where it is rapidly produced under violent symptoms, as a grayish or whitish red, or a gray, or a white, lax,—in the mesenteric glands almost diffluent,—fluctuating, medullary substance, which, in its external features, bears the most striking similarity to encephaloid cancer.

This typhous substance, after abiding for a certain period in its primitive crude state, enters into a process of loosening up and sloughing, which becomes the medium of its removal from the normal textures. In some instances, and some epidemics, this breaking up manifests itself as a development of the typhous substance, both in the follicular apparatus and in the lymphatic glands, to a luxuriating, bleeding, partially necrosed, fungoid growth (Hensinger's muco-membranous fungus). The latter in particular, offers the greatest analogy with medullary fungus.

The elementary composition of the typhous substance is embryonic—

elementary granules, nucleus-forms. Nucleated cells are commonly present in inconsiderable number. This relates, however, more especially to typhous substance in the bowel. That in the mesenteric glands frequently shows nucleated cells,—even parent-cells with several nuclei.

Even the albuminous constitution of the typhous substance, and the genuine typhous crasis itself, to which fibrinous exudation is a stranger, involve an analogy with medullary carcinoma and its crasis. All fibrinous products occurring in the typhous substance itself, or along with it upon the same textures—the intestinal mucous membrane—or in any other organ, are not proper to the true typhous process, but to a secondary croupous crasis, into which the typhous crasis so often degenerates at various periods of its progress.

VILLOUS CANCER.

An alien growth, whose cancerous nature is incontestably proved, both by its attendant cachexia, and by its frequent alliance with the cancers before discussed. Owing to the close affinity of its elementary structure with that of medullary carcinoma, we place it next in array with, or as a variety of, the latter; with which, moreover, it has in common the loose consistency, the abundant vascularity, and the proclivity, to hemorrhage and to inflammation.

So far as we know, it occurs solely upon membranes, for the most part, the pituitous, and most particularly upon that of the urinary bladder, as so-called villous muco-membranous tumor. It also, although far less frequently, affects the common integuments and serous membranes.

At the outset, it appears as a delicate, cord-like excrescence of various length, which arises out of the aforesaid textures with a seeming longitudinal fibrillation, diverging at its free extremity into branches and twigs. Hereupon, if not before, it forms into delicately membraned villi, and with this expansion of its texture, bulges at its free end into a club-like or cauliflower shape. This section of the excrescence invariably contains a whitish, or reddish white, encephaloid sap. At this point it is particularly vascular, and, in its recent state, of a purple tint.

A minute inspection shows the alien growth to consist of a fibro-membranous texture, densely involuted at the pedicle, and developed at the free extremity into a stroma for the reception of the imbedded encephaloid. This stroma is a delicate, structureless or striated, fine-fibred membrane, studded with elementary granules and nuclei, whilst the encephaloid sap consists of elementary granules, nuclei, and cells of every variety of form. Such excrescences not unfrequently vegetate in great numbers, either scattered or densely grouped, upon the mucous membrane of the bladder, imparting to it a long-drawn, villous aspect,— a condition ascribed by Andral to a preternatural development of the muco-membranous villi.

It is very common for them to vegetate particularly densely on a circumscribed patch, to become blended, at the pedicle and at the expanded points, into a diffuse, roundish head, furnished with a neck, which, if it contains much of the encephaloid juice, presents a uniform, pulpous

consistency, and a superficial lobulation, whilst, in the opposite case, its periphery is villous.

The growth often bleeds spontaneously, and its excessive vulnerability occasions, upon very slight injury, exhausting hemorrhage.

From the above description, the medullary, cancerous nature of the alien growth is manifest, particularly its analogy with that encephaloid, medullary carcinoma, provided with a stroma. It is clearly nothing more than medullary carcinoma with predominant stroma-formation.

EPITHELIAL GROWTHS, EPITHELIAL CANCER.

These growths are without doubt often merely local, and curable by extirpation. In many cases, however, notwithstanding precisely the same morphological and chemical relations, they accord so entirely in all their manifestations with the cancers, that we classify them with these as a further variety of medullary carcinoma, to which in their lineaments, also, they approximate the most nearly.

Their occurrence we believe to be limited to the mucous membranes and the common integuments. We have seen them upon the mucous membrane of the larynx and trachea; of the stomach, the rectum, the urinary bladder; upon and in the common integument, and in the subcutaneous textures of the lips and face; in the scrotum, glans, and prepuce; in the external labia pudendi; upon the skin of the lower extremities. In a parenchyma we have met them but once, namely, in the liver, where they were encysted in a capsule of fibro-cellular tissue.

Upon mucous membranes these alien growths usually appear as rather thickly pedunculated, roundish, cauliflower-like, or warty, leaf-like, stella-clavate, whitish, reddish-white, purple, vascularized, sometimes tolerably firm, often flabby, very vulnerable tumors, easily rent asunder by compression. Upon the common integument they sometimes form similar, now and then tolerably voluminous, tumors. More frequently, however, the alien growth appears as a diffuse degeneration of the skin, which presents a warty, foliated surface, overgrown with luxuriating papillæ, or else, under different structural relations of the new growth, a gland-like, sore, whitish-red, or red patch, which, under sloughing and offthrowing of the alien growth, degenerates into one or several ridge-bound ulcers.

A more minute examination shows these out-growths to consist altogether of cells, which have hitherto seemed to us perfectly analogous, both in themselves and in their development, with the epidermidal or the greater epithelial cells of the tessellated structure. The mature cells are often of colossal size, flattened, mostly rhomboidal, furnished with one or two oval, reddish, or yellowish-red nuclei. The younger cells are smaller, roundish, spherical, limpid, or, around the nucleus, granulated in the figure of a sharply defined areola; whilst roundish, pale-red nuclei are present at their side. The older cells are of scale-like flatness,—their nuclei indistinct, or, it may be, completely obliterated.

In ulterior development the cell does not surpass—

(a.) A lengthening in one direction, with transformation to a rhomb or to a riband-like layer terminating at both ends in a short apex.

(*b.*) A parent-cell, within which occurs a second generation of cells, a development indicative of an alveolar disposition in the other surrounding elements.

These elements are held together by a very scanty, imperceptible, intercellular substance, and give way under moderate pressure, or without this, under the influence of acetic acid, or of other acids which serve to dissolve the intercellular substance.

The cells themselves manifest towards acetic acid relations varying with their age, the older ones not being changed, the younger ones becoming more transparent and gradually dissolved by it, whilst the nuclei are brought more distinctly into relief. When rubbed up with water they impart to it a whitish turbidness, and the young cells lend to their laxer bond-substance an encephaloid aspect.

The secondary arrangement of these elements is very remarkable. It consists:

(*a.*) In their arraying themselves in warty, or warty layer-like growths.

(*b.*) In their arraying themselves in cylindrical or facetted fibres or cylinders, which, gathered together into fasciculi, give the new growth a fibred structure, a fibrous torn surface.

(*c.*) In alveolar order. Elongated cells of the secondary form above specified, course around circular gaps in which are impacted a brood of younger nucleated cells, either spherical, or, when very numerous, mutually compressed into polygonal shapes.

In the larynx, this formation constitutes the out-growths denominated by Albers warty, laryngeal tumors; many lax, succulent, seemingly fibrous, for the most part very sensitive, integumental, and subintegumental warts, a large proportion of cancers of the lip, scrotal or chimney-sweeper's cancer, a not uncommon condyloma-like degeneration of the glans penis, cancer of the external sexual organs in the female, and especially of the external labia. Many of these, more particularly cancers of the lips, have a seeming glandular texture determined by the alveolar type. From the common integument they assail subcutaneous textures without distinction,—even bone; from mucous membranes, the submucous textures: at the larynx, the arytenoid cartilages so commonly that one is induced to believe that the alien substance may in some cases originate with these.

Epidermidal cancer ulcerates, in the sequel of inflammation, in a form identical to all appearance with that of the most exquisite cancer. The base of the ulcer is invested with a yellowish-white, or a white, cream-like exudate, consisting mostly of lustrous, reddish nuclei. Lastly, to this alien growth is to be reckoned, without doubt, an ulcer developed out of a wart-like, transparent, hardish protuberance, in form thoroughly identical with ulcerating cancer, and not unfrequently seen to attack aged persons in the face. The base and edges of this ulcer consist of round, lustrous, reddish nuclei in an amorphous bond-mass, and the white, creamy exudate investing the ulcer reveals the same composition. It represents embryonic stages of epithelial cancer. Certain epidermidal cancers of the lip are similarly constituted.

CARCINOMA FASCICULATUM.

(*Johannes Müller.*)

Formerly termed, also by Johannes Müller, carcinoma hyalinum, because of its jelly-like transparency. An alien growth, according to our observation, of very rare occurrence, which we have met with but twice; once in the mammary gland, and once again in most of the internal organs simultaneously, as almost general cancer.

The first case, which we had better means of examining, relates to a growth of considerable size, nearly that of an infant's head, of uneven, clavate surface, of a pale-yellow color, of jelly-like transparency, and withal, of notable compactness. It consisted of an aggregate of tubera, which resolved themselves into a certain number of cones, flat-sided from reciprocal compression, with their notched and ruffled bases directed outwardly, and their apices pointing to within, so that the apices of all the cones constituting a tuber converged to a common centre. The intersection between the individual tubers was occupied by a somewhat more substantial,—that between the cones by a more delicate,—membranous, whitish, areolar tissue-like bond-mass. Bloodvessels, so far as they could be traced in a not highly injected condition, ran in a direction parallel to the cones. A microscopic examination showed the parenchyma to consist of somewhat long-drawn, delicate, hyaline fibres, between which, in an almost limpid juice, lay imbedded elementary granules, nuclei, and a few scattered, elongated cells.

Without conforming to Müller's description in what concerns the presence of embryonic elements, this growth accords with it, nevertheless, so fully in other respects, as to justify us in pronouncing it to be a true specimen of carcinoma fasciculatum or hyalinum.

In the other case, the secondary arrangement of the large conical fasciculi was less orderly, and, throughout, that before depicted, the consistence more lax, the transparency the same. This latter, according to Müller, is inconstant, and it was for this reason that he afterwards substituted for carcinoma hyalinum, the appellation of carcinoma fasciculatum.

The specimens examined by Johannes Müller were of a consistency analogous to that of encephaloid. He admits, however, that in this respect variations may occur, and that firmer specimens of carcinoma fasciculatum are probably to be met with.

CYSTO-CARCINOMA.

Cysto-carcinoma specially affects certain organs, as the ovary, the mammary gland, the testicles, bones. It is mostly a growth of considerable magnitude, and commonly concurrent with cancer in other organs. [*See Cyst and Alveolus.*]

APPENDIX.

[A careful examination instituted by the author in sundry cancerous tumors, more especially of the medullary character, have led to interesting results illustrative of the development and the microscopic structure

of these malignant growths. Without dragging the reader through the details of cases which seem only to represent so many stages of development,—so many links in the chain of evidence,—we shall endeavor to sum up the results in as few words as possible.

Under a magnifying power of 90 diameters, the substance of fungus hæmatodes exhibits a stroma consisting of two distinct webs, which appear to interlace each other in all directions. Of these, the one has the semblance of a transparent trelliswork, studded with caudate cells, elongate nuclei, and long-drawn fibres, all lying parallel to the longitudinal axis of the stroma. The gaps or meshes of this stroma are interlaced or enwreathed with what at first appears like a continuous garland of leaves, but on a closer inspection is seen to terminate in bulb-shaped extremities. Further examination shows this wreath-like tissue, which at first seemed opaque and granular, to be studded with crowds of minute nucleated cells, which, under a magnifying power of 400, are distinctly set forth as round or oval cells, many, although not all, containing one or several nuclei, others engaged in the act of elongation, others again in progress of dissilience.

In preparations representing a further stage of development, the wreath-like tissue presents certain patches much less opaque, its cells for the most part elongated, and many of its nuclei drawn out into disconnected fibres. There is good reason for regarding this portion as in a state of transition from the wreath-like tissue to the supporting stroma first described. In the next, and last, phase of development, is represented the same trelliswork, no longer thinly fibred and semitransparent, but rendered opaque by connected and dense longitudinal fibrillation. These fibred trellises are here distinctly seen to be enveloped in a hyaline structureless membrane, not closely fitting, but loose and projecting on all sides into the fenestrate gaps in conical and bulb-like excrescences. It is remarkable, that from the first period of their fibrillation, these trellis branches are observed to constitute hollow cylinders. This may be owing either to a single cell-layer being alone present within the excrescences, or else to the fact, that of a cell-mass with which the excrescence is replete, only one layer becomes fibrillated and the rest absorbed.

Although the proof is difficult, there is good reason for believing that the hollow cylinders referred to are filled with the same cancerous substance that furnishes the outer material for the excrescences.

Certain external features, analogous with the above, induced Rokitansky to submit to a close investigation those adventitious membranes upon serous tunics, which present, with a honeycombed aspect, a free-villous surface. The process of development resembles that of the cancerous growths, only that in these pseudo-membranous formations the wreath-like tissue more frequently occurs in layers parallel to the gaps or open spaces of the primitive fenestrate layer, or in superimposed order and sometimes in thick masses, tufted with many prominent, short-necked, terminal bulbs. It will be seen, from Rokitansky's great "Essay on Diseases of the Arteries," that the intra-arterial, superimposed layers of coagula present very nearly the same structural development. (See "Die Entwickelung der Krebsgerüste," from the "Sitzungsberichtie der

math-naturw. Classe der Kais. Akademie der Wissenschaften," Marz, 1852.)

In a subsequent essay on villous cancer (April, 1852), and a third on colloid cancer (July, 1852), Rokitansky has made it apparent that, with certain modifications contingent upon the general conformation of the tumors and upon the nature of their contents, the same general relations of structure pertain to these cancers likewise.

In the *colloid cancer* there is a similar formation of a multilocular stroma, which, however, often assumes rather a *membranous* fabric. This honeycombed structure contains within its cancelli, the colloid or gelatinous mass, which is for the most part connected together, so as in a manner to interlace with the said stroma, and only here and there to occur in shut sacs or cystoids, formed through the blending of the membranous framework. Rokitansky has obtained evidence, that from the walls of these shut spaces, bulbous forms arise, and that the colloid globules are formed within these, as the product of a hyaline blastema with which they are more or less replete. He seems to infer that the fibro-membranous stroma is itself but a development out of primitive hollow bulbs.

There is in this theory respecting the aforesaid formation of the encysted masses of colloid, a general withdrawal by the author of one opinion expressed in the section on cysts, namely, that in these new growths the cyst is invariably developed out of the structureless vesicle. The term *cystoid* would therefore be peculiarly applicable to the membranaceous cavities found in colloid cancer, as distinguishing them from genuine cysts.

Villous Cancer.—In all but its external form, this cancer approaches the nearest to medullary carcinoma. A very important part is here assigned to the dendritic excrescences, into which the primitive hollow bulbs, often springing from a densely reticulate germ, speedily resolve themselves; the first shoots pushing forth from their terminal bulbs secondary offshoots in the shape of slender villi, which themselves expand into bulbs, and throw out more of these embryonic excrescences from their termination, so as to constitute by degrees a more or less extensive cauliflower- or coral-shaped tumor. In other cases, a single stem arises out of a nucleus as big, it may be, as a bean, and this stem branches out into dendritic vegetations of the character above described.

Most of these excrescences end in cæcal sacs, some of which may contain a structureless, or a concentrically stratified cyst.

These excrescences are often transparent, containing in their cavity only a clear fluid, whilst, externally, they grow up, as it were, into a more or less tenacious plastic mass, consisting of the same elements that compose the sap of medullary cancer. In other instances, they include a fibrous texture, within which reside elements similar to those that cling to them externally. A remarkable circumstance connected with these excrescences is the peculiar way in which they are vascularized. Both the stem of the tumor and all its individual excrescences are furnished with an ascending and descending bloodvessel, which pursues its course under the formation of frequent loops. These bloodvessels consist mostly

of the primitive hyaline bloodvessel membrane, marked with oblong
nuclei, sometimes also with a row of transverse oval nuclei. There may
possibly be a further layer of connective tissue fibrils. A few of the
excrescences have but a single ascending bloodvessel, terminating in a
sort of bulb.

In rare instances, a nest of apparent excrescences displays open ter-
minations fringed with villi, and filled with the semifluid materials of
medullary cancer. Rokitansky is, however, of opinion, that these are
not true excrescences, but rather lengthy developments of the fibro-
cellular texture which constitutes the base of the tumor; and he believes
these hollow cylinders, which seem rather to resemble the honeycomb of
the wasp, to become filled, not by endogenous secretion, but by suction
of the external medullary fluid.

Seat of Villous Cancer.—Its seat is 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 cysto-carcinoma, where it is recognized as villous cancer, from
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 upon the dura mater, occasionally upon the
general integument (Rokitansky refers to two such cases), and even in
bone,—reckoning for villous cancer those cases in which a bony skeleton
is found in the shape of the wasp's honeycomb structure before described.

Lastly, it occurs in parenchymata, in the uterus, for example; and,
as cancer melanodes, in the liver and in the brain.

It occurs both as a single tumor, and also concurrently with cancer
of various kinds in other organs,—occasionally germinating out of those
broad-based, fungus-like gelatinous cancer-masses that occur upon the
inner surface of the stomach.

"The vascularity of villous cancer determines a predominant feature
in its course, whether upon membranous surfaces, in the interior of
cysts, or in parenchymata, namely, the frequent hemorrhage which so
greatly hastens the general wasting and the fatal issue. Frequent and
excessive hemorrhage from the urethra in males, from the vagina in
females, furnishes strong suspicion of villous cancer affecting respec-
tively the bladder or the uterus, whilst a microscopic examination of the
blood effused will often bring to light shreds or fragments of the cancer-
ous mass."

The same vascularity often causes a fleshy coloration of the tumor.

It is evident from the foregoing, that villous cancer is, to all intents
and purposes, a malignant *new growth:* and not, as Andral and Louis
have affirmed, an anomalous development of muco-membranous villi;
nor, as others have more recently suggested, a tumor arising out of the
hypertrophy of a pre-existent papilla.]

TUBERCLE.—TUBERCULOSIS.

The collective term "tubercle" is made to embrace sundry formations, which have nothing in common beyond their outward form.

Still, after having well sifted this side of the question, we shall ourselves feel bound to comprise under "tubercle," formations in external appearance quite dissimilar to what is commonly called tubercle, nevertheless essentially identical with it; for instance, the primitively yellow, fibrino-croupous tubercle.

If we except the rare instances in which it represents an endogenous deposition within the circulating system, *tubercle* is in the broadest sense an *exudate*—an exudate of solidified protein substances (fibrin, albumen), which as blastema persists at the lowest grade of development; that is to say, in the primitive crude condition determined by its consolidation. It thus occupies the point of transition to the non-organized new growths.

This last attribute is essential and indispensable, imparting to solid blastema the impress of tubercle. It is so important, that every blastema, however much its characters may assimilate to tubercle in other respects, loses the distinctive mark the moment it enters upon a transformation of texture.

This exudate (in its broadest sense) is for the most part distinguished by the tubercle-form; that is, by its appearance as scattered or collected nodules, or where more copiously produced, by its deposition in granulations and stellate masses. It is hereby cognizable at the first glance. Still this is open to exceptions.

Gelatinous and fibrous cancer appear now and then in a tubercle-like form; that is, in the form of little discrete nodules or stellate bodies; and, upon serous membranes, the peritoneum, for example, there occur granular exudates of fibroid and areolar tissues. These are distinguishable from tubercle by their texture.

But, again, even tubercle itself occurs in extensive, irregular masses. There are inflammatory products endowed with an indwelling tuberculous character, although manifesting a total absence of the external habitudes of tubercle.

Tubercle has therefore sometimes a local, but far more frequently a general import and significance. It is invariably so closely linked with dyscrasial processes, that, for a profitable consideration of tubercle, an incessant retrospect to the dyscrasial relations is imperatively demanded.

Nevertheless, the basis and starting-point for an anatomical inquiry concerning tubercle itself, must in our opinion still be the aforesaid *fixed blastema abiding at its primitive stage of crudity.*

In this sense tubercle offers sundry distinctions, some obvious and essential, others less marked. They relate to its color and lustre, its transparence, its consistence, its elementary fabric, chemical composition, &c. These are characters referable to more or less manifest special crasial relations—modifications of a fundamental tubercle-crasis. They determine several, and some of them essential, forms of tubercle,

which we shall proceed at once to portray, selecting for our basis the purest possible forms.

(*a.*) *Simple fibrinous tubercle* appears as scattered or stellate conglomerations of granules of about the size of millet-seeds. It presents, moreover, as the product of inflammation upon serous membranes, smooth pseudo-membranous exudates, as we often find exemplified upon the pleura of lungs involved in florid phthisis.

In the first-known form this tubercle represents the *gray semi-transparent granulations* of Laennec.

The investigations and theories hitherto instituted relate almost exclusively to this tubercle, from which all other tubercle-formations have been derived as from a stereotype basis.

The question of old—What is tubercle? must at this day be changed into—What is this particular tubercle?

In its early stage, at which acutely generated tubercle is often enough to be obtained in the human subject, it appears in the form of the aforesaid granulation,—to the naked eye a roundish, resistant, solid nodule, of about the size of a millet-seed. Not unfrequently, however, we encounter amongst them tubercles somewhat smaller, and representing a less firm, a softer, at the same time more transparent, almost vesicle-like granule.

Nevertheless, however much tubercle may at a first glance wear a vesicular appearance, it invariably originates as a solid corpuscle; and the results of a careful analysis of this substance, as well as its very nature and import, serve to corroborate this fact.

Minutely examined, it only seemingly represents a spherical body. Under a moderate magnifying power,—nay even on a narrow inspection with the naked eye, it is seen at its circumference to branch out more or less. With the textures it is only in so far connected as to lodge betwixt their elementary parts, to take up some of these into its substance, and—what is especially discoverable in tubercle upon serous membranes,—to adhere to them by dint of an indwelling tenacious property. It represents a tolerably homogeneous—now toughish, gritty, fibro-granular, fragile, now softish, uniformly compressible—substance, in various shades and modifications, of a pearly gray color.

Under the microscope it reveals the following elementary composition:

It consists mainly of a more or less pellucid base (blastema), which affords a sort of binding medium for certain form elements. Its components therefore are—

1. The said basement-mass,—for the most part a fibro-glebous, gray, fixed blastema, rendered turgescent and transparent by acetic acid.

2. Certain embryonic form-elements, namely:

(*a.*) Elementary granules of various magnitude.

(*b.*) Nucleus formations, both black-contoured, lustrous, spherical, even oblong nuclei,—and more delicate, dull, granulated nuclei, under various phases.

(*c.*) Nucleated cells; commonly in such small numbers as to tempt one to doubt their occurrence altogether. Nuclei and cells are often to

a great extent misshapen, disorderly, jagged, angular, bulging, dumb-bell-shaped, rudimental, stunted.

Along with this, the tubercle is wont to include various elements appertaining to the textures in which it nestles. The tubercle purest in this respect is that upon serous membranes, which, therefore, like many other new growths upon serous membranes, is the best adapted for examination. Nay, tubercle will even take up and incorporate compound textural constituents, and in particular bloodvessels. The question here suggested as to tubercle-containing vessels of its own will be discussed hereafter.

The metamorphosis which this tubercle undergoes, is limited to *decadence*. After abiding in the primitive, *crude* condition before described it becomes transformed, with the loss of its moisture,—with condensation —to a hard nodule, and shrivels, into a tough, amorphous or indistinctly fibrous, horn-like mass,—in a word, *cornifies*. This determines a complete wasting and death of the tubercle, subversive of all further change. Occasionally this process is associated with bony deposition, the tubercle becoming a partly ossified nodule.

This tubercle does not undergo any other metamorphosis independently. Every other change suffered by it is based upon a combination of its blastema with another, and its *softening* in particular, upon a combination with the ensuing tubercle, namely, the fibrino-croupous. This *softening* process plays so momentous a part in the doctrine of tubercle, that we deem it right to declare emphatically our dissent from the opinion that gray tubercle, the gray tuberculous granulation of Laennec, softens.

Fibrino-Croupous Tubercle appears in the shape of roundish nodules, as also, and that very frequently, of irregular, gibbous, branched masses of considerable diameter, or, upon free surfaces as gibbo-stellate layers of various thickness. The nodules in size often equal the gray tubercle granulations, still oftener do they equal hemp-seed or peas. Usually, every variety of size coexists. The substance of this tubercle is, as we may here once for all remark,—opaque from the very first, now resplendent, in various degrees, yellow, of fibrous or of granular fracture, firmly elastic, or friable, of a lardaceous, curd-like aspect. We distinguish it from the gray tubercle by the designation of *yellow tubercle*. It most probably constitues the pyin-holding tubercle.

The microscopic examination of this tubercle shows, as in the case of the foregoing one, a fixed base, and the aforesaid form-elements. The former is a fibro-glebous, or else an amorphous, opaque blastema. With respect to the latter much variety obtains. The number of cells, of nuclei, especially of the dull, granulated nuclei, of the elementary granules, and especially the quantity of the finest point-molecule predominate.

The metamorphosis proper to this tubercle is *softening*, and again *cretefaction*.

1. The first, namely *softening*, also termed suppuration, consists in this: after the tubercle has tarried for a certain time in the above-described condition of crudity, it loosens up,—for the most part with considerable increase of volume, readily breaks asunder through compres-

sion, moistens. Hereupon it changes into a yellowish, glutinous, fatty, tenacious substance, like melted cheese, and eventually liquefies to a thin, whey-like fluid of acid reaction, wherein flocculent and fragmentary particles, the remnants of tubercle imperfectly broken up, float as tubercle-pus.

In the larger tubercle masses there is often observable, during the said process, a cleft formation on a large scale; or, where the tubercle is spread out in a layer, a fissuring of this latter.

With regard to the elementary character of the tubercle at this stage, we would observe:

The softening consists in a liquefaction and breaking up of the solidified base of the tubercle to a fluid loaded with point-molecule. This transformation results in a separation or isolation of the form-elements of the tubercle, which at the same time undergo within the fluid a more or less marked change. Thus, the cells become turgescent, corroded, dissolved; the nuclei shrivelled and misshapen, irregularly angular, pouched, &c. At length free fat becomes developed in the softened tubercle.

Hence the *liquefied tubercle* consists:

(*a.*) Of a fluid with point-molecule.

(*b.*) Of the isolated nuclei and cells changed in the manner just now specified.

(*c.*) Of free fat in the shape of elementary granules and larger scattered globules.

The softening determines the malignancy of tubercle, leading as we shall presently see to ulcerous destruction of the textures,—*tuberculous phthisis*.

2. The other metamorphosis of this tubercle is *cretefaction*. It never affects the tubercle blastema in its primitive condition, but only in its liquefying or liquefied state.

During the softening process, or after its completion, the tubercle takes up lime-salts and fats, in the shape of free, discrete, or aggregated elementary molecule, or else in granule-cells in the form of big drops and of cholesterine crystals. In this act the softened tubercle is progressively thickened into a moist, unctuous chalk-pap, and eventually converted into a concrete mortar.

Let us now attempt to institute an inquiry respecting the *nature of tubercle*, in its two cardinal forms, as just delineated; whereupon we will proceed to discuss its varieties, its metamorphosis, its local process of deposition, its seat, and, lastly, its relation to the blood-crasis.

In the first place, the ground-work of rapidly solidifying tubercle blastema is, without the least doubt, *fibrin*. Again, in the two cardinal forms of tubercle, it is easy to recognize the two principal forms of fibrin, the *simple* and the *croupous* (*see Fibrin*). Why the former, which we have elsewhere denominated plastic, enters into no textural conversion, why the latter fails to undergo that prompt liquefaction proper to the croupous exudates, are questions which we shall endeavor to reply to in a more appropriate place.

With reference to the varieties of these cardinal forms, we would observe—

(*a.*) Of *croupous tubercle* there occur *several varieties*, together reminding us of croupous fibrin and its resulting exudates. They are determined by opacity, coloration, consistency, tendency to liquefaction, by the corrosive property of their ichor, the proportion of their form-elements, of their point-molecule, and by the character and import of their nucleus and cell-formations.

(*b.*) *Like blastema in general, tubercle blastema is especially unwont to exude pure.* The combination of the two cardinal tubercle-blastemata in different proportions, and their manifold grades of co-ordination and of blending; again, the union of varieties of croupous tubercle with each other, and with organizable blastema (fibrin), break up tubercle into countless varieties.

In like manner, the gray tubercle granulation presents many variations in respect to transparency, coloration, &c., the greenish shade, for example.

A peculiar variety is the *pigmental* tubercle, for the most part *hemorrhagic*, as to its origin.

As metamorphoses of tubercle, we have already been made familiar with its *decadence* or *obsolescence*, its *softening*, and its *cretefaction*. The first is proper to the simply fibrinous, the last two to the fibrino-croupous tubercle. These metamorphoses affect tubercle in common with consolidated blastemata of a certain constitution, whether they occur as exudates (even as extravasate[1]) external to the vascular system, or as endogenous coagula within the bloodvessels. *Their cause is primitively inherent in the tubercle*, conformably with our view respecting the primordial properties of blastemata.

1. The *obsolescence* of tubercle, hitherto disregarded, is cosignificant with its cornification. It implies wasting, extinction of the tubercle.

2. *Softening* of tubercle, a metamorphosis which it enters upon without distinction of volume—resolves itself into that elementary phenomenon, the breaking down of solidified protein substances, and especially of solidified fibrin,—a phenomenon pertaining to this substance only in its determinate croupous constitution. It is proper to fibrino-croupous tubercle *alone*, and is determined by a conversion of the chemical components arising out of an interchange of the elements.

Genuine gray tubercle-granulation never softens. A combination of its blastema with that of fibrino-croupous tubercle alone capacitates it for softening. It was, indeed, formerly taught, that gray tubercle-granulation lost its gloss, its transparency, became opaque, of a yellowish-white or yellow, and ultimately softened and deliquesced. The error probably arose from a readiness, in the frequent cases where the two forms of tubercle are concurrent and even now and then mingle together into a kind of transition link from the one to the other, to take for granted that they represented in reality two different stages of development.

We have, however, a second error to rectify besides. Long ago the softening of tubercle was described as a development,—a progressive metamorphosis,—but in general and not very lucid terms. Present

[1] The term "extravasate" is used by German pathologists in a restricted sense only, namely, to signify the effusion of substantive blood, with *blood-globules*, into surrounding textures,— in other words, internal hemorrhage from ruptured or wounded bloodvessels.

pathology, whilst adopting the older views concerning the softening of
tubercle, is influenced by the microscopic discovery of an incomparably
greater number of nuclei, and especially of cells, in *softened* than in
gray tubercle. They are looked upon as new formations out of the
liquefied tubercle blastema.

We cannot participate in this view. Those elements are not recently
generated out of the liquefied blastema, but proper to the tubercle from
the commencement, and isolated by the softening process. That they
are more numerous in softened tubercle than in the gray, is explained
by the fact, that only that tubercle softens which originally holds them
in abundance, namely, the yellow (croupous) form.

In point of fact, no fluid is less adapted to furnish the blastema for
new growth than the so-called pus of tubercle. The softening of tubercle
takes place sometimes early, sometimes late,—rapidly, or by slow de-
grees. All this depends upon certain peculiarities in the character of
the (croupous) tubercle. In this process it is worthy of note, that in
tubercle masses deposited all at once, the softening proceeds from the
central part; whereas, in aggregate masses thrown out at different
epochs, and perhaps embracing different forms of tubercle, the softening
may commence at any part,—even at the periphery. This fact is
fraught with interest, as corresponding with kindred processes in certain
other morbid products,—for example, the central softening in globular
endocardial vegetations,—in intra-arterial coagula-layers, &c. More-
over, it is important as offering—if at this time of day it be wanting—a
conclusive argument against the assumption of the softening of tubercle
being a process evoked from without through the agency of surrounding
textures. The utter absence, in tubercle, of bloodvessels of its own, the
compression and closure affecting such as penetrate the larger tubercle
masses from without,—the fact that in textures surrounding tubercle
engaged in incipient softening no trace of inflammation is generally dis-
coverable,—that both the latter and suppuration supervene only upon
completed softening of the tubercle,—lastly, the ocular proof that the
softening commences at the point most remote from surrounding textures,
are so many arguments against the assumption referred to, and espe-
cially against that of a mechanical melting down of the tubercle, through
pus thrown out from the inflamed encircling textures.

The sum of these negations is, that the *softening is a spontaneous
metamorphosis essentially proper to the nature of tubercle.*

The softening is that which constitutes (yellow) tubercle a malignant
growth, inasmuch as it commonly leads to that ulcerous destruction of
the textures which represents *tuberculous phthisis.*

The complete solution of a tubercle determines in the implicated par-
enchyma, a gap, generally corresponding to the tubercle in size, replete
with so-called tubercle-pus. The parenchyma has suffered a loss of sub-
stance to the extent only of the texture particles which happen to have
been involved in the tubercle, and have now perished in the tubercle-pus.
This gap represents the *primitive tuberculous cavity* within a paren-
chyma. The contact of the tubercle-pus with the surrounding textures,
occasions a corrosion of the latter. The moderate enlargement of the
primitive cavity thus engendered, is substituted, upon membranous ex-

pansions, the mucous coats for instance, by a deepening destruction of the tissues; that is, of the inner stratum of the mucous membrane. This manifests itself as a millet- or hemp-seed-sized ulcer, which, to distinguish it from the different form arising from consecutive enlargement, has been designated as the *primitive tubercle-ulcer*.

The consumption of textures would here remain inconsiderable, but for the breaking down of fresh tubercle in the proximity of the original ulcer. Inflammation here plays an important part.

(*a*.) This production of fresh tubercle in the vicinity of that softened, and of the resulting primitive cavity,—at the margin and base of the primitive tubercle-ulcer, upon superficial expansions,—determines the enlargement of the ulcer in all directions,—the textures becoming again and again corroded and necrosed by the fresh softening tubercle. And this takes place with a rapidity proportionate to that of the softening of the secondary tubercle—the product of an exalted cachexia. Another accidental mode of enlargement of the ulcer consists in two or more ulcers, already advanced in the way described beyond the primitive condition, merging in a single one. The result is an ulcer marked by its irregular, indented form,—upon mucous membranes, by serrate, jagged edges,—in muco-membranous canals by its affecting the girdle shape. The manner of its development, and its characteristic form, so different, especially on mucous membranes, from that of the primitive ulcer, fairly entitle it to the appellation of *secondary tubercle ulcer*.

The destruction of textures involved in this process, as corrosion and necrosis through contact with tubercle-pus, constitutes the *tuberculous phthisis* of organs. These are either acute or chronic.

(*b*.) *Inflammation* enters, as we shall see by and by, into various relations to the phthisical process. What we have here, however, particularly to remark upon is in how far it contributes to the enlargement of the tubercle-ulcer and to the modification of its character. In the *first place*, it determines, for the most part, yellow tuberculous products, in the form of infiltration, which, conformably with the aggravated dyscrasis, rapidly break up, extensively corroding and destroying the textures involved. In this way inflammatory action occasions an ominous enlargement of the tuberculous ulcer, and the most widely spread tuberculous ulceration in an acute form.

In the *second place* it engenders organizable, solidifiable, fibrino- or albumino-gelatinous products, which pass into a fibroid callus. Thus arises the callous condensation of the textures encircling the cavity; in the muco-membranous tubercle-ulcer (for example, in the bowel) the hardish elevated brink and the funnel-shape of the *primitive*; lastly, the jelly-like infiltration and induration at the base and margin of the *secondary* ulcer.

These products exude, according to circumstances, either pure, or almost pure, or combined together in various proportions. In worn-out individuals, the inflammation, if present at all, furnishes forth thin, albumino-serous products, and the tubercle-ulcer is of a lax and torpid character. In the proximity of cavities seated in the midst of tuberculous infiltration, there is of course no inflammation.

3. *Cretefaction*, as already stated, affects fibrino-croupous tubercle

after it has entered into the softening process. It is co-significant with the cretefaction of fluid blastemata, and analogous with the cretefaction of broken-down fibrin in the vegetations and coagula within the vascular system, in croupous exudates upon serous membranes, and in parenchymata; and again in the cretefaction of pus.

The basis of this (secondary) metamorphosis is as little to be sought extraneously to tubercle as the softening itself. Nevertheless, the surrounding textures may contribute, by their absorbing agency, to the inspissation of tubercle-blastema. What cornification is to the *gray*, cretefaction is to the *yellow* tubercle, namely a process of involution.

Cretefied tubercle resides, for the most part, within textures isolated by products of inflammation, entering into a fibroid transformation and then cornifying and shrivelling into a callous capsule. Both together draw down upon themselves the surrounding textures in scar-like corrugations.

Such are the metamorphoses of genuine tubercle of the one and the other form. There occur, however, *complicated metamorphoses* corresponding to various combinations of the different tubercle-blastema. Thus:

(*a.*) The combination of gray with yellow tubercle is frequent. Where, in this combination, the latter passes into softening, the gray tubercle, like textures in contact with tubercle-pus, becomes destroyed. Where the softened yellow tubercle cretefies before this destruction of the gray is effected, the latter cornifies independently; and if it happen to be peripherous to the other it encircles the cretefied tubercle with a sheath of gray cornified tubercle, differing from that callous exudate-capsule which results from inflammation of the surrounding textures.

(*b.*) Just as tubercle blastemata combine with one another, so, in like manner, does organizable blastema enter occasionally into combination with tubercle. Its existence is of course scarcely demonstrable in yellow tubercle, in the metamorphosis of which it becomes itself destroyed. If cretefaction set in early, it may become organized so as not to be easily distinguished from a subsequently effused blastema, the product of inflammation.

The combination of gray tubercle with organizable fibrin is more susceptible of proof. The instances are not rare in which, hard by pure gray tubercle, granulations are found in which one portion of their blastema is in progress of organization to a fibrous texture, whilst the other abides in its primitive condition, and eventually falls into decadence—cornifies.

There are, indeed, as we shall presently have to show, granular tubercles resulting from inflammation upon serous membranes,—that is, solidifying exudates or granulations as big as poppy-seeds or millet-grains, which in their entirety change into fibroid textures—into areolar tissue. They occur, along with blastemata, consolidated into pseudo-membranous areolar tissue, or along with gray tubercle, or even with both gray and yellow, softening tubercle.

The organizing of these granulations consists in the development of a more or less determinate fibrous texture. They acquire the whiteness, the resiliency and elasticity, the fibrous-torn surface, the general charac-

ters of little fibrous tumors; or else they change into velvet- or felt-like fasciculi of connective tissue.

They are found upon the peritoneum, especially of the liver and spleen, as also occasionally upon other serous membranes. The Pacchionian granulations upon the arachnoid, the granulations upon the investment of the ventricles of the brain, are upon the whole of the same character.

It is intelligible, from hence, in how far, and in what sense, we are warranted in speaking of a textural conversion, an organization of tubercle, as a metamorphosis of this alien growth. It is intelligible, namely, that growths, whatever resemblance they may bear to tubercle, lose their import as such, in other words, reveal their non-tuberculous character, with the slightest textural conversion.

Intimately connected with the above is the question as to *whether tubercle contains bloodvessels of its own?* The question may belong rather to a bygone day. It is for the present day, however, to set this point at rest for all time !

Vascularity, in truth, belongs as little to the nature of tubercle as organization itself. Still it is undeniable that bloodvessels are sometimes met with in tubercles. Two cases are here possible. In the first case bloodvessels may appertain to textures which, whether normal or pathological,—membranaceous areolar tissue, for instance,—had become involved in the tubercle when first thrown out. One or more bloodvessels may traverse the tubercle, previous to an injecting mass,—others impermeable.

In the other case the bloodvessels penetrating the tubercle are doubtless new-formed vessels, and have sprung from an organizable blastema, effused together with the tubercle and incorporated in it. This is attested more especially upon serous membranes, where, as a consequence of inflammation, tubercle becomes deposited along with a considerable portion of blastema, the rudiment of vascularized new textures.

To sum up: the *purer the tubercle, the more certain is its estrangement from all bloodvessel formation.* The less pure, that is, the more organizable blastema it has incorporated, or consociated with, the more susceptible is it of bloodvessel formation.

We have hitherto spoken of tubercle as being an exudate,—a secretion from the vascular system; of which we hardly deem it requisite to furnish proofs. Here, however, a double question suggests itself, namely, first, concerning the *seat of tubercle;* and, secondly, concerning a very weighty point, namely, the *local process of tubercle production*.

The *seat of tubercle*, as exudate, is at any point of any texture, extraneous to the bloodvessels. Wherever there is a capillary range, a deposition of tubercle is possible. The seat of tubercle is without doubt precisely, or at least in close proximity to, the spot of its exudation, its blastema being in the highest degree coagulable. It is most probably for this reason that it does not affect textures nourished from a distance by a slow imbibition of their substance with plasma,—for example, cartilage. We can ourselves testify to the occurrence, both in larger bloodvessels and in the capillaries (as depôts or metastases), of coagula obviously of a tuberculous nature. These are, however, only excep-

tional cases, and the doctrine propounded in accordance with them is founded rather upon the results of so-called tubercle created by artificial injection. It is evident, however, that the tubercle-like depôts so formed were due either to infection of the blood, or to the obstruction of blood-vessels, and that no inference can be drawn from them as to the spontaneous formation of tubercle.

Assuming, therefore, tubercle to be an exudate—an effusion out of the vascular system—the question as to the topical process would resolve itself into this: what are the processes in whose sequel tubercle is thrown off from the circulation? To this we can only reply, that tubercle, like other blastemata, exudes, now almost insensibly in the act of nutrition; then, again, in the sequel to obvious (active) hyperæmia; and lastly, as a consequence of still more manifest inflammation.

1. It is a fact that the incipient production of tubercle takes place, within some organ,—most commonly the lung, and at one particular portion of it, the point de départ, so to term it, of tuberculosis,—in a manner almost imperceptible both to the patient and to the looker-on. The after-death examination reveals no inflammation, or such only as may with far greater probability be interpreted as consecutive. The tubercle is for the most part the gray, withering, and only now and then the yellow, softening, cretefying tubercle.

2. In other cases, on the contrary, a marked hyperæmia of the implicated organs manifests itself during life, and is discoverable after death, as the source of the tuberculous exudation. The tubercle is deposited numerously, and also rapidly.

(*a.*) This tubercle has commonly the form of those scattered granulations, of about the size of millet-seeds, and seldom that of yellow hemp-seed- or pea-sized nodules.

(*b.*) Its blastema is commonly that of the gray tubercle, often combined with that of the yellow; rarely the yellow alone.

(*c.*) Not only is it thrown out rapidly and in great numbers, either all at once, or in successive outbreaks repeated at short intervals; but it scarcely ever restricts itself to a single organ, and whilst seemingly perhaps concentrating its main forces upon some one organ, it assails several others simultaneously, often leaving hardly any of the soft parenchymata unscathed. The tubercles are marked by their uniform size and character, and by the equable distribution with which they are scattered throughout the textures. After their repeated and copious exudation, they gradually become less firm, softer, glutinous, until the fibrinous tubercle—the fibrin being expended—changes into the albuminous tubercle.

(*d.*) The effusion of the tubercle as a coagulable blastema is always associated with that of a non-coagulable or less coagulable, serous, sero-albuminous, jelly-like, adhesive product,—as a sort of vehicle for the first. The textures are manifestly congested; and, around the tubercles, more or less uniformly infiltrated with the product just referred to.

(*e.*) The more generally and more rapidly the tubercle-production extends through the organism, the greater the multitude of tubercles, the more marked those dyscrasial appearances wrought by defibrination of the blood,—by so much the more fully does the general disease partake

of the acute character. As the expression of that defibrination, the blood appears thin and watery, the attenuated blood-serum, tinged with appropriated hæmatin, being thrown out upon and coloring the imbibed textures, which, if highly vascular, appear lax, flabby, and drenched.

(*f.*) As this tuberculosis for the most part proves quickly fatal, a metamorphosis of the tubercle is proportionately seldom observable.

(*g.*) Rare instances excepted, this tubercle is not the primitive one. Tubercle has commonly pre-existed, whether in an advanced state, or retrograding to decadence, in some organ or other; for instance, the lungs or the lymphatic glands.

3. In fine, *tubercle is frequently thrown out in the sequel of inflammation.* It is the product of such inflammation, and its sole distinguishing feature. These inflammations occur in every part, but more particularly upon mucous membranes, and in the larger serous sacs, where they may be most advantageously studied. Thus they are seen especially upon the peritoneum and pericardium, and again upon the mucous membrane of the uterus, of the tubæ, and of other ducts, as the vas deferens, the seminal vesicles, the ureters. They very frequently affect glandular hollow formations—the pulmonary cells, as pneumonia, the follicles of the intestinal mucous membrane,—almost equally often the parenchyma of the lymphatic glands, fungoid bones or sections of bones, &c.

The exudate offers in regard to its so-called *tuberculization*, certain points of interest, as observed most particularly upon serous tunics.

(*a.*) In the first place, only a portion of the entire exudate appears as tubercle, whilst the remainder becomes gradually reabsorbed and disappears. Or else this latter changes into a texture,—to areolar tissue, to a fibroid vascularized texture, often to a redundantly vascular, fibro-cellular new growth, or to a spurious membrane of similar structure. The two allotments may be present in very different quantitative proportions, the one or the other predominating in various measure. The more texture-formation prevails, the more does the tuberculizing portion take the form of scattered miliary granulations, up to more voluminous nodules imbedded in the organized new growth. The more scant the textural formation, the more prone is the tuberculizing portion to represent a confluent, uniform, granulating, stellate, clavate tubercle-mass or layer.

The tubercularizing new growths, as vascularized pseudo-membranes, often become themselves the seat of inflammation, for the most part productive of hemorrhagic exudates of a tuberculous nature.

(*b.*) In the second case, the entire solid exudate, remarkable for its bulk, is tubercle; or the organizable portion is imperceptibly small, and disappears. It forms in considerable, irregular, shapeless masses; or, on membranous expansions, smooth, or stellate, stella-clavate layers.

The tubercle thrown out as a consequence of inflammation, is the gray, or it may be the yellow, or again a combination of both. The fibrino-croupous yellow tubercle is especially often the product of inflammation, and especially marked by its abundance. It occurs everywhere, constituting, upon membranous formations, the aforesaid stellate layers; within muco-membranous canals and cavities of inconsiderable calibre, as, for example, the uterus, the tubæ, the seminal vesicles,—thoroughly

closing plugs; in compact parenchymata,—lesser or greater, roundish or irregular knobs; in the lung-cells and in follicles,—smaller coagula.

Tubercle produced by inflammation generally passes speedily into softening, and thus to a phthisis of the textures, marked by the acuteness of its course; and, as pneumonic tubercle infiltration, by the jagged, eroded look of the cavities; lastly, by the not unfrequent supervention of pulmonary gangrene.

Inflammatory tuberculosis, like the foregoing species, is rarely primary. It generally accedes to antecedent, insensibly generated tubercle, invading either the organ already diseased, or a structure intimately connected with it. Thus pneumonic tubercle and tuberculous pleurisy associate themselves to pulmonary phthisis; tuberculous peritonitis, to abdominal tuberculosis of the lymphatic glands. Or else tuberculoses become consecutively developed according to the same scheme, each fresh one bearing more and more decidedly the impress of its inflammatory origin.

The ordinary succession of the different fibrin-tubercles, and their different modes of exudation, partly deducible from the preceding statements, are highly interesting. Generally speaking, the gray tubercle, insensibly effused into an organ, leads the way. The yellow tubercle is less frequently the primitive one. Nor is the hyperæmic or the inflammatory oftener the primary source of the effusion in either case. Upon the primitive, insensible deposition of gray tubercle follows, with increasing dyscrasis, the exudation of combined gray and yellow tubercle, the yellow progressively assuming the ascendant, until it ultimately exudes alone. Step by step the exudatory process becomes more and more acute in character; hyperæmia, inflammation, and, at the same time, the quantity of tubercle thrown out, more and more pronounced.

Under certain conditions there exudes, in the sequel of inflammation, more particularly in a new growth naturally prone to tuberculization (pseudo-membranes upon serous tunics), a tubercle reddened and pigmented by adherent hæmatin and embodied blood-corpuscles. It might be suitably denominated the pigmented or hemorrhagic tubercle.

The increment of tubercle, as a consolidated, non-vascular exudate abiding in its rude, primitive condition, can only take place through adjacency and blending with a mass recently exuded in its immediate vicinity. It is doubtless thus that many bulky, lobulated, stellate masses have become aggregated out of individual tubercles, dating from various periods.

But, whereupon does it depend that the product of the said processes, and, in particular, that the product of inflammation, in other cases reabsorbed or transformed into textures, is here precisely *tubercle?*

We do not consider the standing explanation of this phenomenon, namely, of the persistence of the exudate in its primitive rude state, satisfactory. It is to this effect:

(*a.*) The lack of an adequate vitalizing influence in the surrounding textures, and in the entire organism, upon the exudate (blastema). To this, it may be objected that the tubercle-blastema *remains crude,* however scanty its proportion, and however unimpaired the energies of the surrounding textures and of the entire organism. On the other side, we

find in the vicinity of diseased parts, and this in persons altogether debilitated and cachectic, bulky blastemata forming into textures both homœoplastic and heteroplastic; even into textures whose development is, generally speaking, very easily arrested; for example, bone-callus in individuals affected with osteo-malacia, cicatrix in, and in the proximity of, ulcerating textures. We might with propriety ask, wherefore does not the portion of an exudate farthest removed from the living textures and their influence,—wherefore, for example, in exudates upon membranous expansions, does not an entire layer uniformly,—why, in the midst of exudates, do only little scattered portions abide crude, that is tuberculize, whilst the remaining major part of the exudate becomes developed into textures?

(b.) The *lack of sufficient moisture*, of water, in the blastema, is alleged as the condition upon which tuberculization depends.

To this we reply that tubercle-blastema exudes under all conditions, and, not at all rarely, with a considerable amount of water, of blood-serum. A primitive lack of moisture in the entire exudate cannot therefore determine the tuberculization, the tuberculous nature, of its coagulable, solidifying portion. If perfected tubercle be poor in water, it is so obviously in consequence of the high degree of solidification of its blastema. This, therefore, namely, its high degree of coagulability, *might* be the cause of the tuberculous nature of the exudate,—of its persistence at its primitive stage of crudity. This, again, might be the reason why the tuberculous exudate takes the form of granulation. Inflammatory products, like blastemata generally, seldom exude pure. Inflammatory products of a tuberculous nature are, therefore, ordinarily alloyed with others of a different kind. Hence, portions of the former emerging, by reason of their transcendent coagulability, from their combinations with the latter, appear to the eye in the shape of roundish coagula, in a word, of tubercle.

But, again, it will be necessary to ask, whereupon does this high grade of coagulability depend?

It can but be founded in an as yet unknown dyscrasial constitution of the fibrin, as tubercle-blastema. There are blastemata dry from primitive poverty in serum; and also others which, parting with their serum and passing into a high degree of condensation, nevertheless do not tuberculize, but become developed into textures, in the plenitude of their mass. It would appear evident, therefore, that the tuberculous nature of a blastema must be *indwelling*, be acquired either during the local process (inflammation) or in the general blood-disease which preceded and prepared its exudation. Accordingly, tubercle would, as once before stated, have to be interpreted, now as a local, now as a general affection.

This *general character of tubercle* is the more marked in proportion as its mass as an exudate is considerable; as its diffusion through the organism is extensive; as its characters are impressed upon any spontaneous coagula formed within the vascular system; and, lastly, as the organism in its totality reflects and manifests the tuberculous habit.

Let us now, as a sequel to the foregoing, discourse respecting *that anomaly of the crasis upon which tubercle is based*. With a view, how-

ever, to establish a suitable groundwork for the exposition of the tubercle crasis, we would first add a few supplementary remarks concerning tubercle itself.

The tubercle crasis is, without doubt, a fibrin-crasis—*fibrinosis*. It is not this in respect to quantity alone—hyperinosis—but also, and this is the more important side of the anomaly, in respect to quality. This is clear even from that varied constitution of tubercle upon which we have founded our classification of fibrin-tubercle. Besides this, the fibrin must have become impaired in a particular way, in order to qualify the tubercle, which, in the one case, as simply fibrinous, cornifies; in the other case, as fibrino-croupous, does not undergo the rapid process of dissolution (puriform liquefaction) proper to croupous fibrin.

This peculiar vitiation of the fibrin may itself become somewhat modified, or admit of some accessory impairment. And this may operate as the cause of many differences in tubercle, recognized to this day only by deviating physical properties; those, for instance, of coloration and lustre, of consistence, external form, mode of aggregation of the granulations, &c. Thus, the gray tubercle-granulation is distinguished at times by its dingy bluish coloration, by a grayish lustre, by its aggregation in sharply defined spheres thinly scattered through the pulmonary texture; the yellow tubercle by a lardaceous aspect. The croupous tubercle effused into the lung during the inflammation and ichorous off-throwing of cancers, is, owing no doubt to the fundamental cancerous vitiation of the fibrin, remarkable for its whitish coloration, its softer glue-like consistency, its liquefaction to a whitish, cream-like ichor.

A point of great moment, in relation to the crasis, is the recognition of an impress upon general nutrition indicative of a predisposition to tubercle, and consisting in certain developmental proportions of textures and organs; in a word, the "*tuberculous habit*." Another point of equal interest is the relation of tubercle to other morbid processes, bound up with primitive or consecutive anomalies of the crasis.

There exists undeniably a habit, expressed in a delicate construction of the soft parts, in imperfect development of the muscular, with preponderance of the vascular, system, and especially in a so-called phthisical build of the thorax, commonly deemed ominous of pulmonary tubercle. It is essential, however, that this build should not, according to the vulgar notion, be imputed to smallness of the lungs within a seemingly insufficient thorax, but rather to very voluminous lungs within a thorax, the obvious narrowness of which, in its antero-posterior diameter, is amply compensated for by its length, with a relatively small abdominal cavity, and small abdominal viscera.

Nevertheless, tubercle does not always, nor exclusively, thrive upon a substructure like this. The tuberculous crasis, like the local tuberculosis of an organ, may become acquired in an individual of quite a different habit, as a consequence of surpassing external and internal mischief.

With reference to the second point, namely, the relation of the tubercle to other morbid processes, no disease offers so much that is interesting, that is corroborative of views already set forth, that is practically serviceable and inductive of ulterior research, as tubercle. It is

especially distinguished by its exclusive relation towards several morbid processes.

The sum of an immense range of experience in point is to the following effect:

1. *Cyst-formation*, as a new growth, is rarely found concurrent with tubercle, either in the same organ or in the same organism generally. In this sense the proliferous cyst-formations are distinguished. Where their seat is in the abdominal cavity, as for instance in the ovary, the immunity against tubercle is augmented by an accessory circumstance, in itself most important, namely, the coarctation of the thoracic space by pressure from beneath.

Experience seems to show that it is more common for cyst-formations to succeed to the extinction of tuberculosis, than the converse.

A comparison of the occurrence of both in the various organs establishes, as the extreme points of the scale, the well-known great frequency of tubercle against the extreme rarity of cyst-formation in the lungs, and the reversed proportion of the two in the ovaries, and next to these in the salivary glands. This relation seems highly important in reference to the affinity which cyst-formation bears to sarcoma and carcinoma.

2. A similar antagonism, as shown from still more numerous observations, prevails between tubercle and carcinoma. Whenever their general correlation is susceptible of proof, cancer has seemed to succeed to tuberculosis, tubercle rarely to become developed after the extinction of cancer and its crasis. Moreover, it must be repeated that to cancer, and in particular to inflamed and ulcerating cancer, there is sometimes superadded, more especially in the lungs, a tubercle, marked by a whitish coloration, a softish glue-like consistence, and a tendency to resolve itself into a whitish cream-like ichor. It has the import of tuberculo-croupous impairment of a carcinomato-dyscrasial fibrin.

A corresponding result of much interest is afforded by a comparison of the scale of frequency of cancer and tubercle, as well as of several special local relations of both.

They are diametrically opposed to one another, as thus:

FREQUENT.	RARE.
Lung tubercle.	Lung cancer.
Ovarium cancer.	Ovarium tubercle.
Salivary gland cancer.	Salivary gland tubercle.
Stomach cancer.	Stomach tubercle.
Œsophagus cancer.	Œsophagus tubercle.
Rectum cancer.	Rectum tubercle.
Ileum tubercle.	Ileum cancer.
&c.	&c.

Again, the special localities present many differences of their own. Thus, in the uterus, the vaginal portion and cervix become affected with cancer, whilst tubercle fastens upon the mucous membrane of the body of the womb, and generally stops short at the internal orifice. The epididymis becomes primarily and essentially tuberculous; the testis, can-

cerous. In the lungs the upper section is peculiarly obnoxious to tubercle, whilst cancer occurs at every part of the lung-parenchyma. In fine, cancer and tubercle possess a different import in the most various organs according as the one or the other is primitive or secondary. Thus, cancer of the liver is not rarely a primitive, tuberculosis of this organ almost invariably a secondary affection, if not a mere participation of general tuberculosis.

3. *Typhus and Tuberculosis.*—Typhus associates itself with tuberculosis only under the influence of very intense epidemics; in other words, it very seldom attacks tuberculous individuals. On the other hand, a fibrino-croupous tubercle-crasis developes itself not unfrequently in the sequel to typhus, and with it local inflammation with fibrino-croupous exudation of a tuberculous nature. This occurs in the shape of inflammations of the lungs, and also of such follicles of the ileum as have escaped the typhous process. This determines, from the softening of the tubercle around the typhous loss of substance, a combination of the so-called typhous with the tuberculous intestinal ulcer.

This tuberculosis in the sequel to typhus is without doubt based upon the not unfrequent conversion of the typhous to the fibrino-croupous crasis. The conversion takes place at different periods, but frequently at a very early stage of the retrogressive typhous process.

There is a similar relation of tuberculosis to the *acute exanthemata;* especially to scarlatina and measles. The tuberculosis following them is, for the most part, fibrino-croupous, and dependent upon a similar conversion of the exanthematous crasis.

4. *Intermittent Fever and Tuberculosis.*—The experience of foreigners places their incompatibility with each other beyond any doubt.

5. *Bronchocele and Tuberculosis.*—Although within the range of our own observation a moderate degree of sporadic goître has not seemed necessarily to possess an exclusive relation towards tubercle, the observations of foreigners as to the exclusive relation of *endemic* goître to tuberculosis merit, nevertheless, to be noticed here. Apart from the affinity in the structural relations of the enlarged thyroid gland, goître presents, in the outpouring of colloid, important points of analogy with cysts, sarcomata, and cancers, in which colloid often constitutes an essential ingredient. It would seem that, in endemic goître, it is not the mechanical hinderance to respiration that occasions *consecutively,* but an anomaly of the crasis connected with the secretion of colloid in the thyroid gland, that determines *primitively* the exclusion of tuberculosis. (See *Colloid.*) This is betokened by the alienation of the general habit contracted with goître, and still more by the fact that, in districts where goître is endemic, tuberculosis does not occur, even in individuals unaffected with the prevailing deformity.

6. *Rickets and Tuberculosis* do not readily combine. Nay, rachitic deformity and coarctation of the thorax are scarcely ever found complicated with tuberculosis. It is, as yet, undecided whether, or what degree of, exclusiveness towards tubercle absolutely belongs to rickets; and, again, whether the latter owe not its immunity to a consecutive disproportion of its own creation, namely, the deformity—the narrowing —of the thorax.

7. Even the *arterial disease upon which spontaneous aneurism depends*, and which consists in the endogenous exudation and stratification of a fibrinous substance upon the internal bloodvessel membrane (see *Abnormal Conditions of the Arteries*) is, in its more highly developed grades, very rarely associated with tuberculosis. The immunity is, perhaps, based upon an exhaustion of the materials for tubercle, due to the deposition of a solidifying blastema out of arterial blood. A more decided immunity is brought about by aneurisms, or by a single extensive aneurism, in the proximity of the heart, involving the endogenous coagulation of great fibrinous masses, and a consequent hydræmia through defibrination of the blood.

8. The relation to tubercle of *venosity* (that is, an habitual preponderance of venous blood in the system) and of *cyanosis*, as resulting from mechanical hindrance at the centres of the organs of circulation and of respiration, is of paramount interest and even of great practical importance. The remarkable exemption from tubercle brought about by these conditions induces us to set forth the relevant facts, as nearly as may be, in their natural order. They determine the *venous* constitution in various ways, generally conforming in this, that they prevent the arterializing of a sufficiency of blood; whilst they engender cyanosis by hindering the return of blood to the right chambers of the heart, the said blood being arrested in the veins, and consequently in the capillaries generally. The relevant facts, ranged in a twofold series, according as the venous habit and cyanosis are dependent upon the heart or the lungs, are as follows:

(*a*.) The first place is due to the fact, confirmed by daily experience and convenient as a starting-point for the ensuing considerations; namely, that persons laboring under enlargement (dilatation, hypertrophy, and their complications) of the heart, whether primary or superinduced by mechanical obstruction at its orifices, do *not* contract tuberculosis.

(*b*.) Nor does tuberculosis co-exist with such congenital vices of formation in the heart or the great arterial trunks [absence, insufficiency, coarctation of either, persistence of ductus arteriosus, &c.] which, with their complications, result in *venosity* and cyanosis, and, as the anatomical measure of their significance, in augmented volume of the heart.

(*c*.) Next in the series we have to mention the immunity afforded by many acquired anomalies of arterial trunks, which resemble congenital vices of formation, such as coarctation from compression, obstruction, obliteration, or again by large aneurisms in the vicinity of the heart. Apart from what has already been said on this point, the immunity is due to the mechanical impediment which the overpowering blood-column in the dilated aortal trunk opposes *directly* to the emptying of the left ventricle, and indirectly to the influx of venous blood into the right heart.

The same immunity is attained in venosity and cyanosis owing to hindrance to the pulmonary circulation; more especially where the impediment reveals its serious character by a dilatation of the right heart.

We may here further adduce:

(*d.*) The observation that the *increased density* of the lungs produced by *coarctation of the thoracic spaces*, in higher grades of lateral curvature of the spine, or in the rickety chicken-breast, excludes tuberculosis. Nay! it is an important fact that, with the establishment of a deformity of the spine in the shape of gibbosity, even when owing to tuberculous caries of the vertebræ, the tubercle-crasis is forever rooted out in consequence of the narrowing of the thoracic spaces.

(*e.*) The fact that the compression exercised by pleural effusion, and a consecutive, abiding increase of compactness of the one lung, as denoted by a sinking in of the thorax, in like manner extinguishes the tendency to tuberculosis. This effect is the more surely produced, the greater the mechanical obstruction, and the consequent disproportion between the blood-mass and the lung-capillaries pervious to it; and the less competent the other (vicariating) lung is to carry on the function of arterialization.

(*f.*) The fact that pregnancy arrests the progress of an established tuberculosis; or, as we would correct and extend this proposition, the fact that advanced pregnancy not only arrests a tuberculosis already in being, but also obviates the formation of tuberculosis generally. It is the effect of that embarrassment of the thoracic spaces, and of that resulting condensation of the lung-parenchyma occasioned by upward pressure from the abdomen; in other words, it is based upon a *venosity* brought about by mechanical means. It is probably for similar reasons that the placenta very rarely,—the fœtus perhaps never, becomes tuberculous.

This relation derives further interest from the rapidity with which, after child-birth, that is, after removal of the conditions which prevailed during *advanced* pregnancy, fibrin-crases with their respective exudatory processes, and amongst them the tubercle-crasis and tubercle-deposits, take place, more particularly through the medium of inflammatory stasis.

(*g.*) To the same class is to be referred the immunity from tubercle arising from every enlargement of the abdominal space, and the consequent narrowing of the thoracic cavity. The exemption allotted to patients afflicted with vast ovarian cystoids probably partakes of this nature.

(*h.*) Again, the fact that even congenital smallness of the pleural sacs, paired with primitive smallness of the lungs, and, as it mostly is, with an inverse ratio of the development of the abdomen and its viscera, serves as a protection against tuberculosis.

(*i.*) That in the earliest childhood (with closed fœtal passages), owing to a condensed state of the lungs caused by predominant abdomen, tuberculosis occurs, if at all, very rarely.

(*k.*) The exemption apportioned to those who labor under chronic catarrh, under vesicular emphysema of the lungs, or under bronchial dilatation, was recognized even by Laennec. The empirical recognition of this relation has even led to attempts to cure tuberculosis by the forcible production of those conditions. The real preservative point was, however, overlooked, both here and in another mode of cure aimed

at by others, namely, that of closing cavities in the lungs by forcible compression of the thorax. The protective and curative impulse consists, even here, in *venosity*. And this venosity is a consequence of the destroyed function, the collapse and eventual wasting of numerous pulmonary lobules, through obstruction of their bronchia with muco-purulent secretion; in bronchial dilatation, through the concomitant obliteration of considerable portions of the lung; in emphysema, through lost contractility of the pulmonary texture for expiration, and consequently embarrassed respiration, more especially, however, through the destruction of extensive ranges of the lung-capillaries.

(*l.*) It will be readily understood that the dropsical crasis, especially when resulting from *venosity*, excludes tubercle.

It will now become necessary to inquire how certain exceptional cases are to be explained. Individual cases of the kind are represented in tubercle associated with cancer, or with *venosity* mechanically brought about.

(1.) The conditions mentioned as excluding tubercle, operate thus only in so far as the latter is based upon a hyperinotic crasis—*an excess of fibrin*. This does not, however, prevent the small fund of fibrin accompanying those conditions from being, under favorable circumstances, expended upon tubercle formation, which then becomes localized in a process of exudation.

(2.) The tubercle may be the product of a *local inflammation*, in which the fibrin becomes tuberculous.

(3.) The entire mass of fibrin may suffer a morbid change, effecting, as intercurrent disease, a consecutive tubercle crasis, which becoming exhausted by a corresponding exudation, again gives way to the original crasis.

It is thus that genuine tubercle, when concurrent with cancer, may be interpreted; and this the more readily, that true hyperinoses and fibrinous exudates not unfrequently do co-exist with cancer. The tubercle may be merely local, and the cancer no less so. It may, however, be local, and yet the cancer be a general disease. Or, again, it may be the product of an intercurrent primitive tubercle-crasis, or of a consecutive one derived from a local process, and co-ordinate with those hyperinoses and fibrin-exudations which not rarely supervene upon inflamed and ulcerated cancer, reflecting a secondary crasis.

(4.) As to the exemption afforded by *venosity*, there is no doubt that, to render it complete, a high degree of the latter is requisite. Since, however, we possess no scale whereby to ascertain directly the grade of a protective crasis, and to illustrate the exceptions, we must inquire whether it be not possible to arrive indirectly and approximatively at this recognition. In the absence of such a scale, certain anatomical changes must serve as the measure, so to speak, of the anomaly. They consist in the degree of heart affection (dilatation) present, this furnishing an available criterion for the amount of the impediment to the circulation, and therefore for the grade of the venosity. This approximative index with the aforesaid inferences, will be especially applicable where the precise extent of the impediment is not to be immediately summed up from anatomical data, as in lung affections, like catarrh and bronchial dilatation, emphysema, and preternatural density of the lungs.

We attach importance to this relation of tubercle to the venosity resulting from mechanical impediments in the heart and lungs,—as affording not alone proof of the fibrin-crasis being the foundation of tubercle, but also valuable indications for medical treatment.

We have now to consider the relative occurrence of tubercle in the different organs and textures, and its peculiar processes of repair.

It will be expedient, however, as a preliminary point, to determine what is signified by scrofula,—what is the distinction—if there be any—between scrofulous and tuberculous substance.

For our own part we hold *tubercle* and *scrofula* to be *identical*—tuberculosis and scrophulosis to be one and the same disease; and this upon the following grounds, namely:

(*a.*) One and the same elementary composition, both anatomical, and, so far as investigation has gone, chemical also. This applies with especial force to scrofulous substance, as compared with *yellow* tubercle.

(*b.*) Both are subject to the same metamorphoses, namely, softening and cretefaction.

(*c.*) The tuberculous and the scrofulous ulcer are identical both in the same, and in different organs; for example, the scrofulous skin- and the tuberculous intestine-ulcer. The same identity attaches to their cicatrix.

(*d.*) Both frequently coexist in the same organ, sometimes without, sometimes with, the appearances of inflammation.

The truth is, that the yellow tubercle is commonly called "scrofulous substance," more especially when it occurs in largish masses, and affects in the usual way the glands—the lymphatic glands—in children. Thus the same substance concurrently affecting the lungs and the bronchial glands is denominated, in the one instance tubercle, in the other scrofula.

A scale of the frequency of tubercle in the various textures and organs, offers but limited points of interest. According to our 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.

The ensuing remarks appear to us well deserving of attention, as affording evidence of the imperfection of *any* summary scale of frequency.

(1.) At every point where capillaries occur, there may be tubercle. Epidermid formations and cartilage are therefore alone exempt from tuberculosis.

There are, however, vascularized organs in which tubercle very rarely, if ever, occurs; such are the salivary glands, the ovaries, the internal bloodvessel-membrane, the œsophagus, the vagina.

Even vascularized new growths may become the seat of tubercle.

(2.) If, which is most important, we consider tuberculosis individually, according to their primitive or to their secondary appearance, an entirely different scale is set up. 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 the new scale, seeing that they seldom, if ever, become the primary seat of tubercle.

(3.) 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 tuberculoses. The liver, spleen, kidneys, nay, in many cases the lymphatic glands, stand in this relation to tubercle.

(4.) 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 supra-renal gland, and of the lymphatic glands; of uterine and tubal, and of peritoneal; of pulmonary, and of intestinal, or of laryngeal tubercle.

(5.) 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. (See "Tuberculosis," vol. iv.)

(6.) Again, the mode of production of tubercle varies in the different organs. Thus, upon serous membranes and in bone, tubercle is, for the most part,—upon mucous membranes, very frequently,—in lymphatic glands and in the brain, not unfrequently the product of inflammation.

(7.) In fine, 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.

(8.) 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 cervix uteri and the vagina remain exempt.

Tuberculosis very commonly proves fatal, if locally, by impeded function, by palsy of the affected organ, in consequence either of the extensive, acute deposition of tubercle into its texture, or else of the ulcerous destruction of the latter in the process of so-called tuberculous phthisis. Or the tuberculosis may, as a general disease, destroy life through impoverishment of the blood, through hydræmia or the serous crasis, an issue vastly favored where the tubercle is copiously and at the same time rapidly thrown out, and where local tuberculosis in important organs hinders the reproduction of blood.

The cure of tubercle may take place in various ways. Each of the metamorphoses of tubercle may become invested with the character of a healing process. Still, neither the decadence of tubercle, nor its ejection through the medium of ulceration, as local healing processes, are fraught with any value for the individual, unless accompanied by the extinction of the fundamental, tubercle-producing crasis.

The cure of tuberculosis as a general disease—as tubercle-dyscrasis—takes place now and then obviously through the intervention of some of the processes and conditions already adverted to as excluding tubercle; at other times, through influences entirely occult.

A question connected with the local healing process of tubercle here suggests itself, namely, as to the absorption of crude tubercle? The resorption of tubercle as formerly believed in, was probably first repudiated by Laënnec, and after him by most pathologists; and although valid grounds can hardly be alleged for its impossibility, neither has it ever been proved by direct evidence, nor is it at all within the compass of likelihood.

The *obsolescence*, the cornification of gray tubercle, represents incontestably its readiest process of involution. As a direct extinction of the tubercle, it would afford the completest cure, did it not concern a growth which would fail to become destructive if it abided in its primitive crude condition.

Of the two other metamorphoses affecting yellow tubercle, cretefaction of what has undergone softening unquestionably presents the most desirable process of repair, as will become evident from the following

remarks concerning the other metamorphosis, considered as a healing process, or as the basis of one.

The softening of tubercle cannot of itself serve for a reparatory process. The *elimination* of softened tubercle through the instrumentality of ulceration in its vicinity, can alone pass current for such.

But, taking into account—

(*a.*) That it can only be brought about by ulcerous destruction of the textures.

(*b.*) That, although the aim of this ulcerous process be to heal, it may, when the tubercles are numerous, readily induce exhaustion.

(*c.*) That the attendant inflammation—the general disease being unextinguished—of itself determines tuberculous products, thus extending, without limit, the ulcerous consumption of the textures.

(*d.*) That even under favorable crasial conditions, an infection of the blood is possible in tuberculous ulcers (cavities).

Taking, we say, all these circumstances into account, this curative process must be regarded as widely subordinate to that of cretefaction, to which it stands in nearly the same relation as the removal from the body of a foreign substance by a debilitating ulceration, to the same substance being rendered innocuous by incapsulation.

The healing of a tuberculous ulcer or cavity,—of tuberculous ulceration,—can therefore only take place provided the accompanying inflammation, owing to extinction of the tuberculous crasis, ceases to deposit fresh tuberculous matter, and determines organizable products instead. The loss of substance is made up for by new-formed shrivelling scartexture. Where the tubercle has not been completely eliminated in the phthisical process, the residue may become isolated by a pap-like inspissation and eventual cretefaction.

ALBUMINOUS TUBERCLE. *Acute Tuberculosis.*

Under this denomination is understood a disease presenting many points, both of resemblance and of dissimilitude with the tuberculoses already discussed. It devolves upon us to investigate these analogies and differences ; the former appearing to us to preponderate so far as to preclude our separating the disease from tuberculoses generally.

There is a disease which, under an acute course, and under typhoid symptoms, determines a tubercle differing in many respects from the fibrinous. It always represents solid, mostly poppy-seed-, rarely, if ever, millet-seed-sized, sometimes limpid, softish, glutinous, gray granulations, either of vesicle-like or of a dull transparency, often only cognizable under a favorable incidence of light ; at other times, although far less frequently, opaque, whitish, or whitish-yellow.

On a closer inspection, this tubercle appears marked by cell-formation. It is found to contain—

(*a.*) The ordinary nucleated, exudate cell, in considerable numbers.

(*b.*) Cells with two or three nuclei.

(*c.*) Cells with filial cell-formation.

(*d.*) A structureless soft basement connecting these elements.

It exudes always in great abundance under the symptoms of hyperæ-

mia, in scattered granulations, uniformly distributed through the parenchyma of the affected organ, and either all at once, or at intervals rapidly succeeding each other. A manifest equality of size and character is observable in all those deposited simultaneously, or during the same attack. With it there is always effused a grayish, sero-albuminous semigelatinous humor, with which the diseased textures become infiltrated.

. This tuberculous deposition affects not only entire organs or large sections of organs and of textures, but commonly several organs and textures simultaneously or in rapid succession; a single one, however, generally operating as the main point of concentration. Its seat is in the lungs, the pia mater, especially at the base of the brain, the spleen, the serous membranes, especially the peritoneum.

This tuberculosis is only in rare instances the primitive one. For the most part, it is based, so to speak, upon a precursory fibrin tuberculosis of the lungs or lymphatic glands. In these cases its point of concentration is generally either the organ previously affected, or some structure standing in immediate relation with it.

The disease proves fatal through palsy of affected organs essential to life, or else through dyscrasial influence.

This tubercle is subject to no metamorphosis.

The dyscrasial character of the blood is manifest, and closely assimilates to the exanthematous crasis. (See "Crases.") In accordance with it are the livid coloration of the common integument in the dead subject, the dark coloration of the muscles, the general appearance of flabbiness, the serous infiltration of the parenchymata.

In this description of the disease we recognize the albuminous crasis, and a product which, in its subordinate coagulability, its soft, gluey character, its cell-development, gives evidence of its *albuminous* nature.

This tubercle renders it probable that albumen, without previous conversion to fibrin, may acquire a considerable amount of coagulability, and become tuberculous; nay, that where the opaque yellowish or whitish-yellow acute tubercle does not form upon a basis of croupous fibrin, even albumen may, without conversion into fibrin, acquire the croupous character.

In this description we recognize not only the distinctions, but also the analogies between the fibrinous and the albuminous tubercle. These analogies stand forward the more prominently if we recall to mind the fibrin tubercle of acute production.

Such analogies, apart from the resemblance in outward form of the two heterologous deposits, that is, the tubercle form; apart from the uniform size of the granulations thrown out at the same period of exudation; and apart from their equable dissemination through the parenchymata; are as follows:

(*a.*) Both are rarely the primitive tuberculoses in an organism.

(*b.*) Both are thrown out under manifestations of hyperæmia.

(*c.*) With both there is effused, as a sort of vehicle for the coagulable portion of the entire exudation, a serous, sero-albuminous fluid.

(*d.*) Both affect the same organs and sections of organs.

(*e.*) The albuminous tubercle bears the same relation towards other diseases as the fibrinous.

(*f.*) In by no means rare instances, a step-like transition from the fibrinous to the albuminous tubercle is incontestable. Upon the ground-work of a fibrin-tuberculosis, which has undergone frequent phases of phthisis, there exudes, with augmented dyscrasis, in the lungs more particularly, a tubercle which, with every fresh act of exudation occurring in rapid succession, becomes softer and poorer in fibrin, until ultimately reduced to a soft, semi-fluid, albuminous tubercle,—a consummated acute tuberculosis.

(*g.*) Occasionally we discover, especially in the texture of the pia mater at the base of the brain, an exudate consisting of albuminous tubercle and tuberculizing croupous fibrin, a primitive combination of the two products.

(*h.*) Not only does albumen enter into the composition of fibrin tubercle, but a certain amount of fibrin modifies that of the albuminous tubercle. A complete exclusion of the one or the other is hardly conceivable, and it is only the predominance of the one or of the other that characterizes the product. Between the extremes of fibrinous and of albuminous tubercle there exist numerous middle and transition forms.

ALBUMINOUS CRUDE BLASTEMATA.

Under this head we shall discuss certain products in their nature probably albuminous, and essentially distinguished from other albuminous blastemata by their persistence in the condition of crudity. Owing to this persistence, as also to their being founded in a dyscrasial element, we rank them along with tubercle, with which, moreover, they occur not unfrequently in consecutive alliance.

They are, for the most part, solidified blastemata, resembling to the naked eye a translucent coagulated albumen. Now and then, however, they are opaque, and of a turbid whiteness. They consist of an amorphous, glebous, transparent basement, and of nucleus formations.

They occur in certain parenchymata in the shape of infiltration—very rarely in that of a collection of roundish nodules from the size of a hemp-seed to that of a pea. The liver, the spleen, and the kidneys are known to become affected with these infiltrations, which give to the surface of the organ a brawn-like aspect with a transparent margin, frequently representing a spurious hypertrophy of the organs named. (See " Hypertrophy.") Whether these blastemata occur in one, or in several, or in all of those organs, they mostly occasion considerable enlargement thereof, and at the same time a notable change in their consistency, the parenchyma becoming compact and of doughy brittleness.

The out-throwing of these blastemata occurs in an insensible manner.

In point of fact, they comprise that partly more or less solidifying, whitish, partly viscidly fluid blastema effused into the parenchyma of the kidney in Bright's disease, particularly in certain of its chronic forms.

As may be inferred from the above, and as experience amply confirms, these blastemata never occur but in connection with high grades of general dyscrasial disease; such, for example, as rhachitis, mercurial

cachexia, inveterate syphilis, ague-cachexia, and especially certain tuber-culoses.

The deposition of these blastemata is, therefore, never a local affec-tion, but invariably indicative of an anomaly of general nutrition. It is clearly dependent upon dyscrasis, which may consist in an excess of albumen in the blood, and be either primitive or secondary, as in the tuberculosis resulting from the exhaustion of fibrin. The consequence of a copious and extensive secretion of these blastemata is the eventual exhaustion of albumen, and a watery condition of the blood [hydræmia], inductive of dropsy, anæmia, &c.

These blastemata usually abide altogether, and throughout in their primitive condition. Occasionally, however, there is observable, at certain spots, a transformation of their mass into molecular fat. They become opaque; of a whitish dulness; friable. This is especially the case in the liver and kidneys, and it is not improbable that cera-larda-ceous infiltration of the liver is the result of a progressive, diffused con-version of this albuminous blastema.

II. UNORGANIZED NEW GROWTHS.

A.—OF UNORGANIZED NEW GROWTHS IN GENERAL.

These lack both the internal order and the definite forms which cha-racterize organized new growths, and their development comes under the dominion of chemical laws. Between the *rudiments* of what *is*, and what is *not* organized, there is no distinction in point of form. In a chemical sense non-organized growths are composed both of unorganized and of organized substances, either singly or conjointly, and it is even common enough for a new growth to be made up through the mechanical blending or interlacing of organized with unorganized materials. All these considerations taken together preclude any marked discrimination between the two.

There are, upon the one side, undoubtedly new growths representing perfect unorganized formations, for example, certain concrements. On the other side, however, non-organized new growths originate under con-ditions and forms which have induced us, notwithstanding their unor-ganized nature, to discuss them along with the organized new growths. We may instance the forth issuing of lime-salts—as cretefaction, ossifi-cation, incrustation; of the free fats; of colloid; of tubercle.

The material for non-organized new growths in general, is contained both in the textures, and in fluid and solid blastemata; the material for a special order of non-organized new growths, in the proper fluids of secretion. Its nature varies considerably. It consists of protein sub-stances, certain gluten-substances, horn-substance, fats, pigments, acids, salts. In a more extended sense, even the various gases and fluids occurring in textures, or in the cavities of the body or of organs, the fluid of genuine dropsy for instance, belong to the class.

Without for the present taking these last into the account, we have to observe with reference to unorganized new growths:

The elementary forms are the amorphous, the glebous, the laminate, the granular (down to the finest molecule or point-mass), the crystalline. Certain substances possess a determinate form, dependent, however, for the most part, upon their peculiarity of composition, upon the conditions under which they become severed from their primitive combinations, and upon their mode of development. Thus protein substances occur, both structureless, and in a glebous or a molecular form.

These materials constitute secondary formations, either alone or with the intervention of a bond-medium, for example, mucus. This is often furnished, together with the external moulding or form of the new growth, by the glutinous basis of a texture; for example, in the so-termed ossification of a fibrous tumor. Both in form and size they manifest great variety, not readily susceptible, however, of generalization. We allude more particularly to calculous concretions! In consistency they are in various degrees soft or firm.

Above all, their chemical composition varies greatly. As regards concretions and calculi, these readily divide into two groups, namely, into such as form out of fluids of secretion, and consist of the respective components of those fluids, and into such as become developed out of blastemata and textures. These last have a composition corresponding with their base, and very commonly consisting of phosphate and carbonate of lime, and of magnesia.

Respecting the origin—the mode of production—of non-organized new growths, it may be stated generally—

1. They are exudates or secretions in a primitive form of non-organization, as exemplified in crude fibrin, and encysted colloid and fats.

2. They are the result of various transformations of such products. To this order belong:

(a.) Formations arising out of the conversion of exuded and secreted protein materials into glutinous, into horny substance, into fat; for example, the conversion of fibrin and albumen to colloid, to horny substance, to fat in the molecular or crystalline form.

(b.) Formations arising out of a process imitating ossification in fluid or solidified, unorganized or textural bases; a liberation of lime-salts (phosphate and carbonate) out of their primitive combinations, as cretefaction, ossification, lime-incrustation, concretion. (See "Bone Formation.")

(c.) Formations brought about by a more palpable deposition of all, or only of certain, components of a fluid in which they are held in solution or suspension. They are most especially prone to form in secreted fluids, and either consist purely of specific ingredients proper to them, or occur blended with other elements. *They constitute calculous concretions.* The cause of their separation is manifold. It may be that the fluid has become more concentrated, for example, by loss of water, their solvent medium, through exosmosis (resorption) or more especially by evaporation. Again, we may mention, besides the precipitation from fluids of certain specific components, the inspissation and exsiccation of secreted and exuded fluids in their totality; for instance, of mucus, of ear-wax, of the smegma præputii, of the bile, of exudate, of pus, &c. Or else it is a consequence of a chemical conversion of the fluid,—of the

solvent, or of the dissolved substance. For instance, the free acid of normal urine retains the phosphatic earths in solution; when, however, the urine is rendered alkaline, be it by the presence of mucus or exudate, or by conversion of the urea into carbonate of ammonia, the phosphatic earths become precipitated. If the lithates present in the urine become decomposed by an excess of acid in the urine, the lithic acid, as the less soluble, is thrown down. The soluble phosphate of magnesia present in almost all the fluids becomes precipitated, the moment that it enters into a combination with ammonia, to ammonio-phosphate of magnesia.

Unorganized new growths possess sometimes a local, sometimes a general import. Thus, urinary calculi may be the result either of mere local contingencies, or of various anomalies of general nutrition, that is, of a dyscrasial process.

B.—OF UNORGANIZED NEW GROWTHS IN PARTICULAR.

We have here, in the first place, to bring forward and to examine in detail the substances which constitute new growths.

1. *Protein substances*.—The primitive form in which these emerge from their solutions, is that of a structureless or glebous mass, in various degrees of coagulation, and that of an elementary granule down to a pulverulent point-mass. The reason for their appearance in these forms, that is, the reason for the general coagulability, and *à fortiori* for their specialities of form and coagulation, is quite obscure. That which spontaneously undergoes rapid and firm coagulation passes current for fibrin; that which coagulates more slowly and less perfectly under a manifest progressive change in the medium of solution, for albumen. The molecular form appertains in particular to the higher grades of oxidation of the protein substances (croupous and pyin-holding fibrin). Since the influences which produce the coagulation and precipitation of albumen in experiments, do not presumably take place within the organism, the discovery of the modifications suffered by albumen through the agency of water, acetic acid, and the like, is highly deserving of attention.

The protein deposits are insoluble in ether and in mineral acids. By acetic acids they are rendered translucent, and ultimately dissolved. By caustic potash and fuming hydrochloric acid they are slowly dissolved —by the latter with a lilac tint. An aqueous solution of iodine colors them yellow.

The *glutinous* and *horny* substances emerging out of the protein-substances are amorphous, or have a glebous or a stratiform, elementary composition. In their physical properties they approximate, more or less, according to their grade of perfection, to gluten and to urea; in their chemical reaction, to various modifications of gluten (gluten, chondrin, pyin, &c.), and of urea.

2. *Fats*.—Their elementary form of occurrence is that of *drops*, or of an amorphous solidification; of granules; of crystals. Little is known concerning the nature of fats originating thus, more especially of those assuming the form of granule (elementary granules, both free and in-celled, discrete and aggregate), or developed out of blastemata and tex-

tures through conversion of protein-substances, and probably even of gluten.

The fats cognizable by their form and chemical relations are :

(*a.*) *Elain.*—It occurs in variously-sized drops, both free and incelled. In this form it is usually set free out of emulsion-like compounds ; in the form of *elaic acid*, out of saponaceous compounds, or out of combinations with other fats, for example, in exudates, in medullary carcinoma. It frequently represents an effusion of the contents of fat-cells, consequent upon gangrenous or ulcerous destruction. The drops resist the action of water and of acids, but dissolve on being boiled with potash, and still more readily in ether or heated alcohol.

(*b.*) *Margarin and margaric acid.*—These occur in microscopic needle-cyrstals, for the most part aggregated in stellate groups or bundles. In this shape the margarin emerges, after the body has become cooled, from its solution in elain, either within the fat-cells, or without. The crystals of margaric acid, soluble in concentrated, heated alcohol, are, according to Vogel, probably a product of decomposition, wrought out of the margarin of the fat, it may be, by a free acid, so often developed in gangrene.

(*c.*) *Cholesterin.*—When cognizable as such, it occurs in tabular crystals, representing rhombic planes. Many, however, of the aforesaid fat-granules are likewise cholesterin. It almost always occurs along with other fats, and often very copiously ; for instance, in gall-stones, in the atheroma of arteries, in encysted tumors. The fact of cholesterin so frequently occurring in fluid and solidified protein substances during their disintegration,—as in exudates, in tubercle, in stratiform coagula upon the inner coat of arteries, renders it probable that, like other fats, it is the product of a decomposition of the elements of those substances. This seems to us more probable than that it exists preformed in combinations which cause it to be held in solution. Its detection in the blood does not appear to us a valid objection to this. It is soluble neither in water, nor in acids, nor yet in alkaline solutions, but only in ether and heated alcohol.

(*d.*) *Stearin.*—Its occurrence is not proved with certainty, although, under certain conditions, in which fat assimilates to the suet of the wether, not quite improbable.

3. *Pigments.*

(*a.*) *Black, brown, russet-yellow pigment* (See "Pigment"), in the shape of molecular granules ; the last two occur, also, adherent to microscopic crystals of ammonio-phosphate of magnesia.

(*b.*) *Bile-pigment,* as a finely granular precipitate of a yellow-brown color, insoluble in water and in most of the acids,—soluble in a boiling potash-solution, with a greenish-brown tint. Nitric acid destroys it, after causing it to pass through phases first of green, then of blue, and lastly of red coloration.

4. *Lithic acid and lithates.*

(*a.*) *Lithic acid.*—The fundamental type of its crystals is the rhomboid prism, which, however, often appears cut down to a rhombic plane. The crystals, frequently seen grouped into rosettes (Simon and Vogel),

are difficult of solution in water, insoluble in acids, alcohol, and ether. Potash causes their gradual solution. They occur in the urine.

(*b.*) *Lithate of ammonia*, as a finely granular precipitate, colored of a dingy yellow, yellow-red, russet, rose-tint; difficult of solution in cold water; less so in hot. The effect of acids is to isolate the lithic acid, which, under the microscope, is then seen to develope its crystals.

5. *Lime-salts.*

(*a.*) *Basic phosphate of lime*, as a gelatino-granular mass, soluble in acids. It occurs both in fluids and in solidified formations, in a soluble combination of protein-substances, with gluten, out of which it separates —especially in the shape of cretefaction and ossification—in the form of molecule.

(*b.*) *Carbonate of lime*, in the shape of granular deposition,—in the cell-incrustation, of stratefaction—either alone or in union with the foregoing substance. Soluble in acids, with effervescence.

(*c.*) *Oxalate of lime*, in octohedral crystals, sometimes remarkably minute; insoluble in water, alcohol, ether, acetic acid; soluble in hydrochloric acid. Found in the urine.

6. *Ammonio-phosphate of magnesia*, in crystals of various shapes. When rapidly formed, they cluster together in stellate groups of needle-shaped crystals, or represent denticulate, leaf-like forms. When slowly developed, they constitute trilateral prisms, in which both angles corresponding to the same lateral-edge are truncated. The crystals are readily soluble in acids—even in acetic acid. The occurrence of this salt is extremely frequent. Wherever a development of ammonia takes place, the wide dissemination of phosphate of magnesia determines the formation of the insoluble triple phosphate.

7. *Sulphuret of iron*, in molecular granules, soluble in acids and precipitable out of these by means of sulphuret of soda.

Such are the principal and the better known substances which, independently of, or in combination with, others, compose the bulky unorganized formations, as so-called concretions or concrements. We shall treat of these generally; dividing them into two great series, namely:

1. Into such as are essentially protein-substances; or into such as consist of gluten or horn-like substance, of fat, and, lastly, of the phosphates and carbonates of lime and magnesia; it matters not whether the latter be directly thrown out as such, or whether they have, as usual, become obviously developed out of the former, that is, out of the protein-substances.

2. Into such as have comparatively a varied composition, and are marked by the specific substances which they contain.

FIRST SERIES.

(*a.*) *Protein concretions*, as coagulations within the bloodvessels— *vegetations;* as exudates in parenchymata, upon membranous surfaces; as free bodies in serous cavities, tubercle, &c.

(*b.*) *Accumulations of gluten-like, colloid substance*, commonly within cyst-spaces; and *cornified protein concretions*, for example, of the valve-vegetations in the heart.

(*c.*) *Fat*, as the cyst-contents; or as accumulations within serous cavities; in parenchymata, in the shape of spherical or irregular masses. Frequently in combination with the following, namely:

(*d.*) *Bone-earth concretion*, so-called cretefaction and ossification, as developed, not alone in the protein and gluten-holding unorganized basements already adverted to, but also in fibroid and cartilaginous textures, in a mode and form which, together with their relations to the normal ossification of bone-cartilage, we have discussed in another part of this volume. [See " Bone Formation."]

The concretions consisting of *lithate of soda*, found in the sheaths of tendons, within capsular ligaments, even in the spongy texture of the articular terminations of bones, constitute an exception only as regards the nature of the substance itself.

The significance of concretions of this series differs with the organ affected; thus, concretions on the heart's valves are, perhaps, the most important of all.

SECOND SERIES.

To this series belong the concretions in and arising from fluids of secretion. We divide them into two species:

(*a.*) They result from the *precipitation of one or of several of the specific components of a secretion*, animal matter entering into their composition for the most part only in small quantity and by way of a bond medium.

They constitute the genuine stony concretions or calculi, which, when diminutive, are termed gravel or sand.

The size of calculi is, as may be inferred from what was just stated, extremely various, from that of a fine, just perceptible sand-grain, to that of a concretion filling up the largest secretory canals and reservoirs.

In smaller, solitary concretions the form is mostly the spherical;—in larger ones, it corresponds to that of the said canals and reservoirs, as in the instance of renal calculi, and is subject to much modification. Where many concretions co-exist, they acquire from reciprocal pressure and friction, the most varied, polyedrical shapes,—as in the case of urinary calculi, and especially of gall-stones. Their surface is smooth, polished, or else knobbed and uneven, stellate, thorny, rough.

Their consistence mainly depends upon their chemical composition.

They reside at large in their respective cavities, or else, filling the latter, they lie firmly impacted. Or again, they adhere as if glued or soldered at some point, through the medium of fibrinous exudate.

Their structure varies extremely. At their nucleus they exhibit an agglomeration of an amorpho-granular precipitate. Or again, they consist of concentrical strata of the same character, or else of a crystalline precipitate; or lastly, they are altogether of crystalline fabric, as in the case of certain lithic acid calculi, but particularly of cholesterin concrements in the gall-bladder.

The first impulse to their formation is sometimes given by foreign bodies introduced from without, or by coagulate,—endogenous products. The concretions represent, in the first instance, incrustations of things

in various degrees alien to their composition. Thus, for example, on the one side, a great variety of foreign bodies which have lapsed into the urinary bladder, give rise to lithic acid calculi; on the other side, inspissated bile, or bile-pigment, to cholesterin calculus in the gall-bladder.

To this category belong lithic acid calculi, salivary calculi, lachrymatory calculi, prostatic calculi, gall-stones, many intestinal concretions.

(*b.*) They are due to the *inspissation and desiccation* [through exosmosis or evaporation] of some fluid of secretion either within or externally to its secreting canals and cavities. Here the concrement consists of the ingredients of the secretion in their totality, including, of course, a considerable amount of so-called animal matter, and with it of organized elements. Proportionately to the degree of inspissation, the concrement is soft; or, it may be, of a stony hardness. Concretions of this kind very often become developed in cyst-like dilatations of the follicles, in which the secretion accumulates and stagnates, and the inspissation of the contents of encysted tumors of new formation, applies here in its most extended sense. The physical and chemical properties are, it will be readily conceived, extremely inconstant and variable.

This group comprises concretions in the follicles of the skin, in mucous follicles, in the tonsils, in the nasal and pharyngeal cavities, upon the glans and prepuce, and certain intestinal concrements, especially those occurring in diverticula; finally, the inspissations of colloid, and of other cyst-contents.

CHAPTER X.

ANOMALIES OF CONTENTS.

IN this chapter we have to treat of:

A. *Pneumatoses* and *Dropsy*, which we have already adverted to as non-organized new formations.

B. *Foreign substances* introduced into the body.

C. *Parasites*, that is animals, and vegetable growths, occurring in and upon the living body. We give them a place in this chapter because, according to the researches of modern science they are to be numbered amongst the things that are received into the organism from without.

A. PNEUMATOSES AND DROPSY.

1. *Pneumatoses*,—the accumulation of various gases has been observed as emphysema, both within textures, and more particularly in almost every cavity of the body and of its organs. The scale of frequency varies, indeed, according to the nature of the gas, and to its mode of origin. There are, however, organs in which gas-accumula-

tions of every kind are extremely common; and again, others in which a development and accumulation of gas are under all circumstances very rare.

The modes in which gas-accumulations originate resolve themselves generally, into the following :

(*a.*) The gas accumulated in the texture or in the cavities of the body or of organs, is *atmospheric air* which has penetrated from without. This applies to most kinds of emphysema, and of gas-accumulations in the pleural sac, partly to those in the stomach, perhaps also to the rare instances of gaseous collections in the uterus, and in the urinary bladder; lastly, to the presence of gas in the blood after the lesion of veins, particularly those of the neck. Most examples of interstitial emphysema and of pneumothorax are the result of lesions of continuity, through either wounds or ulceration in the bronchial passages or in the lungs.

By tarrying in preternatural localities, the atmospheric air suffers a change similar to what it undergoes in the lungs, its oxygen becoming exchanged for carbonic acid, with the superaddition of aqueous vapor.

(*b.*) *The gases are products of decomposition.*—To this category are to be reckoned, besides those gas-accumulations arising out of putrefaction after death,

a. Gas-development out of the blood-mass, from putrid decomposition of the latter; out of blood perishing through absolute stasis; finally, out of decaying normal textures or morbid products, for example, sloughing cancers, or exudates undergoing decomposition.

β. Gas-development in the stomach and intestines, the details of which concern special anatomy.

2. *Dropsy*, whereby we understand *genuine serous dropsy*, that is, a fluid mostly alkaline, in its purity colorless and limpid, and analogous in the quality of its ingredients to, although originally thinner than, the serum of the blood; a fluid which, apart from the accidental admixture of exudates, pus-cells, blood-globules, epithelia and the like, contains nothing beyond unorganized effusions of albumen, pigments, fats (cholesterin), and salts. Under no conditions has it of itself alone the significance of a blastema.

It consists, chemically speaking, of water, albumen, fat and extractive matter, and of salts, the chloride of sodium preponderating over the rest, namely, the carbonates and phosphates, of alkalies, and of alkaline earths. Generally speaking, its proportion of water is greater than that in blood-serum. The albumen is subject to the greatest fluctuation, down to an infinitesimal allotment.

This relation is liable to various and not unfrequent deviations.

A red coloration is due to blood-pigment.

A yellow, or yellowish-green coloration, to bile-pigment.

A whey-like turbidness, a milky-white appearance, may be owing to certain of the admixtures adverted to, such as epithelium, but especially fat, and to an albumen precipitated by an excess of water [relatively to the saline contents].

Sometimes the fluid has a faint acid reaction.

A notable proportion of albumen renders the fluid viscid, adhesive, synovia-like.

This albumen, for the most part, shows itself to be pure; and not to differ from that of blood-serum; or else it exists as albuminate of soda. It occurs, however, in certain other tolerably well-known, and without doubt in many other as yet unknown modifications.

Occasionally the dropsical fluid contains urea. The accumulation of the fluid in the textures constitutes *œdema*, of which species of infiltration every organ may become the seat. Its collection in cavities of the body, or of organs, constitutes the *dropsies*. Moreover, the serous effusion developed beneath the vesicated epidermis in erysipelas, in burns, and through the agency of cantharides, is deserving of general mention here.

The mode of origin of dropsy varies:

(*a*.) The *purest dropsy* arises from *retention of the blood in the veins through mechanical hindrance to the circulation*. Its extension varies according to the seat of the obstruction, being considerable in proportion as it affects the centres of the circulation. It is in all probability the veins, even the larger ones, which, in a dilated and thin-walled condition, suffer an out-throwing of dropsical fluid to take place from the blood. The exudation will be considerable, proportionately to the amount of hydræmia—that is, of the serous crasis—that prevails.

Dropsy is, without doubt, determined by the lymphatics in a similar way.

(*b*.) Nor is there any doubt that serous effusion takes place, in like manner, from the *capillary vessels*. This mode of occurrence applies to the dropsy resulting from general debility; to that arising in palsied parts; to that referable to hydræmia. Again, we may attribute to the same source that acute or chronic œdema consequent upon mechanical capillary hyperæmia, both active and passive; and, lastly, that œdema founded in a slight degree of stasis, or attendant upon consummate inflammation. Of the latter description are those outpourings of the blood-serum precursory to the genuine exudation of plasma in the inflammatory process; the œdema encircling areæ of inflammation; the aforesaid serous collections beneath the epidermis, in erysipelas, in burns, &c.

(*c*.) In fine, *dropsical effusions* are brought about by *attenuation of the blood*,—or the serous crasis, a condition frequently combined with the aforesaid causal influences.

The consequences of serous effusion vary greatly according to the nature of the organs or textures concerned, to the extent of the accumulations, to the acute or chronic form of their occurrence, and to their duration. The *relation of the textures* generally is of much interest. In acute dropsy the textures are in various degrees congested, reddened, and withal—more especially the lung texture—lax, easily torn;—very delicate textures, for example, that of the brain, softened and destroyed. In chronic, enduring dropsy, on the contrary, they are discolored, pallid, bloated with imbibed serum. Smooth membranes become turbid and dull, the contractile fibre paralyzed.

Dropsical fluid is either wholly or partially re-absorbed, or continues unchanged. In the second case, the watery part being first of all absorbed, it becomes concentrated to an albumen, a synovia, or a thin jelly-like mass.

In what manner the various œdemata and dropsies may become perilous, and eventually prove fatal, is sufficiently evident.

B. FOREIGN BODIES.

Inanimate foreign bodies are not unfrequently met with in the organism.

They are introduced accidentally or designedly, either through the natural orifices, as the mouth, the anus, the orifices of the urethra and vagina, the ears, the nostrils; or else by violence, at various parts, as by means of projectiles, of puncture, of a blow, of cautery, &c.

They include things the most dissimilar, as fish- and other bones, fruit stones and seeds, coins, rings, natural and artificial teeth, straw, ears of grain, pencils, needles, tobacco-pipe fragments, dagger and sword points, knives, gunshot materials of all kinds, fragments of dress, of glass, pigments.

These foreign bodies are often got rid of, sooner or later, through the natural channels. Occasionally, however, they abide long—it may be for life—without occasioning serious annoyance, and are afterwards found to have become isolated within a callous exudate-capsule.

In other cases, they give rise to various, more or less perilous symptoms, and not unfrequently prove fatal. Thus, they may act as plugs, or injure in many other ways every variety of organ. Again, they may induce and sustain inflammation and ulceration to the exhausting point.

A certain interest attaches to the migrations of foreign bodies, as now and then witnessed in the case of needles, grain-ears, and bullets; these being, after a longer or shorter interval, discovered or, perhaps, spontaneously ejected through suppuration, at parts of the body remote from the point of their introduction. These migrations are sometimes the result of gravitation, as in the case of bullets. At other times they are obviously quite independent of this motive power.

C. PARASITES.

Under this generic term we comprehend such formations, infesting the organism both within and without, as represent independent entities, either from the vegetable or animal kingdom. Their investigation belongs to pathological anatomy in general, but especially so, inasmuch as the presence of parasites not only implies previous, but also engenders new, morbid conditions. Moreover, they merit a place in this chapter because it is daily becoming more clear that they are not the production of a *generatio æquivoca* out of diseased organic matter, but that they enter into the organism from without, and find there a soil appropriate for their subsistence and growth.

Parasites are introduced into the organism either as seeds, as ova, or in a more advanced condition,—to germinate, become developed, or grow, in or upon the organism. Nor is it less evident that certain pathological states determine a disposition, not exactly to the generation, but to the evolution and redundant growth of parasites, for which they furnish the necessary conditions. Thus, parasite plants (fungi) readily and com-

monly germinate in particular exudates upon mucous membranes, whilst upon normal mucous membranes their sporules remain undeveloped.

Parasites become pernicious in various ways.

I. PARASITE PLANTS (Epiphytes, Entophytes).

These all belong to the lowest forms of plants, the fungi, and unless collected together in redundant growth, they are too minute to be cognizable with the naked eye.

Respecting their origin by propagation through sprouts and sporules hardly a doubt can exist, and as little as to their translation upon and into the organism; although only in a few instances has it been possible to certify this by direct evidence.

It is obvious that certain conditions are requisite for the harboring and the evolution of these germs. This often manifestly consists in pathological conditions, and, at the same time, often in processes of decomposition (fermentation, putrefaction). In the great majority of instances, however, we are in the dark concerning those conditions, and the success of our experiments is dependent upon chance. The former contingency is exemplified in fungi upon muco-membranous exudates, sloughs, and upon mortifying patches of the common integument.

The relation of the vegetable parasite to the concurrent morbid condition varies. The latter sometimes stands in that of a pre-existent state, favorable to the development and multiplication of the fungi; at other times the parasite, harbored through influences unexplained, may become the cause of textural disease; for example, inflammation, suppuration, decadence and loss of hair, &c.

Herewith, the injury they inflict upon the organism terminates. Still they may, where they vegetate extensively, become further mischievous by increasing or specifically modifying some process of decomposition. We may instance the fungi of aphthæ.

If we except the torula cerevisiæ in the contents of the stomach and intestines, the torula of diabetic urine, and perhaps, Goodsir's sarcina ventriculi (possibly an infusorium[1]), parasitic plants, in man, affect the common integuments and mucous membranes only.

With their buds shooting out into more or fewer long, linked, branched threads, they present the form of the thread-fungus.

1. FUNGI UPON AND WITHIN THE COMMON INTEGUMENT.

The most important are :—

(a.) The *mycoderma* in tinea favosa (Schönlein, Gruby). Shut up in splitting capsules, it constitutes the skin-imbedded favus. These fungi, like the torula cerevisiæ, present in their most simple form, roundish or oval cells, and these put forth buds, which shoot out into simple or branched threads. The *favus*-fungus belongs to the genus *Oidium* (Linck), and according to Müller greatly resembles the oidium aureum of wood. Or, according to Corda, it may, together with all thread-funguses, which fructify by simple separation of their links, and in which every link may become a spore, be taken, along with yeast funguses, into the great genus *Torula*.

[1] [It is now, however, pretty generally admitted to be an alga.—ED.]

There is as yet no certainty as to the part played by these thread-funguses. Attempts at inoculation have hitherto failed, with the exception of one experiment made by Remak.

(*b.*) *Fungi* in the *root-sheath* of the hair in *sycosis* [mentagra, Gruby]. They collect around the hair itself within the root-sheath, and are marked by redundant spore-formation. The spores are spherical and the thallus-threads frequently contain in their interior little granules.

(*c.*) Fungi in the interior of the hair-roots [Gruby]. In alopecia circumscripta, areata [porrigo decalvans], the falling out of the hair is caused by a thread-fungus, called by Gruby, on account of the minuteness of its spores, *microsporum*.

(*d.*) In plica Polonica, Günsburg has detected, in the hair-bulbs, a fungus which differs from that of favus.

(*e.*) In Pityriasis versicolor, Eichstedt has discovered a thread-fungus.

Fuchs, Klenke, Helmbrecht, have observed a fungus formation in lepra alphoides, and inoculated it with success.

Langenbeck met with a fungus in crusta serpiginosa.

Finally, the mould formations upon sloughing ulcers, and in senile gangrene, come under this head. They are both frequent and copious.

2. FUNGI UPON MUCOUS MEMBRANES.

These are very often found upon the mucous membrane of the mouth, the pharynx, the œsophagus, the intestinal canal, that is to say in fibrino-croupous, and especially in corroding, aphthous exudates. Aphthæ and diphtheritis of the mouth and throat; croupous exudates in the same localities, in florid phthisis; croupous exudates in the larynx, œsophagus, &c., in the sequel to typhus.

They are assuredly not the morbific agent. The croupous exudates upon which they vegetate are cognizable to the naked eye, for those familiar with the subject, by a peculiar character, consisting in a viscid, curdlike turgescence, dingy yellow or tawny discoloration, and a broken or jagged aspect.

The fungi resemble those of favus. The thallus-threads are, however, mostly much longer, more slender, and have frequently at their free extremity protuberances replete with minute granules (spores). They often form very compact, felt-like tissues.

Amongst these funguses are, no doubt, to be counted the fungus Noma, of Klenke; those found by Bennett in the sputa and lungs, in a case of pneumo-thorax, as also in the black sordes upon the teeth, in typhous patients; again, those seen upon so-called typhus-ulcers in the intestines; lastly, the mould such as we ourselves once observed upon an old blood-clot, unattached within a bronchial sac.

II. PARASITE ANIMALS (Siebold).

Parasite animals are divisible, although not strictly so, into ecto-parasites (epizoa), and into ento-parasites (entozoa). The former infest the surface of the body, the latter its different cavities and parenchymata.

Some of them are parasitic during their entire existence; others only

at certain periods of it. For this purpose the latter migrate, and enter into various metamorphoses.

Some of them inhabit exclusively certain definite parts of the body, both cavities and parenchymata, others on the contrary occur in various regions of the body, and in great numbers all at once. All this is contingent upon their habitudes, and perhaps still more upon the mode in which they obtain access to their place of abode.

With regard to the difficult question of their origin and propagation, modern researches in the least promising domain, namely, of the helminthes, have pretty well succeeded in subverting the older doctrine concerning the generation of parasites, and their relation to the animals which they infest.

They get introduced into the organism as ova, as larvæ, or even as developed creatures; and wherever they meet with a nidus congenial to their nature, live and thrive upon it. For this habitation to last, however, a peculiar disposition on the part of the subject is no doubt indispensable. In the different parasites this sort of predisposition differs materially. Much is assuredly not required to incur a visitation of ascarides. On the other hand, notwithstanding the extensive dissemination of the ova of the helminthes, the disproportionately small number of persons affected with worms; the circumstance that, under certain conditions (for example, disease), worms for the most part, if not altogether, abandon the individual they had infested; and lastly, the fact that different kinds of worms are proper to different animals; testify to the necessity of the peculiar disposition adverted to, existing in persons affected with worms.

1. INFUSORIA.

The most frequent are the *vibriones*, in purulent and other protein-fluids in the progress of decomposition. Donné has detected a vibrio in the pus of chancre, and rated it beyond its worth.

In pus, the *vorticella*, and also the *colpoda cucullulus* (Vogel) occur.

The *trichomonas vaginalis*, detecting by Donné in the vaginal mucus of syphilitic females, is probably not an infusorium, but a misshapen ciliary cell from the uterus or the tubes.

Lastly, we have to cite the *hæmatozoa* occurring in the blood; if they be not rather the embryones of worms, which is probably the case with many of them.

2. INSECTS.

Besides the various *flies* which infest putrid ulcers with their ova and maggots, and the exotic [still problematic] *œstrus hominis*, we have the *flea*, the *lice*, and the *bug*.

(*a.*) *Pulex irritans*, the common flea.

(*b.*) *Pulex penetrans*, the sandflea, 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.

Of *lice* there are—

(*a.*) *Pediculus capitis*, the head-louse.

(*b.*) *Pediculus pubis*, the crab-louse, infesting, the scalp excepted, every hairy part, and penetrating the skin with its head.

(*c.*) *Pediculus vestimenti*, the clothes-louse, infesting parts of the body devoid of hair, and uncleanly vestments.

(*d.*) *Pediculus tabescentium*, the louse of wasting disease, in which it occurs in great multitudes. The notion, however, that there is a disease in which lice are generated beneath the skin, is without doubt fallacious.

Of *bugs*, we have only to mention the ordinary bed-bug, *cimex lectularius*.

3. ARACHNIDA, ACARINA.

(*a.*) The itch-mite, *acarus scabiei*, *sarcoptes hominis*, punctiform, from a quarter to half a millimetre long, ovoid, garnished with transverse, bandlike, dorsal striæ, and with central, acuminate warts; anteriorly a bristled proboscis, prolonged inferiorly to a band upon the thorax; four bristly fore-feet terminating in disk-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 a narrow inspection, discoverable 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.

Researches into the natural history of this mite, together with the results of extended experience, prove beyond a doubt its relation to itch as its sole cause.

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-jointed 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 set up inflammation, and thus give rise to the acne pustule.

4. INTESTINAL WORMS.—HELMINTHES.—ENTOZOA.

Restricting ourselves here to the consideration of such as are peculiar to man, we would preface our special description of them with the following general remarks:

(*a.*) Intestinal worms, *in their consummated development*, are all provided with organs of generation. Those in which the latter have not been demonstrated, are propagated by buds or by offshoots, if they be not imperfect, that is, either larvæ or diseased animals. As opposed to the doctrine of equivocal generation, these facts are important, if we consider:

(*b.*) The migrations and the attendant metamorphoses of the helminthes.

The migrations of the helminthes consist, first in the search for a suitable animal to inhabit, and in introducing themselves into it, when found, through channels formerly unthought of. Secondly, they consist in abandoning the animal dwelt in, for the purpose of casting their ova under favorable conditions, then in passing through one of their metamorphoses, and lastly in searching for another animal for their habitation. They pass, under various phases of development, for the most part through natural orifices of the body, more especially into and out of the intestinal canal. Their occurrence, however, even in the parenchymata, is intelligible upon grounds of direct experience. As illustrative of this, the larvæ of cercarioid trematoda, and of the tetrarhynchi, have been observed to migrate through the parenchymata of mollusca and fishes. It is also deserving of notice, in this place, that helminthes may reach, and settle in any parenchyma through the circulating channels, probably by boring for themselves a passage into the bloodvessels of the intestinal canal. This applies to the nematoid, thread-like animals found by Valentin, Vogt, Gruby, Ecker, and others, in the blood of frogs, dogs, and ravens, and probably representing the embryones of helminthes.

This migration of the helminthes may involve frequent aberrations, and these in their turn many phenomena, which an extended inquiry will perhaps correctly set down to a morbid condition. We refer more particularly to the encysting, the atrophy, and the deformity of certain helminthes; amongst others, of the trichina spiralis, and the tænioid cystica.

(*c.*) The metamorphoses of the helminthes, coincident with their migrations, are of the greatest interest. They constitute a circle of generations, which Steenstrupp, following up the investigations of other naturalists, has pointed out in the trematoda (as in the medusæ, bulb-polypi, and salpæ). A parent animal produces a brood altogether dissimilar to itself, nor identified with it until after three or four generations. These intervening generations of larvæ—these pro-nutrices and nutrices—form without sexual mediation, and are the source of the numerous fallacies taught by the older helminthologists.

(*d.*) All this accords perfectly well with the strict limitation of certain worms to particular countries. The most striking example is afforded in those two riband-worms, the botryocephalus latus, of Russia, Poland, Prussia up to the Vistula, and Switzerland; and the tænia solium of the remainder of Europe.

(*e.*) On the other side, the doctrine of the origin of the helminthes out of intestinal mucus and the like, has not a single point of real evidence in its favor. A disposition to worms exists only in so far as an

organism abnormally nourished offers to helminthes, introduced into it from without, a nidus well adapted for their development.

In mankind the following helminthes occur :—

NEMATOIDEA, ROUND WORMS, THREAD WORMS.

Filaria medinensis, the thread or Guinea-worm, of 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 tropics of the Old World, but especially to Guinea; inhabits the subcutaneous areolar tissue, especially 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, where it tarries in the areolar tissue for a considerable time (several months), after which it again perforates the skin from within, in order to disburden itself of its offspring, or in order, it may be, to migrate for this purpose altogether. These proceedings are attended with inflammation and ulceration, and great caution is recommended, in any attempt to extract the worm, to avoid tearing it; either the elapsing brood or other contents of the worm, having an erosive property which tends to aggravate the said processes. It would seem that, as yet, none but females have been observed. Accordingly these must have introduced themselves in the impregnated state.

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

It infests the cæcum, singly, and also frequently in multitudes [especially, it is true, 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, however, not developed in this locality.

Ascaris lumbricoides, the cylinder worm, a widely disseminated intestinal worm, from five to twelve inches in length, cylinder-shaped, tapering towards both extremities, especially towards the anterior; having four longitudinal striæ, two of which are more strongly pronounced; densely marked with transverse striæ; semi-diaphanous, so that the intestinal canal and the organs of reproduction are transpicuous. The head, divided from the body by an annular groove, displays three little nodules, or rather valves, which encircle the mouth. The caudal extremity, especially in the male, is incurvate. Sexes distinct, the male being smaller and narrower, and having at the caudal extremity a thin, capillary, sometimes double penis. The female is larger, and exhibits at its upper third a fissure from six to eight millimetres long, as the orifice to the organs of generation, which contain ovaries and oviducts of enormous length.

It infests the ileum, often in extraordinary number, in groups and conglomerate masses. A brood is never met with; the ova, therefore, be-

come hatched extraneously to the human body, to remigrate thither afterwards, as the living brood. It gives rise to the well-known worm symptoms. The perforation of the intestine, however [migration extraneous to the intestinal membranes], and its sequelæ are, to say the least, extremely rare.

Oxyuris vermicularis (the *Ascaris vermicularis* of Rudolphi), the hook-tail, maw-worm;—a little, thin, white worm. Sexes distinct. The male very rare and small, from one to one and a-half millimetres long, with spiral convoluted tail: annulate; with a tail terminating in a fine transparent point. The head of either displays 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.

Strongylus gigas [Pallisadenwurm]. Giant strongle; a very large, cylindrical worm, of from five inches to three feet long, and from two to six lines in thickness; when recent, of a fine red color. Sexes distinct; the male smaller, more tapering towards both ends; annulate, with shallow, longitudinal grooves; head obtuse, with six papillæ; at the tail extremity, a funnel-shaped pouch, out of which protrudes a very thin penis. The female, larger, with obtused caudal extremity, and near it the vulva.

Inhabits the kidneys; is rare both in man and in brutes [found in the dog, the wolf, the marten, the horse, &c.]

To these may be added the following nematoda and nematoid pseudoparasites, some of them being very rare, or even but once met with.

The *filaria bronchialis*. [*Hamularia lymphatica*, Treutler—*H. subcompressa B.*, once seen by Treutler in a degenerated bronchial gland in the human subject.] *Filaria oculi humani* [in the liquor Morgagni and in the cataractous lens, Gescheidt, Nordmann]. The *filaria* in the blood [Klenke]; the *anchylostoma duodenale* [Dubini, in the duodenum]; the *spiroptera hominis* [Barnett, in the urine]; the *dactylius aculeatus* [Curling, in the urine]. Finally the *encysted nematoda*.

Trichina spiralis, an incarcerated worm, which one might be tempted to class intermediately between the nematoda and the cystica, were it not extremely probable that it is only a strayed nematodon which, without coming to maturity, encysts itself, perishes, and cretifies within a second cyst thrown out from the textures.

The worm is enclosed within a double cyst, an external one, mostly lemon-shaped, and an inner, oval one; the space of the first, at its two ends, being filled up with very fine dark granules. Both consist of a homogeneous, faintly granular structure; the former being about one fiftieth of an inch long, and one ninety-fifth broad, the latter one seventy-seventh long. In the inner cyst, amidst a more or less 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 twenty-fifth to one thirtieth of an inch long, and about one six-hundredth broad, lumbricoid, thread-like at both extremities, although more pointed at the one than at the other. It possesses internally a winding canal,

interpreted as intestine, and a granular organ, the designation of which, as an ovary, is without doubt erroneous.

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

The *Trichina spiralis* inhabits the voluntary [striated] 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 parallel to the course of the muscles. [Hilton, Owen, Blizzard, Henle, and others.]

TREMATODA, SUCTION-WORMS.

Especially characterized by their peregrinations and metamorphoses.

Distoma hepaticum, and *D. lanceolatum*, Liver-fluke ; flat, melon-seed or lancet-shaped, soft worms, of a yellowish-white color, 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.

The *Distoma hepaticum* is the larger, being from four to eight or to fourteen lines long, and from one and a half to six broad, with a branched intestinal canal.

The *Distoma 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, rarely of man. The *D. lanceolatum* has only once been met with in the latter. In brutes they occur in great multitudes, obstructing and dilating the gall-ducts.

Distoma oculi humani. A minute distoma, once met with in a child between the cataractous lens and its capsule.

Polystoma pinguicola, *Hexathyridium pinguicola* (Treutler). An inch long and from two to three lines thick, oval, superiorly convex, inferiorly depressed worm, with six pores at its head extremity, and a larger abdominal aperture anterior to the tail. Found once by Treutler in the fat of an ovarian fat-cyst.

Polystoma venarum, *Hexathyridium venarum* (Treutler), probably a pseudo-parasite.

CESTOIDEA—TAPEWORMS.

These 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 become developed externally to the animal they infest, to re-immigrate subsequently.

In mankind there occur :

The *Tænia solium*, *T. vulgaris*, *T. cucurbitina*, the ordinary tape-worm, long-jointed tape-worm, chain-worm ; a white, or yellowish-white worm, twenty feet long and beyond it, anteriorly thin, roundish,—pos-

teriorly flat, and from three to six lines broad,—jointed. The joints are flat, square, 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 (vide *Cystica*).

Inhabits the small intestine in man, almost in all districts, except where the botryo-cephalus occurs. The belief that it only occurs singly in man is quite adverse to experience. We have discovered nine of them in the corpse of a lad. It occasions the well-known annoyances, but no visible anatomical mischief.

Botryo-cephalus latus, tænia lata, the broad or broad-jointed tape-worm, resembles the last in many points, equalling it in length, and being in like manner jointed. Its joints are usually broader than those of the *T. solium;* this alone, however, cannot pass for 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 the *T. solium,* exhibits no suction-pores, but two longish grooves.

Inhabits the small intestine in man, but is strictly limited to Russia, Poland, Prussia [trans Vistulam], Switzerland, and to the South of France. If it occur elsewhere it is assuredly imported from one of those countries.

It rarely parts with single joints or links, but usually with a greater or lesser chain of them.

CYSTICA.—VESICULAR WORMS.

In the formation of their head, these resemble tape-worms to such a degree, that even in 1836 Johannes Müller proposed to unite them in a single order, with two subdivisions. Light has, however, been since thrown upon the subject, which warrants us in going a step further, pronouncing these cystica with tape-worm heads to be in truth nothing more than *errant cestoda*, which, owing to their deviations, have sickened, declined, and remained sexless.

The lime corpuscles found upon them, and especially upon the cysticercus, are the same as those occurring upon tape-worms. They have been erroneously held to be ova, and in reality rather represent an outer skeleton formation. These cystica, within textures, are almost always distinctly encysted; that is, shut up within a capsule effused from the textures. In free spaces,—for example in the ventricles of the brain, this is not the case. This adventitious outer cyst is not to be confounded with the cyst proper to the animal itself. They frequently perish, especially through inflammation of the external cyst, being either mechani-

cally crushed by, or corroded and destroyed in, the product. In the sequel, the complicated contents of the outer cyst, after having suffered many changes, progressively thicken, and eventually cretify, *en masse*, within the shrivelled capsule.

The unequivocal proof of the previous existence of an animal in such obliterated cysts is furnished by débris of the animal cyst; by hooklets, from the coronet of hooklets, which have resisted the corrosive agency; and lastly, by the presence of the lime corpuscles before alluded to.

In man occur:

The *cysticercus cellulosus*, consisting of a conical, snow-white, transversely rugous body, and of a vesicle which constitutes 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 a 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, that is 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 than the others, whilst both are so disposed that the larger and smaller hooklets alternate with each other.

The above-mentioned transversely wrinkled, anterior portion of the creature appears as an almost structureless, feebly striated membrane, to which a profusion of fine, black-contoured molecule adheres. It is, moreover, studded with a multitude of roundish or oval, whitish, smooth, sharply contoured, shining, lesser or bigger corpuscles, of from one-eightieth to one-thirtieth of a millimetre in diameter. They are most numerous about the middle part; near the neck and head their number greatly diminishes, whilst, close to the caudal vesicle, they suddenly and entirely disappear. They lie superimposed in several layers, those of the outer stratum being only loosely adherent to the animal, so that they may be very easily scraped away. Treated with hydrochloric or with acetic acid, they dissolve under the copious development of carbonic acid, leaving an organic base-substance behind. In the solution, oxalic and sulphuric acids create a precipitate.

The caudal vesicle consists of the same homogeneous, indeterminate, granulated mass, besprinkled with countless small and larger fat-molecules. The contents of the caudal vesicle consist of a watery, neutral fluid, holding but a scanty portion of albumen.

Wherever the cysticercus occurs in textures, it is inclosed within a second cyst of fibrous texture. When magnified it appears as a delicately-fibred membrane, permeated by delicate blood-vessels, and easily rendered transparent by acetic acid. Where the cysticercus occurs free within a cavity, as within the ventricles of the brain, it is uninvested, showing the outer cyst, in other localities, to be 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 liquefies, afterwards condenses, and eventually settles into a cretaceous concrement. Meanwhile the outer cyst shrivels and dwindles into a thick-membraned capsule, for the isolation of the said concrement.

The cysticercus cellulosus occurs in the brain, in the striated muscles, including the heart, and in the areolar tissue. It also occurs, free, without its outer envelope, 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 the case has happened of its proving fatal by setting up inflammation in its vicinity.

Echinococcus hominis acephalocystis (Laennec). The relation of both to each other, and the import of the last-named animal in particular, will become manifest from the following *description :*

(*a.*) *Echinococcus.*—Within a sac of fibroid texture is inclosed a solitary, independent, thoroughly distended vesicle, containing a limpid, serous fluid ; or else inclosing, as a parent vesicle, other similar vesicles of various size, in various numbers, spherical or flattened by mutual compression, either floating at large in the contained fluid, or sessile upon the inner membrane of the said parent. Its size varies from that of a vesicle just cognizable, and as big as a poppy- or a millet-seed, to the magnitude of a goose's egg and more. In number it may amount to hundreds, so that the serous contents of the parent vesicle are reduced to a minimum. Generally speaking, the lesser filial vesicles are sessile, whilst the larger ones are free.

In very voluminous sacs it is common to find that the parent vesicle appears to be wanting. Either it is mixed up with the younger vesicles, split up, collapsed and dissolved into scattered shreds, or else it has disappeared in the excessive attenuation consequent upon its enlargement.

In their unimpaired vegetation, these vesicles are filled to distension, are elastic, and impart to the touch a sense of tremulous fluctuation, as does the parent cyst replete with them [hydatid tremulousness]. They consist of a substance resembling coagulate albumen, separating into several layers, partly diaphanous, partly white and opaque, frequently accumulated in the inside to considerable thickness, and into gibbous projections. Moreover, they contain a limpid serosity identical with the contents of the parent cyst. When the vesicle is punctured, this fluid gushes forth in a column, and on an incision being made, the parietes of

the vesicle become suddenly inverted. The substance of the latter is a stratified, homogeneous, very fine-granular, structureless mass, whilst their contents exhibit a few lustrous fat-drops, some scattered or agglomerate, elementary granules, and glebous coagula.

These vesicles occasionally contain others similar, of a third, and the latter again in rare instances of a fourth generation.

On a narrower inspection of the inner surface of these vesicles, we perceive in many of them, a whitish, opaque, gritty efflorescence, whilst with the aid of the microscope we here discover densely-nestled animalcules, which prove, by the most various changes of shape, that they long continue to live on in the dead subject. A few of them are even found free in the above-mentioned fluid.

This entozoon is from one-ninth to one-third of a millimetre long, and from one-twelfth to one-fourth of a millimetre broad. It 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 creature is transversely striated. The posterior termination is a transverse cleft, in which is inserted a cordlike formation, by whose means 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.

In its developed state the creature appears in the above form. It is met with, however, under various other shapes. Thus 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, or of a pitcher, or even of a horse-shoe.

The abode of this echinococcus in mankind is, according to our own experience, invariably internal to, and never external to, the vesicles.

(b.) *Acephalo-cyst.* Under this term we at this day understand nothing beyond those vesicles which we have just described as being inhabited by the echinococcus, but which are in some instances sterile. The above name has been given to this formation in order to designate that supposed independent vitality which the absence of organs still renders problematical. The Acephalo-cyst, which Blainville reckons amongst the "monadaires," and Kuhn compares to Agardh's protococcus, with its multiplication by buds, is in our own opinion not to be held separate from the echinococcus, although the precise relation between the animal and the vesicle is by no means clear.

The relation of the primary acephalo-cyst [the echinococcus-vesicle] to the outer cyst, is analogous to that of a new growth incapsuled by exudation from the surrounding textures.

1. W. Griffith has examined acephalo-cysts and their contents. The transparent fluid, of 1·008 sp. grav., coagulated readily by heat or nitric acid, and contained an inconsiderable amount of fat. A thousand parts yielded fifteen parts of solid ingredients, principally common salt. They left 0·85 per cent. of this salt, a little sulphate of soda, a trace of phosphate of lime, and some albuminous extractive matter, but neither cholesterine nor alkaline phosphates. The envelopes of the hydatids left, when

dried, a brown residuum, which dissolved with a deep brown color when boiled with hydrochloric acid, but was not again precipitated on the addition of an alkali. When moist, they dissolved in hydrochloric and in nitric acid, but the solutions were precipitated neither by ferro-cyanide of potassium, nor by tincture of galls. Nothing was dissolved by boiling them in water, for neither by tannic nor by nitric acid was either any precipitate formed, or the fluid gelatinized. When boiled with carbonate of potash, the dried membranes were dissolved with brown coloration, but without any accompanying development of sulphuretted hydrogen, nor any precipitation on the addition of an acid.

Acephalo-cysts, together with the creatures that infest them, are extremely liable to destruction, through hypertrophy, atrophy and consequent perforation of their external coat ; but most particularly through inflammation of the latter with its products.

It is not a rare thing to find, within a sac, individual vesicles imperfectly filled, or collapsed, with walls transparent, tumefied, gelatinized, or even degraded to a smeary mass. The contents of such vesicles are turbid. They consist partly of fat-globules with a fine pulverulent point-molecule in great abundance, and the débris of broken up echinococci. Occasionally this conversion affects most, if not all of the vesicles. They burst or rather open out, owing to the increasing tendency to dissolution, until at length the entire contents of the parent cyst are rendered turbid.

The inflammation of the outer sac, a frequent occurrence, is important. It bears the character of inflammation of a sero-fibrous membrane, and throws its products, for the major part, upon the inner surface and into the cavity of the cyst.

It is in many instances to be regarded as a fortunate event, leading as it does to the disruption and extinction of the acephalo-cyst, with its inhabitant animalcules, and in due time to the shrivelling and decay of the entire sac. The contact of the acephalo-cysts with exudate, and the reception of the latter through endosmosis into the walls and cavity of the acephalo-cysts appear to be amongst the most ordinary causes of their dissolution. After the effusion, gradual resorption of a portion of the contents—that is, of the original serous fluid, and of the exudate—follows, whilst another portion thickens to a grayish, unctuous chalk-pap, and eventually cretifies altogether. The sac shrivelling commensurately with the diminution of its contents, becomes obliterated in such wise as ultimately to inclose a mass consisting of variously superimposed residua of acephalo-cysts (echinococcus-vesicles) and of the said chalk-pap or concrement.

It is not unfrequent for an intense inflammation to terminate in ulceration of the sac, so that an abscess, inclosed within the implicated parenchyma, takes its place. This, together with consecutive suppuration in neighboring textures, may lead to the opening of the sac into another adjoining one; or to its opening externally : or into one or other of the great serous sacs ; into the intestinal canal ; into the urinary cavities or passages ; the gall-ducts, &c. The direction in which such an abscess empties itself decides the question as to the favorable or the unfavorable issue of the case.

The echinococcus and acephalo-cyst are particularly frequent in the liver, less and less so in the subperitoneal, areolar tissue, and in the peritoneum, in the omentum, in the striated muscles, including the heart, in the brain, in the spleen [mostly in concurrence with others in the liver], in the kidneys; very rare in the lungs and bones.

Not unfrequently they occur in several organs simultaneously. Thus they will infest in vast numbers both the peritoneum and the abdominal viscera. In magnitude the sacs may attain, or even exceed the diameter of a foot.

The echinococcus-cysts may become perilous through their volume; and, when present in great numbers, prove fatal through exhaustion and general wasting, as also through the aforesaid inflammatory and suppurative processes.

SPURIOUS PARASITES.

As such are to be reckoned all those foreign bodies reputedly or really, accidentally or designedly, conveyed upon or into the human body; but which are proved either not to infest it in reality, or to be of a nature even manifestly to preclude a parasite existence.

We have to bring into this account not alone animal creatures, and various parts of animals and of plants; but also misshapen, diseased textural parts of the organism, or products of disease. Such are:

1. Animals and parts of animals dead or alive, really voided by stool or rejected by vomiting, such as the larvæ of flies received into the stomach with food in a state of decomposition, or accidentally or designedly added to the matter so evacuated.

2. A great variety of other bodies of the descriptions adverted to.

Amongst the spurious parasites of the present day we may cite—

(a.) The *trichomonas vaginalis* of Donné,—probably a misshapen ciliary cell.

(b.) *Diceras rude* (Rudolphi), repeatedly recognized as the undigested seeds of mulberries.

BLOOD DISEASES—DYSCRASES.

Humoral pathology is simply a requirement of common practical sense; and it has always held a place in medical science, although the limits of its domain have, no doubt, been variously circumscribed or interpreted at different times. Of late years it has met with a new basis and support in morbid anatomy, which, in the inadequacy of its discoveries in the solids to account for disease and death, has been compelled to seek for an extension of its boundary through a direct examination of the blood itself.

Not alone has pathological anatomy demonstrated the existence of blood diseases in unlooked for detail; it has at the same time solved a problem of the weightiest import. It has, we think, decided in favor of

a humoral pathology, by demonstrating a primitive anomaly of blaste-
mata ; by demonstrating the endogenous impairment of the blood within
the vascular system, in the inflammatory process, as the basis of the varia-
tions in exudates [blastemata] ; lastly, by demonstrating the dependence
of local morbid action upon pre-existent impairment of the general circula-
tion.   Our attention will be here directed to diseases of the blood in its
totality, and to local dyscrasial processes, with inflammation at their head,
only in so far as these offer the basis and starting-point for consecutive
disease of the entire blood-mass.   It is remarkable, however, and no less
important for practice than for science, that the essential forms of these
local dyscrasial processes,—perhaps of all local dyscrasial disease,—
occur, likewise, as primitive affections of the entire blood-mass.   This
is proved by the varied character of the products of the inflammatory
dyscrasial process, and a comparison in detail of these products with
anomalies of the general blood-crasis.   Thus, primitive pyæmia, fibrin-
crasis, sepsis of the blood, severally occur independently of all local
beginning, and of all infection.

There are, indeed, two ways of investigating and recognizing blood-
diseases : first, the *anatomical examination* of the blood in the dead sub-
ject, or of blood obtained during life through spontaneous or artificial
hemorrhage ; and secondly, *chemical analysis*.   Both kinds of investi-
gation should be supported, and the results controlled, by a concurrent
examination of the secretions and excretions, of the general condition of
the solids, and of new-formations, especially of such exudates as are the
offspring of inflammation.

In fine, both kinds of research should go hand in hand.   For, although
a deeper insight into the changes suffered by the blood may be reserved
for chemistry, it must needs be based upon sound anatomico-humoral
premises.   Up to the present day chemistry has not taken this duly into
consideration, so that as yet this science cannot be said to have far ex-
celled the achievements of a circumspect anatomical survey, notwithstand-
ing the limited resources at the disposal of the latter.

Upon the chemical pathologist we would strongly urge an unremitting
prosecution of his researches.   We would recommend him to direct his
labors more particularly towards ascertaining the precise character of
the impairment suffered by the proximate ingredients of the blood, and
of the anomalies impressed upon its elementary composition.   The inte-
rests of hæmato-pathology would after all, perhaps, be best served by the
examination, in the above sense, of blood taken from the dead subject,
the diagnosis of the case having previously received the light of a general
post-mortem examination.

Our own task in these pages will be limited to establishing a purely
anatomical pathology of the blood ; we shall therefore restrict ourselves,
as nearly as possible, to anatomical facts, although without neglecting to
avail ourselves of the collateral support of such chemical data as may be
relied upon at the present hour.

It is the business of pathological anatomy to determine both the phy-
sical properties of the blood in its totality, and also the relative quantity,
and more especially the quality, of its more immediate components.

The two main components which come peculiarly within its province are, first, those essential form-elements, the *blood-globules ;* and secondly, the spontaneously separating, coagulating, solidifying *fibrin,*—that component which, owing to its varying tendency to become organized, is, in an anatomical sense, the most important of all. We will here *summarily refer to what has been said in the introduction to the doctrine of blastemata and to the section on exudates,* and then proceed to treat of *blood diseases* in what would appear to be their most natural order. The subject is, however, so intimately allied to that treated of in the chapters referred to, that a certain amount of repetition will, perhaps, be unavoidable in the following pages.

Affections of the blood are, like those of the solids, either *primitive* or *consecutive.* And again, the former, equally with the latter, suggest an inquiry as to whether they result from an immediate influence of the morbific agent upon the blood, or are determined by the nervous system, as the actual percipient, alienated both in matter and in function. This question can, however, hardly affect us in this place, since the latter view mainly rests on speculative grounds, and upon the fact that obvious injury to, or sensible anatomical disturbance of, the nervous system sooner or later results in disease of the blood.

The latter are determined in very different ways by anomalies in the solids. Thus, the hindered eliminating activity of an organ occasions retention of effete matter in the blood ; an abnormal plastic process influences the blood crasis, directly or indirectly, through the anomaly in the interchange of matter. Take for example, the infection of the blood within the range of an inflammation.

To diseases of the solids, as local morbid processes in the broadest sense, affections of the blood stand in a twofold relation :

1. *The anomalous crasis is a pre-existent one—the primitive affection ; the local disease a localization thereof—the secondary affection.* The point of localization, apart from the effect of concurrent external influences, is determined by a specific relation of the crasis to certain organs presided over by the nerves. The forms it assumes are chiefly those of hyperæmia and stasis—inflammation, absolute stasis,—exudation, or, without the latter, a product-formation completed within the bloodvessels ; for instance, spontaneous coagulation of diseased fibrin, pus-formation within a greater bloodvessel or within the capillaries of an organ.

The relation of the various crases to the organs and textures, nay, even to particular sections of organs, is manifold. Thus, the croupous fibrin-crases evince a very marked preference for the mucous membrane of the air-passages, and for the lungs themselves ; the typhus-crasis, for the mucous membrane of the ileum ; the exanthematous crases, for the common integument and for mucous membranes.

2. The anomaly of the general crasis is consecutive ; that is, the consequence of a local disease, and especially of local dyscrasial processes, whereof the products are taken up into and affect the general blood-mass. This happens—

(*a.*) Through resorption of the effused products by means of the lymphatics, or immediately into the veins.

(*b.*) Through reception into patent bloodvessels. This process includes the reception of products thrown out into the cavity of larger bloodvessels,—pus, for example.

(*c.*) Most of all, through the off-flowing, and the return into the veins, of plasma degraded in the local process, in a manner corresponding with the quality of the exudate. [See " Relation of the Inflammatory Process to the Crasis."]

It is, however, to be understood that, neither does a dyscrasis necessarily always become localized, nor a local dyscrasial process invariably give rise to a consecutive dyscrasis of the entire circulation. In the former case, a certain degree of intensity of the dyscrasis is no doubt requisite ; in the latter case, the reception of a sufficient quantity of plasma, degraded in the manner aforesaid by the local process, or else of a heterogeneously diseased,—for example, of an ichorous or septically constituted—plasma is indispensable.

Blood diseases are, moreover, either protopathic, whereby we mean developed out of the normal crasis, or deuteropathic, that is created out of another anomalous crasis. [Meta-schematism.] Deuteropathic crases occur in the simplest manner, as impoverishment of the blood in one or more ingredients, drained away by excessive deposition into textures or upon membranous expansions.

Blood diseases are both acute and chronic, and they are marked accordingly by the rapidity or by the slowness of their career. This is contingent upon the character of the dyscrasis, upon its grade, and upon the significance of the organ in which it becomes localized.

They issue :

(*a.*) *In transition to the normal blood-crasis.* This occurs under various conditions, for the most part obscure; for example, under the return of the free function of an organ, under exhausting localization of the dyscrasis in one of the aforesaid processes, or in some secretion. In this way tuberculosis and cancer may lose their *general* import and become local affections, which either go on vegetating under the normal condition of the blood, or enter into a retrograde metamorphosis.

(*b.*) *In transition to another anomalous crasis* [meta-schematism]. Such transitions are multiform, some of them appearing to be necessary conversions when the original crasis is at its acme, others to represent the final wearing out of some component of the blood. With respect to others, however, we are still in every way completely in the dark.

(*c.*) *In death*, not only through overwhelming localization in vital organs, often coupled with palsy of their function; not only through exhaustion of organic matter and of the powers of life, owing to redundant local production; but also, in many instances, through unfitness of the dyscrasial blood for the upholding of processes essential to life, for the maintenance of nutrition generally, but especially of respiration and of the energies of the entire nervous system, both central and peripheral.

1. FIBRIN-CRASES.

The fibrin-crasis occurs in several most important forms and varieties, which the term hyperinosis—as designating a frequent but by no means

necessary and invariable excess in the quantity of fibrin—does not suffi-
ciently characterize. It overlooks the far more important, and, for the
most part, very marked feature of *quality*. However certain may be the
excess of fibrin, its qualitative deviation becomes more and more distinctly
pronounced in proportion as in the series of fibrin-crases the forms recede
from the characters of *true* fibrin. This, with the exception of a few
hints thrown out by Andral, has been hitherto ignored. In this qualita-
tive anomaly, however, the varieties of the fibrin-crases are founded.
Each may be, and very often is, a hyperinosis, at the same time. Still
the qualitative anomaly is the essential point ; and it may, in a *hypinotic*
crasis—that is, in poverty of fibrin—cling to a minimum of fibrin, and
with it manifest at once that peculiar tendency of fibrin-crases to locali-
zation, and a marked peculiarity in the product.

These different fibrin-crases are, as in the sequel their special delinea-
tion will show, manifested by certain anomalies of appearance and struc-
ture. They are distinguished in common by the proneness of their fibrin
to coagulate, and by its deposition, more or less pure, within the vascular
system, from the heart to the capillaries downwards. They are, more-
over, marked by their localization in inflammations which are wont to
affect very vascular organs, such as the lungs, mucous and serous mem-
branes, and areolar tissue.

The fibrin-crases, including pyæmia, tend more particularly to prove
that all those changes which the plasma undergoes in local dyscrasial in-
flammation, and its products or exudates, take place within the general
circulation and by virtue of its own intrinsic relations, not through any
local reciprocation between the blood and the textures inflamed.

The fibrin-crases constitute, generally, the so-called *phlogistic blood-
admixture*, against which, conformably with the view of a quantitative
exaltation of the vital process, the lancet has ever been opposed. Yet
amongst the processes in question, far-sighted pathologists have always
discriminated some in which much bleeding appeared not only needless
but even mischievous ; we refer more particularly to the croupous pro-
cesses. That in these, a qualitative deviation in the constitution of the
fibrin plays the part chiefly deserving of attention, is proved not only by
the more obvious anomalies of aspect and of structure before alluded to,
but also by—

1. The proneness of these crases to become localized, even where the
amount of anomalously constituted fibrin in the blood is very inconsi-
derable ; as, for instance, in the secondary croupous crases emerging out
of the typhus-crasis ; in the tuberculous crasis, in which the last particle
of fibrin is expended in the deposition (localization) of tubercle.

2. The reaction of many congenerous exudates upon their parent
textures,—this reaction consisting in softening and corrosion.

3. The wasting attendant upon hyperinosis, or the predominance of
fibrin, that element of the blood commonly held in an especial manner to
preside over general nutrition. Here the alienation of functional activity
can only be interpreted as qualitative.

We have already adverted to the localization of the fibrin-crases. In
relation to this, it is a question equally interesting and opportune,

*whether the crases regarded as hyperinoses, in inflammations, be deter-
mined by the latter, or constitute the primary and fundamental disease?*
The view received in France, tends to demonstrate the dependence of
the crasis upon the local inflammation; in other words, the symptomatic
character of the crasis. With the setting in of the inflammation and its
increase, the amount of the fibrin is supposed to become augmented.

We are fully convinced that an inflammation obviously called forth by
external causes may, by the abduction of endogenous, and the resorption
of exuded products, give rise to a corresponding crasis, which will be-
come augmented in proportion as the inflammation increases in intensity
and extent. On the other hand, we believe that spontaneous stases are
localizations of a crasis, and stand to it in a dependent—a conditional—
relation. This opinion is based upon the following facts:

1. Every crasis [by no means the fibrinous crases alone, to which, as
the so-called phlogistic, we might be disposed to concede this preroga-
tive] is capable of localizing itself in the shape of inflammation; take for
example, the typhous and the exanthematous crases. The objection that
inflammations, and especially pneumoniæ, arise during the progress of
typhus, is met by the fact that those inflammations with the character
of a fibrin-crasis are based upon a fibrino-croupous crasis—in other
words, that they are the localization of a fibrino-croupous crasis into
which the typhus has become converted. Genuine typhous pneumonia
(pneumo-typhus) does not develope a fibrin-crasis, any more than does
typhous inflammation of the intestinal follicles or of the mesenteric
glands.

An objection of considerable weight against the opinion promulgated
by Andral, is furnished by pneumonia, the very process commonly con-
current with the most marked hyperinosis. We believe that ordinary
pneumonia [with fibrinous product] is, for the most part, the localization
of a pre-existent, that is, precursorily developed crasis, a crasis charac-
terized by an incontestable relation to the lungs, and to the mucous
membrane of the air-passages. Such a view does away with the paradox
that inflammation of the lungs, a disease which, in its intense form, at-
tacks and disables large sections of the lung, should uphold so enormous
a development of fibrin, whilst other lung diseases lead to crases of the
very opposite kind—in a word, to venosity [Hypinosis, Albuminosis,
Cyanosis].

2. Lastly, the argument derives force from the general disturbance
which always precedes a localization, seeming to bear witness to an
alteration in the crasis. And to this may be added, the nature of the
causal influences, which appear to be rather general than local. We
may instance epidemics, climate, weather, &c.

Fibrin-crases become, for the most part, primitively developed under
the conditions of a free respiratory function. Of this fact, striking ex-
amples offer in the tuberculoses, and the setting in of croupous and of
tuberculous processes after childbirth, that is to say, after the release of
the thoracic spaces and of the lungs, resulting from deliverance of the
womb.

Certain fibrin-crases are *primary*, and distinguished by their localiza-
tion upon the mucous membrane of the air-passages [laryngeal,

tracheal, bronchial, pulmonary croup], upon serous and synovial membranes, and in large accumulations of areolar tissue. Others are *secondary*, that is, the consequence or the conversion of other crases, for example, of the typhus-, of the exanthema-, of the cholera-crasis. In these, a *qualitative* anomaly of the fibrin predominates, as shown by this, that, even where but an inconsiderable amount of fibrin becomes developed, localization takes place, and this of an unwonted kind, as, for instance, upon the mucous membrane of the intestinal tract, of the urinary passages, of the gall-ducts, &c. Others, again, are *primitive, spontaneous,* or even *consecutive affections* of the blood determined by infection with analogous substances. They are often epidemical.

Above all other crases, the fibrin-crases, like the fibrin-exudates, are never thoroughly pure. Every portion of morbid fibrin has, associated with it, another portion of less diseased, or even of normal fibrin.

The products of the localized fibrin-crases [endogenous coagulations, and especially exudates engendered by inflammatory stasis] are partly organizable [designed for regeneration, or expended in hypertrophy], partly unorganizable, liquefying, corrosive, purulent, or ichorous. These exudates correspond so completely with the nature of the coexistent fibrin-coagula within the vascular system, that the character of the one may with safety be inferred from an acquaintance with the other.

It is interesting that coagulations in the left heart, that is out of arterial blood, are not alone more decidedly compact, but also more frequent than those out of venous blood. As evidence of this we may cite the incomparably more frequent globular vegetations in the arterial chambers of the heart.

As yet, chemical analysis has contented itself with demonstrating the quantitative excess of fibrin in the blood. According to our own researches, however, investigations are urgently requisite which have for their principal aim to determine the qualitative impairment of the fibrin. An augmentation of the fibrin is always coupled with a diminution in the amount of blood-globules, and, as chemists maintain, at the same time with an increase in the proportion of fat present in the blood. This certainly, however, does not apply to every fibrin-crasis.

Fibrin-crases attended with great exudation, frequently bequeath, as consequent upon the exhaustion of fibrin, a condition of hypinosis [defibrination], and of hydræmia. They eventually prove fatal from this source, if the patient have escaped the deadly influence of paralysis of the organ of localization, or of spontaneous coagulation in important sections of the vascular system ; for instance, in the ramifications of the pulmonary artery. The highest grades of dyscrasial fibrin-constitution, finally degenerate into sepsis of the fibrin, and, indirectly, of the entire blood-mass.

The crasis may also terminate in restoration of the normal crasis, through conversion of the morbid excess of fibrin into nitrogenous substances, eliminated with the urine and perspiration. The fibrin thus becomes largely converted into excrementitious matter.

(*a*.) SIMPLE [ORGANIZABLE FIBRIN-YIELDING] FIBRIN-CRASIS.

It is the attendant upon inflammations with an organizable exudate,—that is, an exudate susceptible of textural conversion. It comprises the inflammations of wounds healing by the first intention; many inflammations of glandular organs, and of serous and synovial membranes which terminate, not in purulent liquefaction of their products, but in gradual resorption or in textural conversion of the latter,—or in the case of pneumonia, in obliteration of the pulmonary texture.

The product of these processes—that is, the exudate-fibrin determined by these processes—answers to the character of fibrin 2. [See "Fibrin."]

The crasis consists in this: namely, that the fibrin, besides increase in quantity, usually manifests, within the bloodvessels, the character of the exudate-fibrin just adverted to; in other words, those qualities which fibrin acquires in certain processes of inflammation. The tendency of fibrin to coagulate is sometimes aggravated into spontaneous coagulation within the vascular system.

The coagula are white, or yellowish-white, compact, frequently holding enclosed a notable quantity of serum. Under a closer inspection they appear as a glebous, fibro-glebous blastema, here and there delicately fibrillated in wavy curls. Through this are interspersed numerous black-contoured, spherical or fibre-drawn nuclei, along with scattered, dull-granular nuclei, and nucleated cells. All the nucleus-formations are rendered more sharply defined by the influence of acetic acid, the blastema itself becoming turgescent and transparent. [See "Fibrin 2."]

To this category belong not a few of the so-called vegetations or fibrin-coagula within the heart's cavities, not a few coagula in bloodvessels of every calibre down to the capillaries, perhaps also the intra-arterial stratiform coagula, and those endogenous depositions which are the primitive source of phlebolites.

Unless these coagula—produced during life—be, in a state of minute subdivision, taken up again into the blood, they enter into a textural conversion.

The crasis is either a spontaneous, primitive, or else a consecutive one engendered by infection of the blood with a product of a corresponding kind.

The dead subject is marked by great cadaveric rigidity, by firm, deep-red muscles, by tense, dry areolar tissue, and by retarded lividity and decomposition.

(*b*.) THE CROUPOUS CRASIS [PIORRY'S HÆMITIS.]

The *croupous crasis* occurs under several forms, which at the same time represent various gradations of disease of the fibrin. Amongst them we find, on the one side, the most marked hyperinoses; on the other, a scanty proportion of fibrin, but that deeply affected in quality. This is manifested first by its augmented coagulability, by a greatly increased tendency to deposition in the shape of intra-vascular coagulation

[in the capillaries, as capillary phlebitis], and, lastly, by acute processes of exudation.

Both the intra-vascular coagula and the exudates are distinguished by their indisposition to become organized, by their early liquefaction, and very often by their corroding, solvent effect upon the textures. Both are opaque, yellow, or of a greenish-yellow, and contain fat. The adhesive property gradually diminishes.

To the latter, namely, the exudates, must be reckoned some, exhausting by their volume and abundance, others inferior in quantity, but indicating, by their tendency to liquefy and by their reaction upon the textures, the deep impairment of the fibrin.

They are often idiopathic, but more frequently consecutive crases, emerging out of others,—the typhous, the exanthematous, &c. In the former case they are marked hyperinoses; in the latter case they are determined by inflammation, and the infection of the blood with congenial products. They constitute the so-called hæmites of Piorry.

In their processes of exudation, they evince a preference for the mucous membranes, especially of the respiratory and of the digestive tracts, as also for the serous and synovial membranes.

CROUPOUS CRASIS *a*.

It is characterized by the following relations of the fibrin in its coagulation and exudation.

The coagula—engendered in the death-agony—are, in the heart, either clod-like, cord-like, more or less compact masses prolonged into the bloodvessels, or where the energy of the heart's systole has been broken long prior to death, and the mortal struggle protracted, membranaceous, lining the heart's cavities or insinuating themselves in the shape of fangs amongst the trabeculæ. When developed during life, they appear, in the heart, as the liquefying so-called globular vegetations; in wide-calibred bloodvessels, as cylindrical and plugging, or as membranaceous, coagula loosely attached to the internal membrane of the bloodvessel; in capillary ranges, as obstructions of the texture varying in circumference. They are either pure fibrin or contain more or fewer blood-globules, incorporated with them during the act of precipitation. In the former case, they are marked by their opacity, by their dull white, yellowish, or yellowish-green coloration. In the latter case, they are likewise opaque, but, according to the amount of contained blood-globules, more or less reddened.

A closer inspection shows the coagula to consist of a glebous-like, fibro-glebous blastema, or of a faintly striated, membranous basement,—like the inner, the fenestrate, bloodvessel membrane, bestrewn with point-molecule, with numerous granulated, grayish nuclei or nucleus-like formations, and with similar granulated cells. All the nucleus formations are uninfluenced by acetic acid, except that a slight shrinking takes place, and that they acquire a somewhat sharper outline. Not unfrequently the entire coagulum seems to consist of these nucleus- and cell-formations, along with a proportion of point-molecule. [See " Fibrin 3."]

The metamorphosis of these coagula consists for the most part in a tolerably rapid liquefaction of the blastema to a puriform, tenacious fluid holding the aforesaid form-elements in suspension.

The exudates, reflecting the hyperinotic condition of the blood, are generally very abundant, even to the exhaustion of the fibrin. They are reddened in a degree conformable with the amount of extravasated blood which they have incorporated,—as, for instance, in hepatization of the lung. Or they are of a grayish-yellow with a slight shade of green, and opaque. Their metamorphosis consists, possibly with textural transformation of any organizable portion, in disintegration and liquefaction of the blastema to a pus-like, tenacious fluid. [See " Croupous Exudate a."]

CROUPOUS CRASIS β.

Its characters are nearly those of the former, as regards the outer aspect of the coagula and of the exudates, only more strongly developed. Thus, the opacity of the coagula and of the exudate-fibrin is more considerable; their coloration, where they do not include blood-corpuscles, more decidedly of a greenish-yellow. Their metamorphosis consists in rapid, puriform liquefaction.

More narrowly scrutinized, the coagula are found to consist of a fine, dense point-molecule, of nucleus- and cell-formations in different degrees of completeness and of assimilation to the pus-cell. These are held together through the instrumentality of an amorphous bond-mass. Engendered during life, they break up—the blastema liquefying—into a tenacious fluid, in which the elements specified are held in suspension; and which, in proportion as the cells predominate, more and more resembles pus. [See " Fibrin 4."]

The exudates, corresponding in character with the usually hyperinotic condition of the blood, are generally very copious, exhausting, of a yellowish or a greenish-yellow tint, imperfectly adherent to the exudation-surface, rapidly liquescent to a puriform fluid, and of a form-composition identical with that of the coagula. [See Croupous Exudate β.]

In both the coagula and the exudates, the basement or blastema connecting the form-elements has lost the fibrillation so characteristic of coagulating normal fibrin, and even the glebe-like structure.

Certain conditions are common to both varieties of the croupous crasis [α and β]; to the latter, however, they apply in a higher degree.

Both become localized in the shape of exudatory processes upon the mucous membranes, especially of the respiratory tract; in early youth, in the larynx and trachea; at a later period, in the bronchia; and from the period of puberty to the end of life, in the lungs [as laryngo-tracheal, as bronchial croup, and as croupous pneumonia]. Upon the mucous membrane of the womb, as also upon the great serous sacs, especially the peritoneum, with or without congenerous puerperal metritis, they become located, as puerperal processes; in the synovial sacs, as acute rheumatism; again, as endocarditis and inflammation of bloodvessels; lastly, in the areolar tissue, the pia mater, the spleen.

They also become localized as the metastatic deposits of capillary phlebitis.

The high grade of the internal dyscrasial influence is no doubt the cause why a protracted stasis is not requisite for product-formation ; why, therefore, the exudation takes place very rapidly ; and why, notwithstanding the great bulk of the exudate and the lax and vulnerable nature of the textures, it is not hemorrhagic ; in other words, why it is not attended with any extensive laceration of the bloodvessels. There is little doubt that the pneumoniæ stated by Hodgkin to enter at once into yellow and rapidly liquefying hepatization, belong to this class.

By long contact, the deliquescent coagula and exudates frequently exert a solvent, corrosive power upon the textures. In this manner they determine fresh inflammation, ulcerous loss of substance, phthisis of the organs, secondary phlebitis, pulmonary abscess, phthisis of the peritoneum, of the pleura, and, along with these, of the abdominal and thoracic parietes.

Or again, they become re-absorbed, or else, owing to the changes attendant upon their disintegration, they undergo, together with partial absorption, fatty conversion, usually followed by inspissation and cretefaction.

One further phenomenon here finds its elucidation, namely, *the milky blood*. This has been witnessed in pneumonia and peritonitis, and we have ourselves encountered it in a developed form in pneumonia, and in very intense inflammation of the spleen. Such blood has been found to contain an excessive proportion of fat ; it is, however, questionable whether this be the sole cause of the phenomenon. We believe the latter to be due rather to the disintegration of croupous fibrin within the circulation, in other words, to the suspension of the point-molecule [the molecular fibrin] in the blood-serum. It is very possible, indeed, that a fatty condition of the blood may contribute to produce the milky appearance ; and not improbable that in some cases of a different kind, for example, in the blood of dram-drinkers, it may be the sole cause.

The rigor mortis manifests itself in the inverse ratio of the magnitude of the effusion, and the same inverse relation obtains between the latter and the intra-vascular fibrin-coagula. The blood is in part loosely clotted ; for the most part fluid ; owing to simultaneous loss of serum through exudation, tenacious ; and of a dark cherry-red [defibrination]. It forms, in every variety of organs, dirty-red hypostases ; death-patches become rapidly and extensively developed ; the liver is dark-colored. The muscles are lax, the parenchymata collapsed, flabby, lacerable, moist, and where there is no hypostasis, pallid.

CROUPOUS CRASIS γ.   APHTHOUS CRASIS.

In the croupous crases hitherto described hyperinosis commonly prevails ; in the crasis we are now entering upon, the amount of fibrin in the blood is for the most part scanty. By so much the more significant must be the anomaly in the *quality* of the fibrin and in the general crasis, and we shall presently see this to be really the case, if we extend the idea of this crasis in a natural order beyond that constitution of the

blood proper to ordinary aphthæ as affecting the mucous membrane of the mouth.

The exudates appertaining to the croupous crases before described occasion destruction of the textures only after long-continued contact with them, and in their secondary phase of disintegration. In the aphthous exudates this effect is produced at once, and as it would seem during the process of exudation itself.

The exudates are yellow, greenish-yellow, dingy gray, opaque, tough products, which, upon superficial expansions, coagulate in a pseudo-membranous form, and, together with the textures, rapidly break up. They are very often remarkable for the exuberant epiphyte-formations [thread-funguses] which take root in them. The disintegration manifests itself in various forms, doubtless connected most intimately with the grade and with the modification of the crasis, from simple corrosion with secondary reactive inflammation in the vicinity [aphthæ], down to fusion to a variously discolored, fetid, ichorous pulp, or to a tough or a friable slough, that tears like soft agaric.

With this the crasis has attained the phase of a putrid decomposition, a sepsis, a necrosis of the fibrin and of the general circulating fluid. The blood is discolored and contains partly tough, partly pap-like, discolored, ichorous, coagulate fragments—as exemplified in diphtheritis, sloughing tonsils, puerperal putrescence of the womb, septic dysentery, &c. The seat of such processes is above all in the mucous membranes, especially of the alimentary tube, of the urinary bladder, of the female sexual organs and their follicles; in open wounds and sores, in areolar tissue, in common integument. Amongst them may be reckoned thrush [diphtheritis]; certain exudation-processes upon the mucous membrane of the intestinal canal, especially of the colon [as a form of dysentery], and of the uterus after delivery; corrosive fibrin-exudates upon the mucous membrane of the urinary bladder; upon external and internal sores; muco-membranous ulcers, especially upon the base of the typhous ulcers; white gangrene of the common integuments [hospital gangrene].

The aphthous crasis is often idiopathic. It is, however, no less frequently consecutive upon other anomalous crases, and upon the typhous, the typhoid, the exanthematous in particular. The blood, reduced in quantity, presents the characters proper to those crases; the few coagula themselves, the characters of excessively croupous fibrin.

The dead body is collapsed, devoid of rigor. The death-patches become speedily discolored to russet and green. Muscles, flabby, pale. Parenchymata, collapsed, friable. The blood, wasted, dusky-red, fluid, with a few villous, broken coagula, marked by opacity, toughness, discoloration, and septic disintegration.

(c.) THE TUBERCLE-CRASIS.

Although there are, no doubt, tuberculoses purely local, a tuberculosis extending through several organs, or even through one entire organ, is invariably the offspring of a tuberculous dyscrasis.

The latter is for the most part developed out of a *fibrin-crasis*. In the dead subject, a hyperinosis of the blood may not be ostensible. Nay! owing to the frequent and voluminous processes of exudation attendant upon the course of a tuberculosis, a poverty in fibrin is perhaps more likely to reveal itself after death. Excess of fibrin has, however, been demonstrated, in such cases, by the chemical examination of blood withdrawn during life, and it may also be inferred from the extent of the tuberculous deposition. Still the hyperinosis is far from being adequate to account for the disease. It is the qualitative anomalies of the fibrin that must determine its tuberculous nature,—anomalies of quite a peculiar kind, which, as we shall see, may modify every one of the fibrin-crases referred to into the tuberculous.

The gray tubercle answers to the character of the *simple fibrin-crasis*. [See " Gray Tubercle."]

The opaque, yellow, lardo-caseous tubercle, marked by its proneness to undergo softening, answers to the character of the *croupous-crasis*. [See " Yellow Tubercle."]

We have remarked of the products of the fibrin-crases that they are seldom unalloyed. The same observation applies to the products of the tuberculous crasis. The products of the one always occur intermingled with elements of the other ; and tuberculous products may even include more or less of organizable elements which form into textures in the ordinary way.

Again, the transitions of the individual tubercle-crasis from one to another are obviously brought about step by step. The croupous tubercle appears but rarely as the primitive tubercle. It is generally based upon a pre-existent gray tubercle, and the croupous tubercle appears as an aggravation of the simple form.

In the fibrin-crases a minimum of fibrin suffices for the groundwork of a qualitative impairment, and this in its amplest sense applies to tubercle. The smallest proportion of fibrin present in the blood takes on the taint and becomes expended, up to the point of complete defibrination, in the deposition of tubercle.

Still these said crases by no means serve to throw much light upon the nature of tuberculosis. They must needs involve a peculiar—a tuberculous—modification, the existence of which is indeed proved by the tuberculization of extra-vascular fibrin in hemorrhage, and also of intra-vascular fibrin-coagula. *In this modification must be rooted :*

(*a.*) The surpassing proneness of the fibrin to deposition, so frequently brought about in quite an insensible manner ;

(*b.*) The assumption by the deposited fibrin of the particular form of tubercle. The granulation of tuberculous products of inflammation upon serous membranes might indeed be ascribed to a separation of the tuberculous portion, due to its great coagulability. Still the very localization of the tubercle-crasis, in such minute and sharply-defined areæ that little granule-shaped exudates are the result, constitutes a peculiarity—a remarkable peculiarity, seeing that the same form of separation—the same form of tuberculization—attaches to the fibrin of hemorrhage and to endogenous vascular coagula.

(*c.*) The fact that blastemata resulting from the tuberculous fibrin-crases do not undergo the metamorphoses—proper to the pure fibrin-exudates—of textural formation on the one side, and of rapid purulent liquefaction on the other ; but throw off their exudate-water in the act of firm coagulation, and tarry for a while in this crude state of consolidation. This respite is of various duration, but at all events exceeds both that proper to textural conversion, and that common to disintegration, in corresponding, *purely* fibrinous exudates.

As the most marked and obvious phenomena connected with tubercle must be specified its high grade of coagulability, and its surpassing proneness to deposition,—to the localization of its crasis. In these are without doubt centred the peculiarities of the fibrin-crases in their tuberculous modifications.

If, in relation to these peculiarities of tuberculous fibrin, we take into account:

(*a.*) The consummate coagulability of arterial fibrin generally ;

(*b.*) Its supreme sensitiveness towards heterogeneous substances, such as inflammatory products, whose reception, for example, in arteritis, occasions locally so rapid an obstructing coagulation of the blood-column, as to obviate any infection of the general blood-mass from that point. [See "Arteritis," vol. iv.]

(*c.*) The ready deposition of fibrin out of arterial blood, as stratiform coagula upon the inner arterial surface, a disease, in its consummate form, peculiar to arteries.

(*d.*) The very common localization both of the fibrin-crases, whether spontaneous or determined by infection, in the shape of exudate, or of endogenous coagulation within the capillaries of the arterializing organs [the lungs], and again the still more marked relation of the tuberculous crasis to these viscera.

(*e.*) On the one side, the pre-eminent development of the pulmonary organ as predisposing to the fibrin-crases in general, and to their tuberculous modification in particular ;

(*f.*) On the other side, the pre-eminent immunity afforded by exquisite venosity and cyanosis, against fibrin-crases, more especially the higher (croupous) grades, and most particularly against the tuberculous crasis, —taking, we say, all these circumstances duly into account, we are forced on to the momentous conclusion that *arteriality*, that is, *the arterial development of the fibrin, pre-eminently constitutes the cardinal character of tuberculosis.*

The qualitative impairment of the blood-fibrin here again, as in the fibrin-crasis of a higher grade, serves to explain summarily the fact, that in individuals with blood impoverished in fibrin,—or even generally impoverished,—tubercle continues to become deposited. It has been elsewhere affirmed, and it is worthy of repetition here, that in such cases *every atom of tuberculo-dyscrasial fibrin becomes expended in the formation of tubercle.* This view clears up the seeming inconsistency of affirming tubercle to be rooted in a fibrin-crasis, whilst florid tuberculosis is found to be associated with a deficiency in fibrin. It is the prevailing notion of mere *excess* in fibrin that we would impugn.

The tuberculous-crasis itself may, by various chances, become modified

through a combination with other dyscrasial constitutions of the fluids, giving rise, at least in part, to the several known varieties of tubercle.

The tuberculous crasis is commonly protopathic. Frequently enough, however, it results from other crases. The conversion of typhus, of exanthematous hypinosis to the tuberculo-croupous crasis is frequent, and of the greatest scientific and practical interest.

The tuberculous crasis results, through exhaustion of the fibrin, in a defibrinated condition of the blood,—in albuminosis. And, again, the albumen—upon which the wasted fibrin may be supposed to have ingrafted its own morbid character—may take up the work of exudation in the shape of acute, albuminous tubercle, of lardaceous infiltration of the liver, spleen, and kidneys,—of general albuminuria ;—hydræmia and anæmia being the final issue.

The corpses of the tuberculous, responding to the long-continued and copious expenditure of protein substances upon tubercle, present general emaciation, with consumption of the fat and of the bone-medulla ;—flabbiness, wasting, pallor of the muscles ; fatty infiltration of the liver, spleen, and kidneys ; and lastly, œdema and dropsy. The blood, with a few scanty fibrinous coagula, is fluid, adhesive, dark-red ; or else, with the exception of very inconsiderable soft coagula, it is thin, watery, of a pale-red, resembling water in which flesh has been steeped. Wherever abundant fibrin-coagula are met with, they present the characters proper to the fibrin-crasis, often in conjunction with tubercle-like concretions, which in their elementary composition fully harmonize with the tubercle of exudation, that is, the tubercle of the textures.

PYÆMIA. PUS-BLOOD.

This crasis again represents a *local pus-production*, and also a *spontaneous primitive pyæmia* of the entire blood-mass.

In pyæmia it is necessary to distinguish two different grades or stages, in order to bring the various facts into mutual concord, and to avoid contradiction in the characteristic given of this blood disease. Those facts are, on the one side, the coagulation and deposition ; on the other side, destruction, of the fibrin. The latter may become developed, as a higher grade,—as a consecutive stage, out of the former. The higher grade may, however, set in at once, as a protopathic crasis, without being preceded by the first or lesser grade.

In the lesser grade the blood-crasis is characterized by quantitative excess in the production of fibrin [hyperinosis], which is at the same time qualitatively impaired. It is marked by a high degree of coagulability and of proneness to separation from the blood-mass,—manifesting itself as croupous, liquefying fibrin. These main features of the crasis illustrate the following appearances.

The fibrin-coagula endogenous to the vascular system, are remarkable for their opacity, for their varying hues of dull white, of yellowish-green, of reddish-gray [from enclosed blood]. They are soft, and yet tough—their contained serum being of a whey-like turbidness. A closer inspection shows them to consist of a glebous basement, with the rudiments of fibrils, and about these a vast quantity of fine point-molecule. There

are, besides these, nuclei, and nucleated cells the nuclei of which appear to resemble in various degrees, up to complete dissilience, pus-nuclei. Sometimes they exhibit little tubercle-like congeries, which consist of collected elements of pus. Having originated during life, they soften, with liquefaction of the said basement, to a fluid which assimilates to pus proportionately to the amount of pus-cells included in the clot.

This crasis, in its most developed grade, possesses the peculiarity of localizing itself in many areæ in rapid succession. Along with highly acute inflammation with purulent effusion upon mucous membranes, upon serous membranes, and in areolar tissue, these areæ appear in every variety of organ and of texture, and are generally distinguished for their small circumference and their sharp definition. They form suddenly in the textures as red obstructions, which almost as speedily deliquesce with sloughing and ulcerous fusion of the involved textures to a yellow or greenish-yellow pus. Wherefore,—as also owing to the lack of evidence of antecedent inflammation,—they have received the name of pus deposits or depôts; of pus metastases. We have already described the attendant anatomical process, and seen that these acts [like others brought about under the fibrin-crases] consist essentially in a spontaneous coagulation of the blood-fibrin in the capillaries, and its immediate liquefaction, with ulcerous corrosion of the bloodvessel membranes and of the contiguous textures; to which process, inflammation with similar products, as the encompassing inflammatory areola, supervenes. Coagula of the same nature form in the great vessels, and, in the shape of purulent vegetations, also in the heart.

The blood appears, along with the endogenous fibrin-coagula described, as a tenacious fluid of a russet hue. It is seen spread out in a thin layer, and mingled with little soft particles, which turn out to be aggregates of pus-nuclei and pus-cells, along with blood-corpuscles, in a transparent clot.

The dead body, owing to the voluminous separation of coagula, presents extensive livid patches verging upon russet coloration; hypostases; textural redness of imbibition; lack of rigor; flabby muscles; friable, doughy, collapsed parenchymata.

The higher grades of the pus-crasis consist in destruction of the fibrin; attenuation and discoloration of the blood; septic decomposition of the circulating fluid, of a nature corresponding to the rapid ulcerous and gangrenous up-breaking of textures in the local processes.

The more intense is the aggravation of the crasis, the less do we encounter the aforesaid depôts. It is only in the transition to the higher grades that we meet with them, obviously breaking up, along with the involved textures, to a dingy brown, coffee-ground, or olive-colored, collapsing, fetid pulp. The same metamorphosis affects both the exudates and their parent strata. In the *highest grade* extensive passive stases affect the decomposed blood, producing necrosis with dark and hemorrhagic imbibition of the textures.

The dead bodies of persons who have died of pyæmia at this stage, manifest, apart from the external and internal local processes—due, it may be, to an earlier phase—long retention of animal warmth, little and evanescent rigor, flabby and pale muscle, more especially discolora-

tion and lacerability of the heart, and rapid decomposition with extensive, brownish death-patches. The parenchymata are lax, easily torn, serum-drenched, pale, or of a spurious redness in various shades, owing to hyperæmia and imbibition of dissolved blood-pigment. The lungs are the especial seat of hypostatic congestion, with a dark coloration verging upon cherry-red or upon brown. The blood in the heart and vascular trunks contains scanty, colloid-like, cruor-holding, red, and sometimes greenish-red, coagula, and is itself of a cherry red, adhesive, or else attenuate, brownish; having stained the bloodvessel membranes and the endocardium with its coloring matter. Pyæmia is not unfrequently *primitive* [protopathic—or deuteropathic, that is arising out of other anomalous crases—for example, the typhous, the exanthematous]. More commonly, however, it is *consecutive* to the reception of pus into the blood, or else to infection, brought about in the various ways, fully detailed at page 274 of the present volume.

This applies to both grades of pyæmia of which it has been stated that the second also occurs independently, determined through infection of the blood by a foul pus decomposed through stagnation. To this category belongs, amongst others, the infection proceeding from the poisoning of wounds with pus out of the dead body. Darcet brought forth the disease by injecting corrupt pus-plasma, a disease into which, as a purulent sepsis or necrosis of the blood, the minor grades of pyæmia with hyperinosis become spontaneously exalted, probably owing to the pus in the blood becoming oxidized in the respiratory process.

The circumstance that pus secreted bodily into the canal of a bloodvessel, commonly produces a more intense infection than pus-plasma probably received into the blood by resorption, might lead one to conclude that it is to the pus-cell that pyæmia is attributable. Nevertheless, apart from the incomparably larger proportion of pus received into the blood in the former case than in the latter, the occurrence of primitive pyæmia precludes our adjudicating in favor of either the pus-cell or pus-plasma as the causal agent. Far more depends, without any doubt, upon the quality of the pus; and it is certain that a large proportion of bland pus taken up into the circulation proves far less mischievous than an incomparably smaller quantity of purulent ichor. That the pus-cell taken up into the blood, or even that other cell formations of larger size—certain cancer-cells, for example—should, by obstructing the capillaries, give rise mechanically to so-called depôts [metastases] will scarcely be credited at the present day.

Pyæmia generally proves fatal, as purulent poisoning: inconsiderable grades of it, however, are susceptible of cure. This occurs, without doubt, partly through a conversion of the pus, analogous to the metamorphosis of fibrin—partly through elimination of the pus in exudatory processes, especially upon extensive mucous membranes, like that of the intestinal tract. The elimination of pus through processes of secretion, for instance, its passage through the kidneys, with the concurrent disappearance of pus-deposits, is a phenomenon much talked of. The pus-cell both in the urine and in exudates is incapable of becoming reabsorbed, and equally so of passing out of the bloodvessels, either into the uriniferous tubules or at any other part. It follows, therefore,

that pus-cells, either in the urine or in an exudate, must be a new creation out of effused plasma.

2. VENOSITY, ALBUMINOSIS.—HYPINOSIS (SIMON).

This constitution of the blood is characterized by *deficiency in fibrin* but *preponderance of albumen, and generally speaking, also of blood-globules.* The blood is upon the whole thickish, tenacious, dark-red, and contains, if any, only a few soft, gluey or jelly-like coagula, in which there is much cruor pent up.

It *has a very extensive domain*, comprising a vast number of special crases, which reveal their kindred nature by the general characters of the blood just defined, by the metamorphoses which many of them undergo in common, and by the general sameness of their products; whilst again they differ in some particular attribute of the latter, and by specific relations to particular textures and organs [localization].

Their range comprehends the most important and most perilous, acute and chronic diseases; plethora [general hyperæmia], venosity of the lungs, and heart diseases, the acute exanthemata, especially scarlatina and measles, the so-called substantive fevers, *chronic* rheumatism and gout, rickets, typhus, Asiatic cholera, so-called acute tuberculosis, Bright's disease, and lardaceous degeneration of the liver, spleen, or kidneys, mollities ossium, cancer, the crases of acute convulsions, of tetanus, of hydrophobia, diseases of the nerve-centres, chronic mental alienation, hypochondriasis, chronic metal-poisoning, especially with lead, narcotism, finally the crases accompanying atrophy after acute, exhausting diseases, the so-called suffocative death-seizures generally.

To discover the nature of the special crasis in so heterogeneous states, is reserved for the future, and rather for chemistry than for anatomy.

Many acute crases issue in *septic destruction of the albumen* and putrid decomposition of the entire blood-mass. This consummation is especially frequent in the exanthematous and the typhous crases, and in acute convulsions. The acute crases are moreover liable to frequent transformations, especially to the croupous crasis and to pyæmia. There occurs frequently an acidifying of the blood, which localizes itself in miliary eruption and in acute softening of the stomach.

The crasis is sometimes *protopathic*,—habitual, persistent, ingrained in the individual, or acute and evanescent. At other times it is *deutero-pathic*, or the sequel to exhausting and especially to defibrinating disease. It is, moreover, a primitive blood-disease, called forth by poisons, by miasma, by contagion, or else it is consecutive to disease of solid parts [for example, organic heart disease], and determined by neurosis.

The products placed under its control [exudates and new growths] are distinguished by an excess of albumen, by very subordinate coagu-lability, by lack of disposition to become organized, by persistence at embryonic grades of structural development.

A not unfrequent sequel to extensive exudation is hydræmia, or, it may be, tarlike inspissation of the blood with anæmia. The former

becomes developed without any notable serous effusion, the water being otherwise disposed of in the morbid process. The anæmia is commonly due to a shattered condition of the nervous system.

Several of these acute crases have a decided relation to the mucous membranes, and especially to their follicular apparatus, to the lymphatic glandular system, to the common integuments, to the spleen. The dead body presents, especially in the acute crases, dark coloration of the common integument; rapidly developed, extensive, and very dark death-patches; early decomposition; a very marked but for the most part evanescent rigor, and a lax, doughy condition of the parenchymata. Hyperæmiæ and stases arising in the different organs, not unfrequently become exalted into hemorrhage. In the tarlike inspissation of the blood, the corpse is in a high degree emaciated or rather shrunken, dry,—the common integument, of a lead-color, or livid.

Let us endeavor to submit the more important of these crases either singly, or where the distinctions are not very marked, more collectively, to an anatomical muster.

(a.) PLETHORA.

It is characterized by *excess of blood, by a preponderance of the blood-globules over the fibrin, by a deep red, tenacious blood.* It involves the direct manifestations of venosity in the inverse ratio of the amount of blood which the organism is capable of arterializing. It occurs under two opposite and contrasting relations. First, in conjunction with florid nutrition of the textures, fulness of muscle, and especially ample areolar tissue and fat formation. Secondly, as a very marked phenomenon in union with general emaciation,—wasting of the solids [so-called nervous tabes]. Under the latter circumstances, it is observable both in very delicate children, during the first months of their life, and in insane adults [in hypochondriasis, melancholia, &c.]

In the dead body, the general overloading of the vascular system, and occasionally surpassing hyperæmia of various organs, especially of the lungs or of the brain, or of the liver and entire portal system, are manifest. According to the degree of intensity of the crasis, all of the soft parts are more or less deeply colored. In the emaciated, the common integuments exhibit vast patches of a purple, or of a bluish leaden hue.

Plethora predisposes to congestion, to hemorrhage, to blennorhoid, albuminous, and serous exudations of greater or less moment in proportion to their amount and to the importance of the organs concerned. In corpulent, square-built [apoplectic] individuals hyperæmiæ of the lungs are frequent. In these the plethora often of itself, but more commonly through acute serous effusion into the bronchia and lung-cells, proves speedily fatal. Moreover, the plethora occasions dilatation of the heart, with subsequent, progressive augmentation of its substance [hypertrophy].

(b.) THE TYPHUS-CRASIS.

It compasses the entire nature of typhous disease, and is at the root of all its phenomena, whether of substantive change or of functional disturbance.

The typhus-crasis is marked by the destruction—the diminution—of the fibrin, and the comparative preponderance of the blood-globules. The typhus blood is in various degrees fluid, and of a deep purple color. It forms, if any, but scanty, loose, soft, and humid, deliquescent coagula, reddened by the imbibition of pigment-holding plasma.

The corpses of typhous individuals are remarkable for the deep, dingy, bluish-gray coloration of the common integument, for the deep purple of the death-spots, for the dark russet hue and the rigidity of the muscles, and for the dryness of the areolar tissue. The serous membranes, and especially the peritoneum are of a dull gray, lack-lustre, and occasionally suffused with a tenacious humor. All the textures in contact with blood appear discolored from imbibed hæmatin, of a peculiar shade, verging from violet color upon brown.

In the next place, the multifarious local hyperæmiæ have to be noticed. They are due to the paralyzing influence of the blood upon determinate ranges of the nervous system, either at the periphery or at the centres. Foremost amongst them are local hyperæmiæ of the mucous membranes, of the lungs, of the brain, and its membranes, of the spinal cord, of the common integuments. They often display the attributes of so-called hypostasis. Upon mucous membranes they frequently degenerate into hemorrhages, which occur also, although far more rarely, in parenchymata, for example, in the brain.

The typhus-crasis manifests a very marked relation to mucous membranes, especially to the lymphatic glands and to the spleen. In middle Europe it is the mucous membrane of the intestine and especially of the ileum, rarely the bronchial mucous membrane with the lungs and the bronchial glands; in the North, it is rather the last mentioned, namely, the respiratory tract; in the South [in pest-typhus], it is the peripheral lymphatic gland system, in which the crasis becomes localized. In the form of a typhous inflammation it determines, in the follicular apparatus of the ileum and in the mesenteric glands, a peculiar marrow-like product, which, in intense cases, closely resembles medullary carcinoma.

The very variable consistency of the typhus-substance points to variations in the typhus-crasis itself; to different degrees of plasticity in the typhous blood-plasma.

Pus-formation, we have to observe, *is alien to the genuine typhous process* whether general or local. Wherever it does occur, it is founded in a degeneration or change in the typhus-crasis, of which we have to say a few words. No other crasis offers such manifold interest in reference to *degeneration* or *conversion*. Not alone are there several conversions of the kind, but they are remarkable for an impress the exact reverse of the original typhus. The recognition of these phases and their interpretation as degenerations or transformations, are not only of the greatest scientific interest, but also of the most obvious practical utility. These changes resolve themselves into the following. They are more or less demonstrable in the sanguineous fluid, as also more or less proclaimed in corresponding local processes.

1. *Conversion to the croupous crasis.*
2. *Conversion to pyæmia.*
3. *Degeneration to acute softening* [acidification of the blood].
4. *Degeneration to gangrene* [Sepsis,—necrosis of the blood].

1. *Conversion to the croupous crasis.* A fibrin of a constitution characteristic of the croupous crases forms in the blood. Perishable coagula [vegetations, plugging clots, so-called capillary phlebitoids] originate in the heart, in the greater vessels, in the capillaries ; but, above all, exudative processes, upon mucous membranes. Those croupous inflammations of the mucous membrane lining the tracheal canal, the œsophagus, the stomach and intestines, the female sexual organs, as also croupous pneumonia, all belong to this type. Again, similar processes upon serous membranes, the yellow, fibrinous, crumbling products by which the typhous infiltrations of the mesenteric and Peyerian glands are modified.

The exudates are wont to exert a solvent influence upon their parent strata, deep corrosions of the mucous membranes beneath the exudates, more especially at the glottis and epiglottis, being not at all uncommon.

This conversion takes place at various periods of the typhous process—even at a very early stage. It is deserving of notice that a vast number of cases in point happen at the commencement of epidemics of cholera, a disease in whose typhoid [so-called reaction-] stage croupous inflammations are so frequent. [See "Exanthematous Crasis."]

The conversion of the typhus-crasis *to the tuberculous*—the *tuberculo-croupous—crasis* belongs to the same class. Its localization generally attaches to the lungs in the shape of lobular—not unfrequently of a comprehensive lobar pneumonia ;—pneumonic tubercle-infiltration.

2. *A second conversion* of the typhus-crasis, kindred with the preceding one, is that to *pyæmia* and local *pus production*. It occurs, for the most part, at a later period than the one just referred to, often complicating the last stage of the local typhus-process upon the mucous membrane of the ileum, and protracting itself into a *sequela* to the typhus. Examples hereof are the pus-deposits in the typhous patches upon the mucous membrane of the ileum, and in the infiltrated mesenteric glands ; the pus-producing areolar-tissue inflammations ; the purulent exudates upon serous tunics ; the circumscript, suppurating coagulations in the capillary system of the lungs, the spleen, the kidneys ; the boil-like obstructions of the capillaries in the substance of the mucous membranes and of the outer skin.

3. *Degeneration* to the *acute softening process*, that is, to a crasis in which the latter is founded, and which is localized in softening of the stomach, &c. We believe this process to be a peculiar one, quite distinct from putrid decomposition and its characteristic, gangrenous sloughings. That which concerns us here occurs as black, or Indian ink-colored softenings or meltings of the textures in an acid fluid, especially in the cœcal sac of the stomach, on the left side of the œsophagus, in the lungs, upon the mucous membrane of the cœcum, and in the urinary bladder.

We believe *this process to be derived from the blood in the capillaries of the parts referred to, and to be due to an acidification of the blood-mass,—to the presence of a free acid in the blood :*

(*a.*) It is developed out of a hyperæmia and stasis in the implicated organs, and in point of fact, out of the blood engaged in the stasis, which experiences the first effect of the liberated acid upon itself, in the shape of inspissation and coagulation to a black, pitch-like, friable mass, destructive of the walls of the vessels and of other contiguous textures.

(*b*.) The reaction of the structures softened is invariably acid.

(*c*.) Our view seems to derive support from the determination of the blood under these circumstances to the cœcal sac of the stomach, which, with the spleen, appears to us to perform the office of a de-acidifying apparatus to the blood-mass, for the immediate secretion of the gastric juice and in behalf of the hepatic function.

(*d*.) A very frequent appearance associated with the impending softening, is that of a miliary eruption with acid reaction of the contents of the vesicles.

4. *Degeneration to sepsis ; putrid-crasis.* Primary gangrene of the solids. It occurs either very early, or only as a sequel to typhus.

The blood and the dead body exhibit the peculiar changes to be described in another place.

Where the putrid character is early developed, the faint impression in its localization, especially with respect to plasticity of its products, is remarkable. The Peyerian gland-groups are turgid with sero-albuminous infiltration, lax, and, together with the mucous membrane of the ileum, generally ecchymosed.

As the local manifestation of this degenerate state, sloughing takes place in parts exposed to hypostasis and pressure, for instance, in the sacral region, at the trochanters, &c. This is not all, however : hyperæmiæ and stases become developed even in parts beyond the range of hypostasis, leading incontinently to mortification of the textures,—for example noma of the cheeks, sphacelus of the external sexual organs in the female.

All these degenerations may become localized in the typhus-ulcers, leading, as will be seen, in the account given [see vol. ii.] of the local typhus-process in the intestinal membrane, to a destruction overstepping the limits of the textures, and frequently to perforation of the intestine.

Other sequelæ of the typhus-crasis are protracted albuminuria [Bright's disease], anæmia [with wasting], hydræmia [Œdema, Dropsy].

(*c*.) THE EXANTHEMATOUS CRASIS.

Its domain, viewed from the anatomical side is a very extensive one. However, we might wish to limit this crasis to scarlatina and measles, a number of acute blood diseases naturally cluster around them, become localized upon the greater mucous membranes, and not unfrequently, especially in epidemics, produce exanthemata presenting more or less analogy with genuine measles, or pure scarlatina. Amongst them are some which determine a more or less plastic, albumen-loaded, coagulating or colloid-like, thinly purulent, almost serous product upon mucous membranes ; exudatory processes which bring the muco-membranous texture into a state of dissolution; Asiatic cholera ; numerous puerperal affections; acute diarrhœa, especially in children; nay, even exanthematous typhus ; in fine, many substantive [exanthematous] fevers without exanthema. At the uttermost limits of this domain are placed blood diseases, associated with tonic spasm and convulsions,—with affections

of the nervous centres. Last of all, acute tuberculosis. In the present section, however, we shall only speak of the exanthematous crasis and of those nearest allied to it, leaving the remainder to be discussed in separate chapters. The crasis in question is the most distinctly marked in scarlatina; and it is here that we have the best opportunities for studying it in the dead body. In degree, the crasis of measles is perhaps nearly the same, as are also the blood diseases already stated to follow next in the scale.

Upon the whole, *the exanthematous crasis has the greatest affinity to the typhous. Only the blood is still more fluid, whilst the violet tint present in intense typhus is wanting. The blood verges more upon purple,—upon cherry-red.*

The dead body manifests a certain degree of turgor. There is a lack of that tenseness of the muscles, and of the common integument, as also of the dingy-gray coloration of the latter. The skin is indeed rather white, although with extensive, very saturated death-patches. The serous membranes very often exhibit a viscid, ropy, colorless covering. Local hyperæmiæ, partly of hypostatic nature, and imbibition of the textures with blood-pigment, are observable.

Along with these differential points, and apart from the resemblance in the anatomical characters of the blood, *special analogies come forth between the typhous and the exanthematous crasis.* Such as:

(*a.*) *The relation of the exanthematous crasis to the mucous membranes, and also to the lymphatic glands.* It is expressed in the well-known catarrhal, erythematous, and other affections of the respiratory and gastro-enteric tracts of mucous membrane;—in the well-known enlargements of peripherous lymphatic glands accompanying the course of the exanthemata. What is, however, particularly characteristic, is the development of the follicle apparatus of the ileum and of the mesenteric glands in scarlatina, and in the entire series of analogous blood diseases.

(*b.*) The identical conversions of the crasis observed in typhus. The most frequent are the conversion to the croupous crasis, including the tuberculo-croupous, with corresponding local products, and the often early degeneration to putrid decomposition.

Among the sequelæ there is one proper to typhus, and also a very frequent consequence of scarlatina,—namely, protracted albuminuria.

Another consequence of the exanthematous processes, is intense inspissation of the blood, with marked hyperæmiæ and stases.

As far as its crasial source is concerned, variola does not seem to constitute an exception. It is, however, essentially obnoxious to a speedy transition into the croupous-crasis and into pyæmia, the latter often outrunning its normal term. In common with the exanthematous crasis generally, it is liable to degenerate early into putrid decomposition, which, anticipating the croupous and the pyæmic phases, precludes the formation of any products due to these modifications of the exanthema, and causes the pocks to degenerate into the so-called putrid.

Amongst the diseases following closely in the wake of the more prominent exanthemata, the under-mentioned are peculiarly deserving of notice, namely :

A large proportion of puerperal fevers—especially when bearing an

epidemic impress. The characters presented by the dead body, and the anatomical relations of the blood, answer to the exanthematous crasis; to which may be added, the presence of the exudatory processes above specified upon the uterine mucous membrane, the character of exudates detected upon the serous membranes, especially the peritoneum, and finally, the frequency of concurrent exanthema, in the shape of erythema and of scarlatina. These puerperal processes are marked by their tendency either to become converted into the croupous crasis, or else to degenerate into putrid decomposition. Nor is acute softening of the stomach a rare coincident phenomenon.

A crasis appertaining to this class often becomes localized, as one of the *exudatory processes adverted to, upon the intestinal mucous membrane,* as in many instances of *acute diarrhœa* and of *dysentery.* These often prove fatal through paralysis of the intestine—through exhaustion. A very remarkable and momentous phenomena, however, is a resulting thickening of the blood to a dark red liquid, of a tarry appearance, and of the consistency of treacle. It proves fatal under the symptoms of anæmia in vital organs [lungs, brain], of rigor [tonic spasm] of muscular organs, or else through local hyperæmiæ [for example of the brain]. Eruptive phenomena in the progress of the dysenteries are not at all uncommon.

Conversions of the crasis to the croupous and to protracted pyæmia are frequent.

Next in the series is the *cholera-process.* It is a more or less rapidly developed hypinosis, with the characters of the exanthematous. Its localization extends over the entire intestinal tract as an exhausting exudatory process, multifarious in its products, and either proving rapidly fatal under acute inspissation of the blood and the aforesaid contingent phenomena, or else passing over into a so-called stage of reaction.

In the former case, the dead subject presents a dingy, blue-gray coloration of the common integument—a puckered state of the latter and of the areolar tissue, with herb-like dryness and rigidity, and with dark coloration of the muscles. The blood, if we except certain stases in different ranges of the vascular system, especially in the bloodvessels of the membranes of the brain, is found acccumulated in the vascular trunks, and in the heart, as a dark tar-like mass, without fibrin-coagulum. The lungs are for the most part dry, inflated, of a deep red; the serous membranes—more especially the peritoneum—moistened with an abundant viscid coating. The intestinal canal is the seat of an extensive, and equally rapid and intense process of exudation, and presents the general lineaments of paralysis. Surcharged as it is with fluid, it is nevertheless collapsed, soft, and flabby-membraned, pallid, rarely presenting any intussusception. It contains, in varied measure, a serous fluid, rendered turbid, whey- or rice-water-like, whitish or yellowish-white, by the débris of epithelium and minute particles of protein substances, or else slightly reddened by the intermingling of blood. The mucous membrane is denuded of its epithelium, and bare; or the coagulable portion of the exudation may adhere to it in the shape of a loose bran-like covering, or of membrane-like formations. Its texture is bloated, and is for the most part readily scraped off, as a reddish-white

pulp; its follicles, especially at the ileum, distended by exudate to the bigness of millet or hemp-seeds. The spleen is shrivelled, the urinary bladder empty. In the ganglia of the sympathetic we detect little hemorrhagic spots, as big as a poppy or millet-seed.

In the so-called stage of reaction, the crasis reverts to the normal, or else the hypinosis changes under expansion of the blood into a typhoid disease. The latter is remarkable for a secondary localization upon the mucous membrane of the intestines, in the shape of repeated processes of exudation, and also for exanthematous processes simulating measles, scarlatina, pemphigus or erysipelas, upon the common integument. During the thus protracted course of this hypinosis, it is very usual for a fibrino croupous crasis to develope itself, and for the mucous membrane of the intestine, of the stomach, of the œsophagus, of the trachea, to display croupous exudation,—the lungs, croupous pneumonia.

This hypinosis is moreover convertible to pyæmia, to acute softening, and also liable to putrid decomposition.

(d.) HYPINOSIS IN DISEASES OF THE NERVES.

The similarity of the crasis in this class of diseases with the typhous and exanthematous crases is very striking. It even partakes, in common with these, of a proneness to localize itself upon the intestinal mucous membrane in the follicular apparatus of the ileum,—in a turgescence [product-formation] of the Peyerian and solitary gland capsules.

To this category belong diseases with obvious anatomical disturbance in the nervous centres, and again diseases in which such disturbance is either wanting or subordinate and consecutive. Such are meningitis, acute hydrocephalus, apoplexy, and the like; and again, acute tonic spasm and convulsions, tetanus, trismus, puerperal convulsions, protracted epileptic convulsions, &c.; lastly, hydrophobia.

In the latter diseases, more especially, in which up to the present day, no anatomical disturbance is demonstrable, the question arises as to whether the nervous system be substantially impaired at all,—whether the anomaly in the crasis be not the primary cause of the nervous phenomena.

It appears to us that, although the most accurate examination may be inadequate to prove any palpable anatomical disturbance, a primitive affection of the nervous system must nevertheless exist, and be that which determines the [secondary] anomaly of the crasis.

This crasis not unfrequently degenerates into putrid decomposition. It often becomes converted into pyæmia, and not unfrequently issues in acute softening.

In fine, those rapidly destructive liquefactions of the blood may be here classified, which, under the name of asphyxia furnish forth the majority of *instances of sudden death*, commonly through hyperæmia of the lungs with acutely developed pulmonary œdema.

(e.) THE DRUNKARD'S DYSCRASIS.

That dyscrasial condition induced by the abuse of alcoholic drinks, and especially of gin, occurs under two forms, differing in the course

which they run—in other words, there is *a chronic* and also *an acute drunkard's dyscrasis.*

The first, namely, the *chronic crasis,* manifests itself as plethora, with a remarkably dark coloration, a thickness and a simultaneous fattiness of the blood. This occasions, and at the same time accounts for, the condition in which we find the solids.

The pigmented appearance of the skin, the excessive, and, at the same time, anomalous fat-formation, and blennorrhœæ, are all characteristic of the crasis. The corpses of inveterate dram-drinkers present very marked appearances. The skin is tinged of a dingy brown, and this is coupled with the fact, that not unfrequently parts naturally rich in pigment—the scrotum, for example, become deprived of it. At the same time the skin is of a soft, unctuous feel, like that of the negro, and its epidermis layer is thin. Subjacent to the skin, and also in the mesentery and the omenta is deposited fat, in an excess if not absolute, at least relative to the state in which we find the muscles, and possessing a peculiar character not unlike that of mutton suet. Together with this, the muscle-flesh appears to have lost in volume, and to have become pallid. The fat-formation steals into the muscles in the shape of fatty conversion. The liver has undergone fatty degeneration. Even in the bones the fat formation has gained ground at the expense of the bony texture.

All the mucous membranes, but especially the bronchial and intestinal, are affected with chronic thickening and with blennorrhœa, the chronic gastric catarrh [gastric irritation] being particularly marked. A similar state of hypertrophy presents itself in the habitually congested cerebral membranes, in the form of dulness, thickening, chronic œdema.

The brain is affected with an atrophy like that met with in the aged, with or without considerable dilatation of, and serous effusion into, the ventricles.

The blood appears dark-colored, grumous, defibrinated, viscid-unctuous to the touch, often intermingled with fat in large quantity, as fat-drops. In rare instances the disease occasions a chyle-like opacity of the plasma —milky blood.

The chronic drunkard's crasis often undergoes conversion to *fibrin-crases* of various kinds. Amongst these are inflammations with fibrin-products, even tubercle. Pneumonia is a very frequent and very fatal disease with drunkards—pneumonia running an acute course, and possessing an eminently croupous character. Chronic hepatitis, determining organizable products, and leading eventually to liver-cirrhosis [granulation], is a very common termination.

In drunkards tuberculosis runs an eminently chronic course. The deposition of tubercle is for the most part inconsiderable ; the granulations are generally of a dingy, or a greenish-gray, and do not soften as such. The yellow tubercle and resulting phthisis are more rare.

The natural issue of the drunkard's crasis is in eventual hydræmia— in dropsy—which assumes a local form, especially that of ascites, the more speedily, the earlier heart-disease or liver-cirrhosis becomes established.

The acute crasis has a marked resemblance with the exanthematous,

and with the crasis in nervous affections. The liquefaction with discoloration of the blood and, as a consequence thereof, the tendency to transudation of blood-serum, are for the most part more developed. The fat is wont to separate from the blood in the form of largish drops.

The corpses present extensive, very saturated death-patches; evanescent rigor of the dark red muscles; congestion of the cerebral membranes, and still more of the lungs, especially as hypostasis; scattered patches of stasis in the intestinal mucous membrane, &c. The parenchymata are lax; those affected with hyperæmia, imbibed with a colored blood-serum. In the cavities of the serous membranes—more especially of the pleura—are dingy-red, serous [spurious hemorrhagic] effusions. The corpses emit a peculiar sweetish smell, and pass rapidly into decomposition under gas development.

The inflammatory stases developed during the progress of this crasis are, for the most part, hypostatic pneumoniæ. They determine a product dark-colored from adherent hæmatin, lax, soft, incompetent to hepatize the lung-texture.

This crasis never becomes developed in aged persons broken down by repeated attacks of delirium tremens, but invariably in drunkards in the early years of manhood, who are endowed with a powerful muscular system. It runs a very rapid course, leading, in a very few days to decomposition.

We are aware of no instance of this crasis passing into the fibrinous crasis, or into pyæmia. On the other hand, it often becomes exalted into decomposition, and not unfrequently issues in softening of the stomach.

It is not improbable that this crasis is, in the majority of cases, due to injury sustained by the brain during a violent or protracted fit of intoxication, and that it ought rather to have found a place in the preceding chapter. It is not in aged drunkards, with an atrophied brain, that it occurs, but in younger individuals with a brain of normal development, keenly sentient of congestion, and of an alcoholized condition of the blood.

(f.) THE CRASIS OF ACUTE TUBERCULOSIS.

This crasis has the greatest resemblance with the exanthematous, and the disease assimilates so closely in its manifestations during life to typhus [intestinal typhus], as only to be distinguishable from the latter by the absence of abdominal symptoms, the more marked phenomena pointing to exudation in the membranes of the brain and upon the linings of its ventricles.

The product of this crasis is a tubercle presenting many peculiarities. It is a scattered corpuscle, mostly smaller than a millet-grain, and no bigger than a poppy-seed, or even a pin's point. It is now of glassy transparency, vesicle-like; now grayish, semi-opaque, soft, gluey;— then, again, verging upon whitish, or yellowish-white, and opaque. With it there always exudes a grayish, more or less albumen-sated, semi-gelatinous serosity, infiltrating—drenching—the involved textures. The deposition of these products always affects an organ in wide extension.

Generally speaking, indeed, several organs are implicated at once, more especially the lungs, the cerebral pia mater, the spleen, the liver, and the serous membranes. The tubercle is always deposited in great numbers, and is equally disseminated throughout an organ, as the examination of an involved lung or spleen shows at a glance.

The tubercle is sometimes primitive, but more commonly successive to a pre-existent fibrin-tuberculosis. Looking at the physical properties of this tubercle, apart from any chemical analysis, and connecting it with the crasis of which it is the product, we are constrained to set it down as an albuminous formation.

With reference to the crasis itself, which, as we have said, is marked by a hypinosis closely resembling the exanthematous, it may be asked [and the question is replete with interest]: is acute tuberculosis primitive, or is it but a consecutive state of defibrination of the blood, brought about through the antecedent out-throwing of a large proportion of fibrin, in the shape of tubercle?

Or is it one of the so common tuberculoses connected with hydrocephalic effusion of the internal membranes of the brain, that is, consecutive hypinosis, determined by disease of the brain?

Seeing that acute tuberculosis occurs under conditions which preclude either of the contingencies here referred to, not a doubt can remain as to its protopathic character.

A further question is: how, in this hypinotic crasis, is the tubercle brought about?

[For a reply to this question the reader is, to avoid entire repetition, referred to the section on "Albuminous Tubercle," in the present volume, p. 246.]

Acute tuberculosis probably always proves fatal.

Occasionally, owing to very extensive deposition, the crasis approximates to hydræmia. It does not pass into putrid decomposition, nor is it converted into pyæmia. On the other hand, acute softening of the stomach is a frequent follower in its train.

The corpses present, generally speaking, the same phenomena as in typhus; namely, pale skin, extensive, deep-colored patches of lividity, tense, dark-colored muscles, hypostatic hyperæmiæ. The parenchymata, especially those which have been the seat of tubercle-deposition, are turgescent, and drenched with sero-albuminous fluid.

(g.) CANCER-DYSCRASIS.

A crasis the existence of which is shown from the exclusive relation stated, in the general section on Tubercle, to exist between cancer and tuberculosis.

To demonstrate a cancer-dyscrasis from anatomico-clinical data is one of the most difficult tasks. The basis of such a demonstration is the immediate character of the blood, the peculiarity of the cancer-formation, and of other exudates brought about in various ways under the crasial influence; and, lastly, their relation to new growths proper to other known crases.

The blood itself affords evidence of a *hypinosis*. This is, however,

not of itself alone cognizable as a specific cancer hypinosis. To prove this the presence of cancer-formations is indispensable, and even these must needs give evidence of their *general* import, either by redundant growth or by multiplication; in short, they must in some way betoken a direct relation with a dyscrasial state of the blood.

Further evidence respecting albuminosis is afforded by the presence of albuminuria, of lardaceous infiltration of the liver, spleen, kidneys; more particularly, however, by inflammatory products, as albuminous, white, emulsion-like, in part slowly solidifying, ulcerating, or cancer-forming exudates; lastly, by the exclusion of concurrent fibrinous products, and especially of fibrinous tubercle.

The abundant fat-formation not unfrequently co-existent with carcinoma may be cited in proof of the participation of fat in the cancerous albumen-crasis. This is exemplified in osteo-porosis from the excessive formation of bone-medulla; in the fatty contents of the cancers, and of albumino-cancerous exudates; in the deposition of fat as cholesteatoma, as gall-stone, &c.

The cancerous hypinosis manifests its impress in various grades. It is intense in cases of voluminous, exuberant cancers; in very widely-spread cancer-production, whether spontaneous, or called forth by the extirpation of bulky carcinomata; but most especially in cancer of acute growth, and of the medullary character. The blood often contains, in nucleus- and cell-formations, the elements of cancer. In chronic vegetation, and especially in pure fibrin-cancer, the hypinosis is often less marked; whilst, in cancer of local import, it may be altogether wanting.

The cancer-crasis is either primitive or consecutive, that is, developed out of a *hitherto local* cancer. It is either acute or [more often] chronic.

The acute crasis is in rare instances protopathic; more commonly, however, it is developed out of the chronic, especially after the extirpation of extensive cancers. It localizes now in the more vigorous growth of a cancer already in existence, now in the simultaneous or in the successive, hasty production of new cancers [of the medullary form] in the most various organs and textures, conducing thus to rapid wasting of the blood, and proving fatal within a term not exceeding that of the most acute crases known.

In its chronic development it terminates in marasm of the blood, in hydræmia, in anæmia, the more rapidly, in proportion as the seat of the cancer [in the stomach, for example] is calculated to interfere with the work of nutrition, or in proportion to the loss of blood by hemorrhage.

Under such conditions the cancer-crasis may wear itself out, and the cancer-tumors participate in the waste and decline of the entire organism. This explains the circumstance that in a venosity verging upon hydræmia and depending upon central organic impediments to the circulation, cancers hardly ever occur.

The cancerous hypinosis is, as we have already pointed out, absent in local carcinoma. The concurrent crasis may be the normal, or some anomalous one not of a cancerous nature.

The *fibrin-crases*, however, accompanying, or at least coincident with

cancer, are of great interest, not alone, as running counter to our theory of the nature of cancer, but more particularly because, if correctly seen and comprehended, they afford the best means of demonstrating the specific character of the cancer-crasis.

1. In the first place, it is conceivable that fibrin-crases may become developed along with local cancer. They are, no doubt, sometimes primitive, localizing themselves in the local cancers, as inflammations; sometimes consecutive, that is, brought about in the cancer itself by mechanical or medicamental influences. The fibrin-crasis concurrent with local cancer may even be of a tuberculous character, and lead to tuberculous deposition.

2. A fibrin-crasis may, however, become developed even conjointly with cancer of general import, that is, out of cancerous hypinosis or albuminosis. The cancer-crasis is co-ordinate with other, similar [hypinotic] crases, out of which we have seen that fibrin-crases, more especially those of a croupous character, may emerge. They may arise either directly out of the hypinosis, as a conversion of the latter, or else through the instrumentality of an inflammation with cancero-dyscrasial blood, in which a development of fibrin takes place.

The cancerous fibrinosis, in whichever way brought about, localizes itself in inflammations of the serous tunics, in carcinomato-fibrinous hepatizations of the lung, as also in spontaneous coagulations within the vascular system, including the capillaries [cancero-capillary phlebitis]. Both these and the exudates are distinguished for their opacity, their whiteness [changed by contained blood-globules to grayish-red or red], their soft, lax consistency, their albuminous contents, their medullary characters. They are sometimes fundamental to cancer-formation,—the most acute and most extensive cancer-formation,—both intravascular and extravascular. At other times they liquefy to a white, cream-like, lardo-glutinous ichor. They contain the rudimental elements of cancer in redundant quantity.

In the description just given, a *peculiar constitution of the fibrin* under the conditions both of its organizability and of its liquefying tendency is undeniable. It is essentially proper to cancer, and affords incontestable proof of the specific constitution of the albumen in cancerous hypinosis. Where a fibrin crasis developes itself, whether in the totality of the blood or in a local process [inflammation], this *peculiarity* of constitution is, without doubt, transferred from the albumen to the fibrin. A proof, this, how intimately it clings to both substances; a proof of the existence of a cancerous fibrin-crasis; and at the same time an indication of the sense in which the balance between cancer-crasis and fibrin-crasis is to be understood.

This *cancerous fibrinosis, in fine, is the parent of a peculiar tubercle,* of cancero-fibrinous character, which corresponds well with cancer-crasis, and more particularly with such of its highest grades as have attained the point of fibrinosis; a tubercle, moreover, which, as we have seen at page 237, answers in all respects to cancerous fibrin.

3. HYDRÆMIA; ANÆMIA.

(a.) *The Serous Crasis; Hydræmia.*

Fibrin, albumen, blood-globules, are here all diminished in quantity; *the amount of water increased.* The blood is attenuate, watery, pale in various degrees to the point of water in which flesh has been steeped, wanting in tenacity. It contains very inconsiderable, loose, soft, curd-like coagula holding much serum, which, by pressure is reducible to a few drops.

The water transudes through the parietes of the vessels in dependent parts, or in such as, owing to mechanical influences, are particularly obnoxious to hyperæmia, drenches the textures in the form of œdema, especially the areolar tissue, even to the medullary system of the bones, and forms, in serous cavities, dropsical effusion. It may transude pure, or may contain a certain proportion of albumen and even of fibrin, which latter [as so called spurious fibrin] determines in the textures a soft curd-like coagulation of the dropsical fluid; and in the cavities separates in the shape of soft curd-like flakes. Inflammatory products are marked by the large amount of their serous contents, and by their poverty in plastic materials.

The dropsical crasis occasions defective nutrition, with pallor of the textures, relaxation of the contractile fibre; in the dead body, the development of pale death-patches.

It becomes mortal through insufficiency of nutritive matter in the blood; but for the most part proves fatal at an earlier stage through local œdema of the textures, and dropsy of the great serous cavities.

Not every dropsy is, however, the result of hydræmia. We allude to those local and general dropsies brought about by mechanical impediments to the circulation in the veins, in the heart and great vascular trunks, and in the lungs.

The serous crasis is sometimes idiopathic, produced by climatic relations, by peculiar alimentation, by anomalies in the chylopoietic system, by repeated, exhausting hemorrhages, &c. Nay, it may be even congenital and constitutional. The condition of the blood in hæmorrhophilis, seems to be essentially that of hydræmia. In most instances, however, it is secondary, developed as a sequela to some other crasis, for example, as a consequence of the habitual outpouring of albumen, the separation of fibrin in large aneurisms, the deposition of fibrin and albumen in inflammation-products, in tubercle, in cancer, in albuminuria. Or else it ensues upon a specific, chronic or acute blood-consuming dyscrasis, upon metallic poisoning, typhus, and the like.

(b.) *Anæmia.*

Deficiency of blood, in various degrees, by no means offers any distinctive crasial characters, if we except the hydræmia—the excess of water—into which every persistent anæmia eventually resolves itself. The anæmia or oligæmia, is brought about in various ways; for, what-

ever be the crasial constitution of the blood, it is liable to an accidental reduction of its mass.

It is, most frequently of all, a consequence of loss of blood through the various kinds of hemorrhage ; next to that, of insufficient alimentation, of excessive bodily and mental labor, and of the continuous loss of fluids ; of the inordinate production and increase of new growths, even of redundant fat formation, especially in children ; of disease of the nerve-centres, especially of the brain, such as hypertrophy, heterologous growths, concussion. Or it is the sequel and issue of intense typhus-crasis ; of chronic metallic poisoning, &c. It accompanies all general atrophy, both in old age and in earlier life.

Moreover, oligæmia is not unfrequently a congenital, constitutional state, and affects by preference the female sex. It involves a corresponding defective development of the calibre of the arteries, with smallness of the heart, and with a generally stunted growth of the animal frame. The female sexual organs seem more especially crippled in their development. It was stated that blood of every admixture may suffer an accidental reduction of its mass through hemorrhage, without becoming alienated from its original crasis. In like manner, anæmia is probably never purely such ; that is to say, never brought about by the equable reduction of each of its constituent parts, but at the same time invariably a dyscrasial condition. How inextinguishably the dyscrasis clings to croupous blood, even after the most copious bloodlettings ; how, in the highest grades of blood-deficiency, the tuberculous constitution attaches to the smallest remnant of fibrin, we have already seen.

The most striking picture of anæmia is furnished in the dead bodies of persons who have died of hemorrhage. Collapse and pallor are the outward signs reflected from within. The deathmarks, if there be any, are very pale. There is considerable rigidity of muscle, firmly contracted heart, presenting the aspect of concentrical hypertrophy, bloodlessness, both of the endocardial cavities and of the vascular trunks, especially the arteries. In corpulent individuals with a white skin, the common integument is of a waxy paleness. In profound dyscrasial anæmiæ [the consequence of typhous or metallic poison] the dead subject retains, together with the pallor, the characteristic cachectic hue.

4. DECOMPOSITION.   PUTRID, SEPTIC CRASIS.   SEPSIS OF THE BLOOD.

We have repeatedly had to refer to a *decomposition*, a *putrid decomposition* [*sepsis*] *of the blood*, as a consecutive crasis resulting from the degeneration of another crasis.

The conditions we are here concerned with vary, as the anatomical results show, with the causal influences at work, as also with the differences due to pre-existent crases.

Generally speaking, these conditions manifest themselves in decomposition, in dissolution, in necrosis, in *a death* of the blood, and they comprise the commonly called scorbutic, the chronic and acute, putrid states of the circulating fluid. A very broad line of demarcation, coming under anatomical notice, and which separates the states referred to into *two*

series is that, *in the one case, the sepsis has the character of a fibrin-crasis, in the other, that of a deficiency of fibrin*. Accordingly, if we except the two common features of thinness and discoloration of the blood generally, a comprehensive view of all these states is not feasible.

Regarded from a clinico-anatomical point of view, the various conditions of septic crasis occur in the following forms:

1. *The purest and most simple forms of sepsis are :*

(*a.*) A decomposition or necrosis of the blood brought about without any cognizable agent of fermentation is that due to a shattered state of the nervous system and of its function, proving fatal with lesser or greater rapidity [sometimes in a very few moments], according to the measure and amount of the shock. To this category are to be referred decompositions of the blood consequent upon concussion and severe injuries, concussion from a fall, from the extensive laceration or the crushing of soft parts or of bones; upon extensive amputations; upon the continuous, exhausting activity of the muscles in violent convulsions, of whatever kind; upon electrical shocks received [lightning]; upon mental emotion of an overwhelming nature. A very striking exemplification offers in the decomposition of the blood not unfrequently called forth by a difficult and exhausting act of parturition involving palsy of the womb; cases which often prove fatal after a very few days, or even hours.

In all these cases the blood is found attenuate, in color comparable to a raspberry jelly, or of a dingy red, facile of imbibition, expanded in volume, often engaged in gas-development, frothy. The blood-corpuscles are swollen up, the serum being deeply reddened by hæmatin withdrawn from them. Coagula, if present at all, appear as very inconsiderable, soft, curd-like fibrin-clots. The frequent large peritoneal exudates, occurring more especially in puerperal decomposition, are dingy-red, dull, thin, sometimes rather viscid fluids. The dead bodies present but little and evanescent rigor, much inflation, extensive and deep lividity. The internal organs, the heart's muscle, the parenchymata are lax, flabby; the bloodvessel coats and the endocardium, discolored from imbibition; those parenchymata whose bloodvessels are most injected are more or less discolored by imbibed serum. The blood is always largely accumulated in particular sections of the vascular system, be it in the nerve-centres, in extensive patches of the mucous membrane of the stomach and intestines, in the sexual organs of women, but most particularly in the lungs, as hypostasis.

The corpses pass into putridity, under the phenomena of gas-development in the bloodvessels, emphysema in the textures, copious transudation of a dirty-red serum into the serous cavities, and spontaneous vesication of the epidermis.

(*b.*) Those decompositions consequent upon faulty diet [true scurvy], the reception of corrupt matter, of miasmata, of animal poisons into the blood, &c.

The dead bodies present, generally, the characters already specified. Owing, however, to the expansion of the decomposed blood, transudations of blood-dyed serum in the shape of ecchymoses of the textures, and actual hemorrhages, are especially apt to take place.

2. *A second form of decomposition* is that so frequently attendant

upon hypinotic crasis, and which we have before described as an exalta-
tion or degeneration of the typhous, of the exanthematous, of the
cholera-, of the drunkard's dyscrasis. Its relation to the fundamental
crasis may be regarded as a varying one. Here we may observe:

(*a.*) The putrid decomposition, impelled by a special external agent,
becomes complicated with the hypinotic crasis, a septic venom being
superadded to the exanthematous contagion or the typhous miasma.
There being essential putrid decompositions independent of those other
agents, and the symptoms of septic poisoning often occurring very early
during the progress of the other diseases cited, such a relation is placed
beyond all doubt.

(*b.*) The sepsis is a dissolution of the blood in the hypinotic crasis,
resulting from the profound injury inflicted by this crasis upon the
nervous system. This implies, either a very intense hypinosis [a very
intense miasma or contagion], or else a very susceptible nervous system.
Thus it may happen that typhus and exanthemata pass into putrid de-
composition even in epidemics by no means of a malignant type.

(*c.*) Or, in fine, it is possible that a hypinosis occasioned by miasma or
contagion, may, of itself and without the mediation of the nervous system,
become degraded into a putrid crasis, simply through putrid conversion
of the received miasma. It is in this sense, more particularly, that the
exaltation or the degeneration of a primitive crasis to the putrid is to be
understood. The blood and the corpse present the same appearances as
in the first form; the marks of decomposition and putrefaction being,
however, if possible, still more clearly defined. The different hyperæmiæ
are also more distinctly expressed—such hyperæmiæ as are proper to
the original hypinoses. They occupy, frequently in the shape of ecchy-
moses and hemorrhage of the textures into which they have degenerated,
those organs or parts of organs in which the original hypinosis had
localized itself; for example, the intestinal mucous membrane, in putrid
typhus; the common integument and the great tract of the respiratory
and intestinal mucous membranes, in exanthematous processes. Not
unfrequently, the septic crasis of this form localizes itself in deep-colored,
absolute stases, especially in peripherous organs, where, without a trace
of organizable products, they terminate in necrosis of the blood and of
the textures, with conversion thereof to a soft, humid, dingy, deep-red
mass,—a gangrene-slough.

A stringent differential diagnosis from the blood itself is, however, not
feasible in all the cases of the first and second forms. It is only to be
deduced from the anatomical disturbance of the solids generally, and, in
cases of the second form, in particular, from the products of the original
hypinosis,—typhus, exanthema, &c. For instance, where the intestinal
mucous membrane reveals the marks and residua of a typhous process,
the sepsis will have arisen out of the typhus-crasis. Where obvious
anatomical disturbance does not exist,—for example, in the case of con-
vulsions,—or where the products of a hypinosis, owing to the early
supervention of sepsis, are inconsiderable or only faintly indicated, and
where, lastly, clinical records are wanting, the diagnosis must needs rest
upon probabilities alone.

3. A third form, differing from the two former, is that of a sepsis of

the blood resulting from the fibrin-crasis, especially the aphthous, and from pyæmia. It manifests itself, in the first instance, as a putrid decomposition, as a necrosis of the fibrin, which forthwith possesses itself of the entire blood-mass. It has been already discussed under croupous crasis γ, and under pyæmia, as aphthous and purulent sepsis.

INDEPENDENT ANOMALIES OF THE BLOOD-CORPUSCLES.

An anomalous relation of the blood-corpuscles, founded in dyscrasial conditions of the plasma, occurs under several forms, some of which have been already adverted to, especially those of turgescence, pallor, or preternaturally deep coloration ; of augmented or diminished adhesiveness; and the like. As an independent anomaly, their diminished number in genuine chlorosis is alone recognized.

Other crases deserving of notice are:
The hemorrhagic crasis [hæmorrophilis], specified in the chapter on " Hemorrhage."
A crasis which determines the deposition or stratiform coagulation of a protein substance upon the inner coat of bloodvessels cannot, in our opinion, be pretermitted. The little that we have to advance upon this subject, however, we shall reserve for the chapter on " Diseases of the Arteries," in vol. iii.
The retention of urea in the blood occasions and sustains a hypinotic crasis; and in certain cases, as in acute nephritis and acute albuminuria, tends to induce a complete decomposition of the blood.
A biliary dyscrasis is produced in two different ways: first, through diseases of the liver and gall-ducts,—inflammation of the liver, obstruction of the latter through retention in the blood of the elements of the bile ; and secondly, through endosmosis of bile (resorption) into the bloodvessels.
The croupous-crasis and pyæmia often give rise, without demonstrable liver affection, to the elements of the bile being set at large in the blood.
In fine, there is a spontaneous biliary crasis which, running a very acute course under the most intense typhoid symptoms, and under colliquation of the parenchyma of the liver, proves fatal through decomposition of the blood. So intense and violent is the conversion to bile in the blood, that even in the portal circulation, previous to its entrance into the liver, the blood has the look of blood impaired by artificial contact with bile. It is a dingy-brown or yellow-red, tenacious, ichorous-looking fluid intermingled with whitish fat-streaks and jelly-like particles of fibrin. The bile secreted in the liver is so saturated, and at the same time so excessive in quantity, as to utterly dissolve the parenchyma of

that organ,—that is, the hepatic cells—determining a state of collapse and softening, which, in its appropriate place, we shall treat of as " Acute Yellow Atrophy of the Liver."

With regard to the constitution of the blood in gout, syphilis, chronic skin eruptions, and many other diseases, although in each a particular anomaly does no doubt exist, it has not as yet been given to morbid anatomy to substantiate its nature.

END OF VOL. I.

ROKITANSKY'S

PATHOLOGICAL ANATOMY.

VOLUME II.

A MANUAL

OF

PATHOLOGICAL ANATOMY.

BY

CARL ROKITANSKY, M.D.,

CURATOR OF THE IMPERIAL PATHOLOGICAL MUSEUM, AND PROFESSOR AT THE
UNIVERSITY OF VIENNA, ETC.

VOLUME II.

THE

ABDOMINAL VISCERA.

TRANSLATED FROM THE GERMAN,

BY

EDWARD SIEVEKING, M.D.,

MEMBER OF THE COLLEGES OF PHYSICIANS OF LONDON AND HAMBURGH,
PHYSICIAN TO THE NORTHERN DISPENSARY, ETC.

PHILADELPHIA:
BLANCHARD & LEA.
1855.

EDITOR'S PREFACE.

The principal hospital of the Austrian capital, the largest in the world, offers very extensive opportunities and unusual facilities for the cultivation of Pathological Anatomy. Exclusive of the Lying-in Hospital and the Lunatic Asylum, which occupy the same range of buildings, the Kaiserlich-Koniglich-Allgemeine-Krankenhaus[1] (Imperial Royal General Hospital) contains 104 wards, capable of receiving 2214 patients; 1247 beds being destined for males, and 967 for females. We find that, in 1838,[2] the number of patients treated amounted to 20,545; of these, 2678 died, giving a mortality of 13·03 per cent., or one death in 7·6 cases. As I am not provided with tables of mortality for other years, I am unable to state the annual average mortality in the hospital; but it does not appear, by a comparison with the mortuary tables of the Viennese Foundling Hospital, that the year 1838 was marked by peculiar endemic or epidemic influences. By the laws of the hospital, post-mortem examinations may be made of all who die within its walls.[4] " To examine all, or one half, would be impossible;" but as " generally from four to six bodies are opened daily,"[5] the extent of the field presented for cadaveric research may easily be estimated. For a series of years, the Professorship of Pathological Anatomy has been held by Dr. Carl Rokitansky, and the numbers of medical men of all nations who are attracted to Vienna by him, are the best

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[1] Knolz, Darstellung der Humanitäts und Heilanstalten Wiens, &c. Wien, 1840, p. 169. Wilde, Austria and its Institutions. Dublin, 1843, p. 124.

[2] Knolz, l. c. p. 316.   [3] Ib. l. c. p. 56.

[4] Ib. l. c. p. 190.   [5] Wilde, l. c. p. 180.

evidence of the manner in which he has availed himself of the opportunities at his disposal. All who have been fortunate enough to attend the Professor's demonstrations, will be able to award him the praise of untiring industry, of acute judgment, and candid research. Records of every case, taken down at the dictation of the Professor, are kept, and all interesting specimens are preserved for the Pathological Museum. Rokitansky has embodied the facts observed, and the conclusions deduced from them, in his "Handbuch der Pathologischen Anatomie," published in Vienna during the years 1841–1846. The original forms three large octavo volumes; of which the third, containing the Pathological Anatomy of the Organs of Respiration and Nutrition, and of the Uro-Genital Tract, appeared first; the second, embracing the Morbid Anatomy of the remaining Organs and Systems of the Body, followed; and the first, in which the Professor gives a philosophical survey of the entire Science of Morbid Anatomy, was published last. The Council of the Sydenham Society have determined upon issuing the translation in a similar sequence. Owing to the acknowledged difficulty of the author's style, it has however been thought advisable to divide the translation into four volumes, each of which is intrusted to a different editor.

The present volume contains the morbid anatomy of the Digestive Apparatus and the Uro-Genital Viscera, which constitute the greater part of the third volume of the original. The succeeding two volumes will embrace the remaining portion of the third, and the second volume of the original, and each of these will be complete in themselves, as far as regards the Special Pathology of the parts of which they treat. The first volume, which contains the Principles or Theory of Morbid Anatomy, is a scientific *exposé* of the deductions and inferences drawn by the author from the facts and illustrations given in the other sections of the work, and will be the last to appear. His views, as laid down in the volume of General Morbid Anatomy, are unintelligible to one who has not previously studied the volumes of Special Pathology; this accounts for the apparent inconsistency of publishing the translation in the

order adopted. Were it not that the Council of the Society have desired to adhere as much as could conveniently be done to the original, the present volume might with perfect propriety have been termed the first, and the succeeding volumes have been numbered in the order of their publication.

The fact of the Work having been selected for translation by the Council of the Sydenham Society, is in itself a proof that it is deserving of the high estimation in which it has been held by all pathologists acquainted with continental literature; but it may not be superfluous to state that the value of the Professor's remarks is enhanced by his being entirely unfettered by preconceived notions or prejudiced views, as to the disease of the individual brought to the dead-house for examination. "Rokitansky," as Mr. Wilde correctly remarks, "differs from all other pathologists, in not engaging in the study or treatment of disease during life; he is not a practical physician, and seldom sees one of the many hundreds of cases, whose bodies he dissects." English readers will probably sometimes desire more positive statistical data than the author vouchsafes, and I cannot but express a hope that in the new edition which Professor Rokitansky is preparing, he will in some measure repair an omission, which necessarily weakens his conclusions, and deprives them of that basis which the student looks for in pathological anatomy, more even than in other departments of the natural sciences. I may, however, venture to assert, that no one will read his descriptions of post-mortem appearances without feeling convinced that they are drawings from nature.

Of the difficulties connected with the translation, I will only say that they are much increased by the figurative style of the author. He constantly uses terms in a sense peculiar to himself, and his total disregard for the ordinary rules of composition is an additional and frequent source of obscurity. It has been necessary to adopt a few terms in the translation which, though new to the reader, have been thought to convey most accurately the peculiar and idiomatic expressions of Professor Rokitansky; this has not, however, been

done except where no word or phrase familiar to British pathologists could be found exactly to convey the meaning of the author.

In regard to the translation generally, I can only express a hope that I have not perverted the sense of the original by the necessary reconstruction of many passages, nor that, in adhering too closely to the German, I have failed in making the English edition readable.

In conclusion, I avail myself of this opportunity to acknowledge the honor conferred upon me by the request of the Council of the Sydenham Society to undertake the translation; and I have great pleasure in recording the obligations which I am under to Dr. J. R. Bennett, the Secretary of the Society, for the courtesy and assistance he has afforded me while the work was going through the press.

E. S.

Brook Street, Grosvenor Square.

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PART I.

ABNORMITIES OF THE DIGESTIVE APPARATUS.

CHAPTER I.

ABNORMITIES OF THE ALIMENTARY TUBE.

SECT. I.—ABNORMITIES OF THE MOUTH AND FAUCES.

§ 1. *Deviations in Form and Size.*—As excess of development, we have here to mention the more or less complete repetition of one or more parts, which sometimes advances to such an extent that the bones of the jaws, the mouth, and the tongue are double, and unite in one common gullet. As defective formation, which may generally be distinctly traced to an arrest of development, we meet with complete absence of the cavities of the mouth and fauces (astomia), or imperfect development of individual parts, as of the superior maxilla, giving rise to an imperfect development of the face (ateloprosopia), of the lower jaw (agnathia and atelognathia), of the lips (achelia and atelochelia), of the tongue, &c.

The most common and important cases of arrest of development are: Fissures of the upper lip, on either or both sides of the mesian line, corresponding to the union of the intermaxillary with the superior maxillary bones, which may or may not present a fissure also (harelip, labium leporinum); fissures of the palate, caused by the absence of the os incisivum and the middle portion of the upper lip, or by the mere disunion of the palatal processes in the middle line; or again, by defect in the latter on either or both sides, with or without an accompanying absence of the os incisivum; the fissures of the soft palate, varying equally in degree, from complete division to a mere indication of the anomaly in a slight notch of the uvula. Fissures of the tongue are extremely rare, and seldom present more than a mere trace of division; fissures of the nether lip, and of the lower jaw in the mesian line, are equally rare.

Closure of the mouth (atresia oris) is a rare occurrence, as contrasted with the frequency of a similar condition at the anus.

Numerous morbid processes are followed by anomalies that resemble the above congenital malformations, such as partial or total loss of the lips, of the cheeks, of the palate, contraction of the mouth to a degree approaching atresia, adhesion of the cheeks to the maxillæ, of the tongue to the cavity of the mouth, contraction of the fauces, &c.

Increase of size, as a result of hypertrophy, occurs chiefly in the shape of hypertrophy of the lips and the tongue; it varies in degree, and is peculiar to the scrofulous cachexia and to cretinism; it is also presented as hypertrophy of the tonsils, and of the glandular stratum of the soft palate, as hypertrophy of the uvula, and occasionally of the gums.

The opposite condition, i. e. diminution of size and, taken in reference to the capacity of the oral cavities and the fauces, contraction, occurs in an eminent degree in the shape of atrophy of the tonsils, and also of the other muciparous glands, and of stenosis of the isthmus faucium. The latter results from cicatrization of syphilitic and scrofulous ulcers, and occasionally proceeds to such a degree that the isthmus scarcely permits the passage of a pea.

§ 2. *Textural Diseases.*—Of these, inflammatory processes, and especially those affecting the mucous membrane and its glands, demand primary consideration.

Catarrhal inflammation is particularly liable to attack the pharyngeal mucous membrane, and to be associated with a marked affection of the tonsils, in the shape of cynanche tonsillaris. It is either acute, or chronic, is apt to return, and become habitual; it frequently, and in many individuals constantly, passes not only into superficial ulceration, but even into phlegmonous inflammation and the formation of abscesses in the tonsils; or it leaves a permanent relaxation of the fauces, with a varicose state of the vessels, elongation and œdema of the uvula, chronic hyperæmia, and tumefaction of the tonsils, and blennorrhœa of the tonsils and fauces. It frequently extends to the mucous membrane of the rima glottidis and of the larynx, as well as to that of the Eustachian tube.

The croupy process of the mucous membrane of the mouth and fauces occurs, in the first instance, in the well-known form of thrush and aphthæ in children; and in adults, commonly with an epidemic character of an adynamic septic type, as malignant (gangrenous, aphthous) sore throat (angina gangrænosa, the diphtheritis of Bretonneau). In the former case, after a previous vivid or dark purple reddening of one or more papillæ, and the vesicular elevation of the epithelium at the point and the sides of the tongue, dots or patches, of the size of a lentil or pea, appear on the inner surface of the lips and cheeks, and finally, on the mucous membrane of the fauces. They present an exudation, which has a frosted, or flocculent, or villous appearance, or is more of a membranous character, and extends into the cavities of the follicles; it is of a grayish, or yellowish-white color, and of a lardaceous, or soft, creamy, or fluid consistency; if removed, a shallow excoriated depression, surrounded by an inflamed margin, remains, on which the exudation is repeated, involving a further destruction of the mucous tissue.

In the second instance, livid spots, which rapidly coalesce, and become invested with a dirty, gray, shaggy, pultaceous and sanious exudation, form upon the softened, bleeding gums, and the mucous membrane of the cheeks, the fauces, and the tonsils. The gums themselves ultimately degenerate into a bad-looking, pulpy, sanious mass, and the mucous membrane of the cheeks and fauces, underneath the exudations, is equally found converted into a friable, fetid pulp, or a firm slough.

These processes often extend to the pharynx and the œsophagus, though scarcely ever to the respiratory passages; they are sometimes

complicated with exudative processes on other mucous and serous membranes.

Genuine (primary) pharyngeal croup occurs rarely; it is either the result of an extension of tracheal croup, or, similar to exudative processes with products of a different nature, an anomalous process of a specific, acute, exanthematic, impetiginous, typhous character, or it is the result of a spontaneous or purulent disorganization of the blood. It not unfrequently leads to acute gastric softening.

Pustular inflammation occurs in the fauces in variolous disease; the mucous membrane being tumefied, and invested with a plastic mucous secretion.

There are other circumscribed inflammations of the buccal and pharyngeal mucous membranes, which are remarkable for their tendency to pass into ulceration, viz. the syphilitic, syphiloid, mercurial, and scrofulous inflammations. They are generally characterized by their peculiar red tinge and defined edges, and give rise to various products which dissolve the tissues in a peculiar manner, and consequently to specific ulcers. Syphilis, more particularly, is, in this phase of its existence, and so far as the alimentary tract is concerned, limited to the fauces.

The last-mentioned ulcers and aphthous ulcerations, give rise to more or less considerable loss of substance in the mucous membrane and the subjacent tissues, after the cure of which, white, indurated, elevated retiform, and tendinous cicatrices remain, which induce a corresponding contraction.

Among the inflammations attacking individual structures, we have to mention inflammation of the gums, especially the rheumatic variety, with coexisting affection of the alveolar periosteum, as also the scorbutic forms and the inflammation of the tongue with its occasional termination in deep-seated suppuration.

An important disease that we must here speak of, is *noma*, a phagedenic ulceration which commences at the inner surface of the cheek, and rapidly spreads, involving the soft parts in gangrenous destruction. A livid congestion of the mucous membrane precedes, corresponding with which there is an erysipelatous redness externally; a hard tumor then forms; the tissues are broken up into a pulpy sanious mass, the subcutaneous cellular tissue is dissolved into a pale yellowish, gelatinous, oily mass; the superficial integument, at the same time, becomes pale, and is converted into a similar mass, or dries up into a dry, brownish-black eschar; the surrounding parts presenting erysipelatous redness and œdema. This process not unfrequently spreads over the entire cheek and gums, denuding the maxillary bones, and involving them in a species of calcination. (Froriep.) It is rarely met with except in children, and commonly attacks weakly cachectic individuals; it frequently occurs as a sequela of exanthematic diseases, and of typhus, and then represents a degeneration or an anomaly in the latter.

§ 3. *Adventitious Growths.*—Among these we have first to notice the fibroid tumors occurring on the alveolar processes under the name of *epulis*, and in the fauces as *polypi;* they have a broad base, or are pediculated, are of soft or hard texture, of a rounded, oval, or lobulated

form, and are invested with a spongy, ulcerated, and often bleeding mucous membrane.

Cancerous morbid growths do not often occur, if we except two cases in which a malignant tumor has made its way into the mouth or the fauces from without. Cancerous degeneration of the tonsils is peculiarly rare, and the cases that have been recorded as such have almost invariably proved to be instances of mere hypertrophy with induration. Still cancer of the lips, and especially of the nether lip and of the tongue, where it chiefly attacks the posterior half, is not unfrequent; from these points it branches out between the muscles at the floor of the mouth, at the sides of the fauces down to the neck, and on the tongue it gives rise to an irregular, sinuous, callous, and fungous ulcer, which is surrounded by an indurated margin of mucous tissue.

§ 4. *Anomalies in the Secretions.*—We have, under this head, to notice, in addition to those already spoken of, the different secretions which cover the mucous membrane of the mouth, and especially of the tongue, in various chronic and acute diseases, and the concretions occurring in the sinuses of the tonsils. In scrofulous subjects the tonsils are often affected, in addition to hypertrophy and habitual hyperæmia, with a peculiar blennorrhœa, and the purulent secretion not unfrequently becomes inspissated, so as to form tubercular cheesy plugs, or even chalky concretions. These, in their turn, keep up a perpetual state of irritation in the tonsils.

SECT. II.—ABNORMITIES OF THE PHARYNX AND ŒSOPHAGUS.

§ 1. *Defect and Excess.*—It is only necessary to allude to the congenital absence of this passage as occurring in acephalous monsters, to its partial defect with a blind termination, its fusion with the trachea, to the saccular dilatation of the canal resembling the craw of a bird, to its being double in disomatic monsters, and to the very rare occurrence of insulated fissures in individuals that are otherwise normally built (Meckel).

§ 2. *Acquired Abnormities of the Calibre, and the thickness of the Parietes.*—Anomalies of the calibre present themselves in the shape of *dilatations* or *contractions*.

Dilatation may affect the pharynx and œsophagus throughout, or almost throughout, and give them a cylindrical or a fusiform appearance; when it affects the œsophagus, it may be partial, in which case either pouches are formed, which involve all the coats of the œsophagus, and which may be developed at all points of its circumference; or the mucous membrane alone dilates, giving rise to diverticula or herniæ of the mucous membrane through the muscular coat.

The *first* variety has only been observed in a few cases, though when it occurs it is developed to an advanced degree, and presents thickening of the parietes, and particularly hypertrophy of the muscular coat. It appears to be sometimes the consequence of concussion of the œso-

phagus by a blow or contusion of the chest. One preparation, in the Viennese collection, presents an œsophagus large enough to allow the passage of a man's arm; in another case (Hanney), the circumference of the dilated passage was six inches.

Dilatations of a lower degree sometimes occur, in which the œsophageal coats are in a condition of paralytic relaxation and attenuation.

The *second* variety is seen at various points, and in various degrees, above contractions, and especially above scirrhous strictures.

The *third* variety is rounded; or, if it increases to a considerable size, we find cylindrical or conical dilatations of the mucous membrane, occupying the lateral portions of the œsophagus. They may form at all parts of the œsophagus, but they are most frequently seen near the bifurcation of the trachea, and they attain the greatest size at the inferior section of the pharynx (Baillie), where the fibres of the inferior constrictor have a horizontal position. The mucous membrane is protruded between the muscular fibres, and becomes dilated by the food that enters; it is at last forced out in the shape of a cylindrical appendix, which lies between the vertebral column and the œsophagus, in a line with the axis of the pharynx, so that all ingesta pass into it, and death from starvation results.

The origin of the diverticulum is in many cases peculiar; thus we are acquainted with an instance in which the mucous membrane of the œsophagus was dragged out in consequence of the shrivelling of an adherent tracheal gland.

The fauces and the œsophagus are not unfrequently subject to contraction, from being compressed by the enlarged thymus gland, by aortic aneurisms, adventitious growths, &c.; but the contractions resulting from textural changes in the coats are of more importance, and among these we must more particularly allude to stenoses brought on by cicatrization after corrosion by caustic substances, and by cancerous affections (cancerous stricture). Of both we shall have further occasion to speak in the sequel.

§ 3. *Anomalies of Position.*—Among these we may reckon the position of the œsophagus to the right of the spinal column, accompanying a lateral transposition of the intestines, the changes produced by curvatures of the spine, the flexures or dislocations of the pharynx and œsophagus, brought about by hypertrophy of the thyroid gland, by aneurisms, abscesses, morbid growths, &c.

§ 4. *Solutions of Continuity.*—Among these we reckon, besides wounds of the pharynx and œsophagus, by means of fire-arms, or other penetrating instruments, the injuries and perforations caused by foreign bodies that have been swallowed, the perforations from softening, ulcerations, gangrene, or from absorption in consequence of pressure, e. g., by aneurisms, by which means the most various passages, communicating with the neighboring serous cavities, the respiratory organs, the adjoining vascular trunks, &c., may be established, and lastly, those very rare occurrences of spontaneous rupture, without previous alteration in the tissue.

§ 5. *Textural Diseases.* 1. *Inflammation.*—Catarrhal inflammation :—This is rarely seen very intense in the acute, but certainly not uncommonly in the chronic form. The appearances produced in that case are œdema of the mucous membrane, with a dirty-brown or slate-colored tinge, enlargement of the follicles, blennorrhœa, and an exuberant formation of epithelium, and hypertrophy of the muscular coat. It is possible that when the cardiac orifice is the seat of inflammation, the consequent hypertrophy of the circular fibres, and the narrowing of the passage, may give rise to those enormous dilatations of the œsophagus, of which we have already spoken. It frequently occurs as an idiopathic, but also as a secondary affection, and in the latter case chiefly in connection with impetigo.[1]

Croupy (exudative) inflammation—occurs as an aphthous process in children, as true diffused croup, coexistent with, or unaccompanied by, croup of the tracheal, bronchial, and pulmonary (pneumonia) mucous membrane, mainly in typhoid cholera, but also as a secondary affection and as an abortive exanthematic and typhoid process, the product of a purulent condition of the blood, brought on by tubercular and cancerous cachexia.

Pustular inflammation :—To this class belongs the rare occurrence of varioloid pustules, the pustules of metastatic herpes, and the pustules which occur at the lower third of the œsophagus in consequence of the internal administration of tartar emetic in large doses.

In addition to the above varieties, we meet with inflammation, which is produced by the corrosion of caustic substances; the coexistent affection of the oral cavity and the fauces being commonly of a lower, that of the gastric mucous membrane of a higher degree. We refer the reader, for an investigation of this process and its consequences, to the following pages, as we purpose examining it among the diseases of the stomach, in reference to all the tissues we have alluded to ; at present we merely add, that in those cases in which the mucous membrane has been destroyed by the energetic action of the poison, it is replaced by a serous and sero-fibrous tissue, which gives rise to peculiar valvular and annular strictures of the œsophagus, somewhat analogous to those consequent upon dysentery.

2. *Softening.*—Softening occurs at the lower third of the œsophagus, and is commonly associated with softening of the stomach. On account of the identity of the two affections, we refer the reader to the section on the Diseases of the Stomach ; the more, since the process is observed more frequently in the latter, if not in a more fully developed form. We must however add, that it is particularly liable to affect the left side of the œsophagus, and then to cause perforation, in consequence of which we have destruction of the cellular tissue and the left mediastinum, and effusion of the gastric contents into the left pleura.

3. *Morbid growths.*—*a.* Anomalous fibrous and fibro-cartilaginous tissue occurs as a fibroid or fibro-chondroid tumor, in the shape of a

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[1] [To render this passage intelligible, it may be well to remind the reader of the theory very prevalent among German pathologists, which attributes the majority of chronic diseases to dormant or suppressed cutaneous eruptions. Autenrieth may be mentioned as the chief supporter of this doctrine.—ED.]

movable bluish-white concretion, varying in size from a pin's head to a kidney bean, and occupying the submucous cellular tissue of the œsophagus; and also as a fibrous polypus, attached by a neck to the perichondrium of the cricoid cartilage, and depending from it into the œsophagus; the free surface is frequently lobulated, and it is invested by mucous membrane.[1]

b. Tubercular deposits are rarely, if ever, found in the œsophagus, and they must not be confounded with the tubercular degeneration of the neighboring lymphatic glands.

c. Carcinomatous affections, in the shape of scirrhus and medullary sarcoma, are more frequent. This is generally a primary disease, though the œsophagus may become secondarily involved in carcinomatous degeneration of the mediastina. In the former case the cancer may be found in every portion of the pharynx and œsophagus; but the upper part of the thoracic portion of the latter, and the inferior part of the former appear to be more frequently attacked than the cardiac portion of the tube. The degeneration generally affects the circumference of the passage, and thus gives rise to annular stricture, the extent of which must correspond to the extent of the carcinomatous deposit. The œsophagus soon becomes fixed by the adhesion of the diseased mass to the spinal column. The metamorphosis of the morbid product frequently gives rise to the formation of large sanious cavities, the carcinomatous parietes of which are covered with fungoid granulations, and with which the œsophagus communicates above and below in a transverse or slanting direction. The sanious discharge frequently causes ulcerative destruction of the neighboring tissues, by which means communications are established with the trachea and the bronchi; occasionally even the arterial coats, which are otherwise endowed with great power of resisting such influences, become involved, and communications with the arterial trunks in the vicinity, and more especially with the aorta and the right pulmonary artery, are established.

Cancer of the œsophagus generally occurs in an isolated form, i. e. without a coexistence of the disease in other organs.

§ 6. *Foreign Bodies.*—Sometimes small hard bodies, such as cherry-stones, give rise to serious occurrences, by causing, at different parts of the œsophagus, but chiefly at the lower constrictor of the pharynx (Baillie), the formation of diverticula. Very large and hard bodies, such as are sometimes swallowed by lunatics, remain fixed at a certain spot, and may cause inflammation and suppuration; or, by extreme pressure, even give rise to gangrene and perforation of the œsophagus. Pointed and rough bodies, and especially needles and fish bones, are still more likely to produce perforations of the œsophagus in different directions, and to reach the aorta or trachea.

SECT. III.—ABNORMITIES OF THE PERITONEUM.

§ 1. *Defect and Excess of Formation.*—Arrest of development in the peritoneal sac occurs in the shape of fissure in the mesial line, or exter-

[1] Oestr. Jahrb. xxi. 2.

nal to it; in the case of the diaphragm being absent, of a fusion with the pleura; as defective development of the mesentery at various points, as defective development or complete absence of several other folds, the omentum, the appendices of the omentum, as deficiencies in these parts, &c.

Excess of development frequently occurs in the shape of unusual length of the duplicatures, e. g. of the omentum, the mesenteries, &c., or of supernumerary folds and peritoneal pouches. These are chiefly found in the hypogastric, and more especially in the iliac and in the inguinal regions, and near the fundus vesicæ. There is access to these sacs by a well-defined fissure or ring, which is frequently surrounded by a tendinous band, lying in the duplicature. In the case of their inclosing portions of the intestine, they may give rise to internal incarceration, which, on the one hand, resembles external hernia, on the other, does not afford the diagnostic signs peculiar to this affection, and may, therefore, be considered as forming a transition between external and internal hernia. Similar formations, such as a delicate serous envelope of the small intestine, must be explained by an original anomaly in the development of the peritoneum.

§ 2. *Anomalies in the Size and Form of the Peritoneal Sac.*—Among these we reckon a general increase of the peritoneal surface, corresponding with a congenital enlargement of the abdominal cavity and the intestines; the acquired extension, which may be uniform, as the result more especially of an accumulation of serous fluid (ascites); or partial, as presented to us in congenital or accidental hernia, and in the abnormal size or acquired elongation of single folds; the latter are brought about by dislocations of the abdominal contents, which arise spontaneously, or from a variety of causes, are most frequently seen affecting the mesenteries and the omentum, and are of signal importance in reference to the causation of internal hernia.

A small peritoneum is the result of an arrest of development in the abdominal cavity, subordinate to the development of the pleura; an apparent diminution may be caused by dislocation of the abdominal contents, as in large scrotal or diaphragmatic hernia.

The anomalies of *form* are involved in the above anomalies of size.

§ 3. *Solutions of Continuity.*—The peritoneal sac is liable to solutions of continuity from penetrating wounds of the abdomen, from the effect of powerful concussion, of excessive bodily exertion, from spontaneous ruptures of the hollow or parenchymatous organs it invests, in consequence of traumatic injuries, from contusion, rupture, and separation of the subjacent tissues. The extent and nature of the injury vary as much as its situation.

§ 4. *Abnormities of the Tissues.* 1. *Hyperæmia.*—Hyperæmia is either general, or, when caused by the congested state of an organ invested by the peritoneum, partial. It gives rise, on the one hand, to an increase of secretion and to dropsical accumulations in the peritoneal cavity; on the other, to hypertrophy and thickening of the serous tissue,

and to the development of a subserous fibroid or fibro-chondroid growth. The peritoneal investment of the spleen offers the best illustration of the latter.

2. *Inflammation* (Peritonitis).—Inflammation of the peritoneum presents the symptoms common to inflammation of serous membranes. It may occur as an idiopathic affection, or in consequence of traumatic lesions of the abdomen, of pressure from incarceration, or from contact with the atmosphere, with the contents of the stomach or intestines, with bile, urine, vaginal secretions, blood, or pus. It may be presented to us in the form of spontaneous or rheumatic peritonitis; it may occur as the result of a propagation of disease from the organs contained in the peritoneal sac. The most frequent form is the one attributed to metastasis, in which the peritoneum, from the large serous surface which it offers (and in this respect it presents an analogy with the vast tract of the intestinal mucous membrane), and, owing to its proximity in many cases to the primary seat of disease, is converted into a focus of extensive exudative process. To this class we refer more particularly the inflammatory and exudative processes of puerperal fever, of which we shall have occasion to speak more fully at a future period.

The affection is either general or partial. In the former case, it involves the peritoneum of the abdominal parietes, of the parenchymatous viscera, and of the colon (enteritis peritonealis), though generally with a predominance in one or the other. In both it may appear in the *acute* or *chronic* form.

Acute general peritonitis very often terminates fatally, with symptoms of intestinal paralysis, and with imminent or existing ileus; or death is caused by exhaustion, which gives rise to the formation of large fibrinous, puriform and purulent exudations. We then find, in addition to the symptoms of serous inflammation, an enlargement of the intestine; it is expanded by gases (tympanitis), by thin watery and feculent matters; the coats of the intestine, and chiefly the interstitial cellular tissue and the mucous membrane, are tumefied, the muscular layer is pale, and they are all fragile and friable.

The tumefaction of the intestinal coats is commonly owing to an infiltration of the tissues by a watery fluid, and increases in proportion to the degree in which the mucous membrane participates in the exudative process. It occurs in the most exquisite degree in the so-called metastatic form, in that inflammation of the peritoneum which is the local expression of a general disorganization of the blood, i. e. in the puerperal type. In this case the mucous membrane presents a relation similar to that exhibited by the peritoneum in exudative processes of the mucous membrane, in Asiatic cholera, in colliquative diarrhœas generally, or in dysentery in the shape of a mucous secretion, or of a delicate indication of plastic exudation, evidenced by mere loss of brilliancy and smoothness.

The ileus,[1] which occurs in general peritonitis, is, like the dilatation of the intestine, the consequence of paralysis of the muscular coat; a relation observed to exist wherever muscular fibres are subjacent to serous

[1] Vide Oestr. Jahrb. xviii. 1.

membranes. The exudation of plastic lymph, especially in the case of various abnormal contortions, is also likely to contribute to its occurrence by binding down the intestine. We may easily infer which will be the terminal point of the antiperistaltic movement, or of ileus, in cases of enteritis peritonealis. As the inflammation of the peritoneum is accompanied by paralysis of the entire intestine, it can be no other portion of the intestine than the duodenum, at the lower end of which the peritoneum, and consequently the inflammation and paralysis, terminate, and which by itself is, under no circumstances, capable of controlling, by its peristaltic action, the accumulated contents of the small intestines which are being thrown into it. Yet cases which, like puerperal peritonitis, are generally accompanied by diarrhœa, form exceptions to this rule.

The exudations seen on the peritoneum, exhibit, in reference to quantity and minute structure and to their metamorphoses generally, all those variations which we have cited in the general remarks on inflammation of serous membranes. The general remarks there made with regard to the acute and chronic forms of the process, are equally applicable here. Nevertheless, we observe numerous peculiarities in peritonitis, to which we must here advert. We very frequently find extensive cacoplastic, disorganized, discolored, septic exudations, accompanied by an almost imperceptible increase of redness and vascularity; they are more especially associated with puerperal, septic processes in the uterus. Plastic exudations become organized into cellular or cellulo-serous tissue. This remains attached to the peritoneum in the shape of a pale, grayish-white, or bluish-red and vascular, or slate-colored accumulation; or it forms a new movable cellulo-serous investment to all organs enveloped by the peritoneum, or it assumes the shape of flakes or strings, which pass from one to the other in different directions. In the two latter cases, various tense or loose adhesions between the abdominal viscera, among themselves, or with the parietes of the abdomen, will result; of these the following are the chief:

Adhesions of the intestinal coils, producing very manifold transpositions among themselves, and with the mesentery, with the colon, in reference to the hypogastric parietes of the abdomen and the pelvis, the bladder, and the internal sexual organs of the female;—adhesions of the omentum in various degrees with the hypogastric parietes of the abdomen, and more particularly of the inguinal region, and with the internal sexual organs of the female; the omentum may be folded together, or rolled up, and stretched across in a slanting direction to either of the inguinal regions, or it may descend with a furcate fissure to both, so as to attach itself at these points, and form a vertical, or, if passing under the colon transversum, a rounded horizontal band; thus giving rise to a species of diaphragm, which separates the mesogastric and hypogastric regions;—and adhesions of the parenchymatous viscera to the adjacent parietes and to the neighboring viscera.

Adhesions between the omentum and the colon, and the anterior parietes of the abdomen, are found chiefly in chronic peritonitis, but they are not of frequent occurrence.

The corded exudations may, in various ways, cause incarcerations of the intestine.

Or the exudations undergo metamorphoses, so as to give rise to tendinous or fibro-cartilaginous laminæ, sometimes of uniform thickness, and with defined edges, sometimes of areolar or cribriform structure, at others, uneven, lobulated, granulated, thinning off towards the circumference, glued on to or fused with the thickened peritoneum. This is remarked, principally, on the omentum and the fold of the intestine in hernia, or on the hernial sac itself, and also on the convex surface of the spleen, on the liver, sometimes on the uterus and its appendages, and in rare cases on the entire extent of the peritoneum.

The *chronic* form of inflammation, which affects the exudations that have already been deposited, and creeps on with occasional exacerbations, presents the following peculiarities: It occupies the intestine only; or at least that part of the circumference of the intestine chiefly which is not affected by adhesions resulting from a previous process, as well as the opposed parietal surface, which in various degrees is limited by, or free from, adhesions. The consequence is the formation of a coagulum, which covers the anterior surface of the already agglutinated intestines, passing from them to the parietes, and thus inclosing a sacculated space which contains the fluid portion of the exuded matter. In such a case the intestines, and more particularly the small intestines, form a flattened round mass, riding upon the vertebral column, and invested anteriorly by the posterior lamina of a pseudo-membrane, which contains in its cavity a varying amount of fluid.

Hemorrhagic exudation is frequently seen on the peritoneum; it forms large, saturated coagula, disposed in thick layers. Thin strata present a deep black or bluish-black discoloration, the effect of the intestinal gases.

Peritonitis occasionally terminates in suppuration or gangrenous decomposition, *phthisis* and *gangræna peritonœi*. With the exception of those cases, in which purulent or gangrenous disorganization and perforation result from a propagation of the disease from other tissues, this termination occurs under the following conditions:

a. The peritonitis itself yields a purulent exudation, and the peritoneum is destroyed by suppuration, followed by the denudation and suppuration of the subjacent tissues. This occurs chiefly in partial, circumscribed peritonitis, when the exciting causes, viz., suppurative inflammation or gangrenous infiltration of an organ, accompanied by a purulent or ichorous discharge on the peritoneal surface, continue.

b. Occasionally a certain portion of a fibrinous exudation does not become organized, but being diffused through the interstices of the adventitious membrane, melts into a creamy pus, which, being in close contact with the latter, produces at once in it and in the peritoneum suppurative inflammation and suppuration.

In either case, ulcerative perforation of the intestine or of the abdominal parietes frequently proceeds from the morbid process in the peritoneum; and when, as is sometimes the case, both occur simultaneously, fistulæ result.

Partial peritonitis in many cases appears to be a *molimen naturæ* destined to circumscribe destructive processes, to arrest imminent or existing discharges which are hostile to the integrity of the peritoneum. To

these belong, first of all, the circumscribed inflammations of the peritoneum, which take place in the vicinity of approaching or existing perforations of the stomach, the colon, the vermiform process, in the vicinity of purulent accumulations external to the peritoneum, threatening perforation and discharge into the peritoneal cavity, and the like. By this means, general peritonitis, and a consequent rapid and fatal termination are frequently postponed for a long time; yet, whilst the exciting cause continues, peritoneal phthisis, with its consecutive disorganization, must ensue; or the adhesions which limit the focus of inflammation give way, and, in consequence of the free discharge of its contents, general peritonitis follows, or this may take place without the occurrence first mentioned, in consequence of the violence and extent of the inflammatory process at the original seat of the disease.

Gangrene of the peritoneum occurs as a yellow slough, in consequence of pressure or traction caused by external or internal hernia, in consequence of its being deprived of the subserous cellular tissue where it overlays perforating ulcers of the intestines and abscesses; or as gangrenous disorganization and conversion into a blackish, moist, ragged, and friable tissue.

3. *Heterologous formations. a. Anomalous occurrence of cellular and of serous tissue.*—This appears on the peritoneum in the shape of the above-mentioned organizing processes of a plastic character, and especially as serous cysts, in which case the pseudo-membrane includes, during its organization, a portion of the fluid exudation, and receives an internal serous investment. Such bladders are either connected with the peritoneum by means of a neck or stalk, or adhere to it by a broad base. In rare cases we find cysts with various contents as new formations on certain portions of the peritoneum, and then most frequently on the omentum.

b. Anomalous fibrous (fibro-cartilaginous) tissue—owes its origin to the inflammatory process in a similar manner as that above described. But there are, besides, other instances of the occurrence of this tissue, in the shape of fibro-cartilaginous smooth or lobulated laminæ, projecting granulations, &c., occupying the subserous layer of the peritoneum. They are observed in old hernial sacs; rarely, as compared with the pleura, on the parietal, but very frequently on certain portions of the visceral plate of the peritoneum, owing to the hyperæmia which takes place here, as, for instance, in the case of the spleen. This tissue also occurs in the subserous cellular tissue of the uterus and its appendages, and on the colon in the shape of a fibroid growth; in the former case, it reaches a considerable magnitude; in the latter, it rarely exceeds that of a lentil or a pea.

c. Anomalous osseous tissue—is developed from the above-mentioned tissue in the shape of compact, smooth, or uneven lobulated plates of varying thickness. The fibroid growth in the subserous cellular tissue of the intestinal canal is very rarely the seat of ossification.

d. Tubercle.—Tuberculosis of the peritoneum occurs in the various forms of which we gave a general sketch when treating of the tubercular disease of serous membranes, both as an acute and as a chronic affection.

It is very frequently found in the circumscribed form on those parts of the peritoneum which correspond with tubercular ulcers of the mucous membrane ; and we here trace all the forms peculiar to peritoneal tubercular generally. It commonly does not appear until the secondary tubercular infiltrations have extended from the inner surface of the intestinal tube into the muscular coat, and thus involved the tissue of the peritoneum itself.

The tubercular exudations on the peritoneum likewise give rise to all the adhesions we have above spoken of, generally producing a more intimate agglutination between the viscera. Peritoneal tuberculosis occasionally appears as a primary affection, the peritoneum being the first tissue attacked by tubercular deposit ; but it occurs more frequently after the cachexia has been evidenced by tuberculosis of another organ. Thus it allies itself to pulmonary, intestinal, and cerebral tubercle, and it very commonly terminates in tubercular affections of the abdominal lymphatic glands, and in the female sex more particularly in tuberculosis of the uterine and vaginal mucous membrane. The acute forms of peritoneal tuberculosis are, in most cases, complicated with a corresponding affection in the spleen, the liver, the kidneys.

The reflected action upon the adjacent muscular fibre, which occurs in peritoneal inflammation, is presented to us in a much higher degree in the tubercular exudative process. We find that the intestinal coats, in addition to being tumefied, become very friable ; there is increased exhalation from the inner surface of the intestine, and liquefaction of its contents, the muscular coat becomes pale, is easily lacerated and broken up, and even the muscles of the abdominal parietes waste and lose color.

Peritoneal tubercle, and especially the granulated variety, rarely passes into the stage of softening ; when it does so, it may cause tubercular suppuration or peritoneal phthisis, and consequently phthisis of other adjacent tissues ; cretification is a still more unusual occurrence, but the tubercular disease frequently becomes stationary.

e. Carcinoma.—The peritoneum is either secondarily affected by carcinoma, a cancerous growth originally generated externally to it, approaching and involving it in its metamorphosis, perforating it and penetrating into its cavity ; or the carcinoma is produced without such antecedents, though commonly occasioned by a carcinomatous affection in the neighborhood, in the vicinity of which it is formed ; or, lastly, it occurs in some rare cases, altogether independently of such causes, as a primary affection of the peritoneum. It must, therefore, with the exception of the last-mentioned unfrequent case, be considered as the product of cancerous cachexia which has already found a nidus and a local habitation.

The most common form of carcinoma, into which, however, the other carcinomatous growths, which give rise to its appearance on the peritoneum, usually degenerate, is the areolar, and, second in order, the medullary species.

The former appears as a hard, crystalline, transparent, and discrete cancerous follicle, resembling tubercle, and of the size of a hemp or millet seed ; in the acute variety it is generally thickly sown over a large extent, and even spreads over the entire peritoneum, or it occurs as a layer of areolar cancerous tissue, varying in thickness, or as a circumscribed,

round, lobulated aggregation. The omentum is very commonly found to shrivel up and to degenerate into a transverse band, or, in the opposite case, with an enormous increase of size, into areolar cancer.

Medullary carcinoma frequently occurs in the acute form, as the white or colored (melanosis) encephaloid variety, either deposited in layers, or more commonly as compressed, rounded, medullary nodules, of different dimensions.

In the fibro-carcinomatous degeneration of the peritoneum and its subserous cellular tissue, which occasionally extends over the entire peritoneum, we invariably perceive an atrophy and condensation of the tissues, and, in consequence, a contraction of the carcinomatous folds of the peritoneum, e. g. in the mesentery.

Peritoneal cancer is commonly complicated, in the manner above described, with gastric intestinal and ovarian cancer, and then also with uterine and hepatic cancer, the medullary form prevailing in the latter case. We must not omit to allude, at this place, to the nodulated retro-peritoneal cancerous formations of Lobstein, which commonly take their origin in the glands of the lumbar plexus or other subordinate portions of the absorbent system, and which extend into the mesentery.

§ 5. *Morbid Contents of the Peritoneal Cavity.*—In reference to this subject we may direct the reader to the preceding remarks, to the general investigation of the abnormities of serous membranes, and to subsequent paragraphs. At this place we merely allude to the presence of gas (meteorismus abdominalis) and of serous fluid (ascites) in the peritoneal cavity. The former occurs in rare cases as a joint product of the inflammatory process, or as the result of decomposition affecting an exudation of low vitality, and in extremely rare cases as the product of a deranged secretion; but it is more frequently a mere extravasation of intestinal gas, resulting from rupture, gangrene, ulceration, or softening of the stomach or intestine.

Extensive accumulation of serous fluid gives rise to ascites. It is very often the result of an hydropic cachexia, dependent upon a variety of causes, and is then commonly associated with other dropsies. In the first instance, the predominant hydropic symptom is mostly the consequence of granular liver, heart-affections, frequently of Bright's disease of the kidneys; it accompanies carcinomatous formations on the peritoneum, &c.

SECT. IV.—ABNORMITIES OF THE STOMACH.

§ 1. Original arrest of development of the stomach involving at the same time a large portion of the intestinal canal is found in very imperfect monstrosities, and more particularly in acephalous fœtuses,—the stomach is occasionally absent in individuals otherwise normally built and provided with a well-developed intestinal tube, or it may only be indicated by a small saccular dilatation of the œsophagus.

§ 2. *Deviations of Size.*—Congenital malformations belonging to this class, are either unusual enlargement, or unusual diminution of size; the latter peculiarly affecting the female sex.

Either of these conditions, but chiefly the abnormal increase in size, occur likewise as acquired diseases.

Dilatation of the stomach is either spontaneous, or it is caused by stenosis. The former variety presents a uniform increase of size, and sometimes acquires such a surprising extent, as to fill the entire abdominal cavity. Repeated repletion, in consequence of a morbid appetite, may give rise to this development, or it may occur as a result of paralysis from concussion, traction, or dislocation produced by large scrotal herniæ, and it kills slowly with vomiting, with or without gangrene of the mucous membrane, under symptoms of complete paralysis.

Dilatation from stenosis varies according to the seat of the latter. In common stenosis of the pylorus, it is mainly developed at the splenic portion; it equally reaches an enormous degree, and proves at last fatal by paralysis. When stenosis occurs at a different spot, more or less considerable saccular dilatations take place in other sections of the stomach, and in different directions.

A diminution of the stomach is sometimes produced as a permanent condition in consequence of an insufficient supply of nutriment; in other cases it is the consequence of textural disease, especially that produced by cicatrization of extensive ulcers. Contractions or stenoses are the result of hypertrophy of the gastric membranes, of carcinoma, particularly when occurring at the pylorus, and of cicatrization after ulcerative destruction of the tissue at this and at other points.

In reference to the thickness of the parietes of the stomach, we may observe, that extreme thickness, not connected with degeneration of the tissues, is the immediate consequence of the hypertrophy of one, or more commonly of both of the internal coats. The pyloric region is chiefly liable to the affection, which is sometimes limited to the annular portion; it is developed to a greater extent when resulting from stenosis of the pylorus, accompanied by the above-described dilatation, and it then affects mainly the muscular fibres. Hypertrophic disease of the pylorus must be carefully distinguished from carcinomatous thickening.

Attenuation of the gastric coats not unfrequently occurs at the fundus in consequence of extreme dilatation of the stomach resulting from stenosis of the pylorus. The thinning which occurs as spontaneous atrophy, or tabes, with or without an accompanying change in the capacity of the stomach, is of greater importance; it is a very rare and tedious disease, but one which we have invariably seen associated with universal tabes, and with atrophy of the lungs (emphysema senile) and the heart.

§ 3. *Deviations of Form.*—Among these we reckon, first of all, those rare congenital malformations of the human stomach, in which an annular contraction divides it into a cardiac and pyloric stomach, or in which two or three such contractions form three or four sacculated divisions, and thus cause a resemblance to the stomach of ruminants. We distinguish these from the contractions produced during the agony of death, by the fact that the latter may be removed by inflating the stomach.

Similar and various other malformations are observed as acquired

conditions; they have their origin mainly in loss of substance and in cicatrization of the so-called perforating gastric ulcer, and we shall recur to the subject when we discuss the latter.

§ 4. *Deviations of Position.* *Congenital.*—Position of the stomach external to the abdominal cavity in eventration, and in umbilical hernia; in the left side of the thorax, the diaphragm being wholly or partially absent on this side; vertical (fœtal) position, with the pylorus downwards; the position of the fundus, in the right hypochondrium, corresponding to the reversion of the formative type in lateral translocation. *Acquired.*—Protrusion of the stomach, externally, in consequence of extensive penetrating wounds, or into the thorax, after injuries to or rupture of the diaphragm; the position of the stomach in large hernial sacs, especially of umbilical and scrotal hernia; the dislocation of the stomach from its natural position by enlargement of the organs in its vicinity, by morbid products, by effusion into the peritoneal cavity, by traction of the omentum and transverse colon; lastly, the spontaneous sinking of the entire stomach into a lower abdominal region from increase of volume or weight, as in the case of a scirrhous pylorus.

§ 5. *Solutions of Continuity.*—We merely allude here to those rare occurrences of wounds of the stomach produced by penetrating instruments, and by firearms, occasionally healing up with a fistulous opening, and to those circumscribed separations of the membranes of the stomach from one another, accompanied by extravasation of blood, which occasionally result from concussion.

§ 6. *Diseases of the Tissues.*—As we have already treated of the diseases of the peritoneum, we shall now discuss those of the gastric mucous and submucous tissues, and the consecutive affections of the muscular coat of the stomach.

1. *Inflammation.* *a. Catarrh of the gastric mucous membrane (gastritis mucosa).*—The opportunity of observing the first stages of the genuine acute catarrh of the gastric mucous membrane, of the gastric-saburral, gastric-bilious, and allied conditions, in the dead subject, is rarely, if ever, offered; we see the blennorrhagic stage, chronic catarrh, and the occasional acute exacerbations of the latter, more frequently.

The latter is developed from repeated active hyperæmia, or from lasting mechanical hyperæmia, and the gastric catarrhs observed in gourmands, and especially in drunkards, and accompanied by ulceration, and by the formation of morbid products in the stomach in chronic heart disease, or in pulmonary phthisis, are particularly remarkable. The latter are generally complicated with catarrh of the entire intestinal tract, and with bronchial catarrh.

The anatomical signs of this condition are, a dark, reddish-brown, or slate-gray, or even blackish-blue discoloration of the mucous membrane, copious secretion of a stone-colored, occasionally glassy pituita, thickening, increased condensation and induration, i. e., hypertrophy of the mucous membrane, which presents itself in various degrees:

a. In the lowest degree, the mucous membrane shows simply an increase of thickness and hardness in its tissue;

β. In a higher degree, it presents, in addition to its increased thickness, an uneven, racemose, or warty surface, a *surface mamellonée;*

γ. In a still more advanced degree, it forms prolongations in the shape of permanent, firm folds, or of polypus.

The submucous cellular tissue, and the muscular coat, also participate in this hypertrophy in various degrees—the entire parietes of the stomach presenting unusual thickness, firmness, and hardness.

The pyloric portion is the chief seat of chronic catarrh, and it is there that hypertrophy of the mucous and other membranes is most prominent.

b. Croupy inflammation.—This form never occurs as a primary and substantive affection except in the shape of delicate flocculent exudations in the aphthous process of children, but always, and even that rarely, as a sequela or degeneration of exanthematic processes, in variola, in typhus, in the absorption of pus into the circulation, and particularly in puerperal inflammation of the uterine veins. The false membrane which sometimes invests the entire stomach, presents a very regular areolar surface.

The operation of tartar emetic upon the gastric mucous membrane may produce a similar process, it is however commonly limited to a few streaks.

c. Inflammation of the submucous cellular tissue.—Idiopathic inflammation of the submucous cellular tissue of the stomach, resembling pseudo-erysipelas, and passing on to suppuration, is a very rare phenomenon; it not unfrequently occurs as a secondary process, analogous to the metastases of specific, acute dyscrasiæ. The parietes of the stomach appear thickened; the stratum of submucous tissue is distended with pus; it is soft and friable; the superincumbent mucous membrane is reddened, and, at intervals, tense. After a time it gives way at these points, and, by numerous irregular cribriform openings, the pus exudes into the cavity of the stomach.

d. The process, which is caused by the operation of caustic substances on the membranes of the upper part of the alimentary canal.

We limit ourselves to a statement of the *modus operandi* of the more common substances which are taken accidentally, or are administered as poisons, such as the mineral acids, and especially the sulphuric acid of the shops, and white arsenic, and give the results afforded by numerous post-mortem examinations.

The effect of the mineral acids generally extends over the cavity of the mouth and fauces, the gullet, the œsophagus, the stomach, and occasionally beyond the latter; sometimes it is limited to the former, so that scarcely a trace is visible in the stomach.

In reference to the intensity of the effect which may cause superficial or deep mortification of the tissues with greater or less rapidity, we distinguish several degrees. The effect is influenced by the quantity and the strength of the liquid, and the duration of the period during which it remained in contact with the parts alluded to. We generally find the effect to be less intense in the cavity of the mouth and fauces,

more marked in the œsophagus, and, provided an appreciable amount
of acid reached the stomach, most powerful at this point. In the lowest
degree the effect is limited to destruction of the epithelium. The mucus
of the mouth and the fauces contains flocculent coagula; the epithelium
is converted into a thick, grayish-white, rugose layer; it peels off here
and there, and the subjacent mucous membrane is pale.

In an advanced degree, the superficial layers of the mucous membrane
of the fauces and œsophagus, under the destroyed epithelium, are found
corrugated, of a dirty, whitish, leaden hue, and the capillary network
blackened by its carbonified contents. The lower strata of the mucous
membrane, and the submucous cellular tissue, present serous infiltration.
In the follicles at the root of the tongue, the mucous secretion is coagu-
lated into dirty white masses.

In a still higher degree, the entire mucous membrane is destroyed, and
converted into a dirty gray mass, which is traversed by black vessels;
the submucous cellular tissue is infiltrated, and partially ecchymosed;
the muscular coat of the œsophagus itself is shrivelled, pale, ashy.

In the highest degree, the mucous membrane of the œsophagus, together
with the submucous cellular tissue, is converted into a soft, black mass,
which is distended by a sanguinolent fluid, and is easily detached from
the muscular coat. The latter is itself either destroyed in the same man-
ner, or is perfectly colorless, friable, and presents an ashy, gelatinous
appearance.

The mucous membrane of the stomach almost invariably suffers the
changes of the third degree, though in varying extent and thickness. It
is either affected in single folds, or streaks which pass from the cardiac
orifice to the lesser curvature, and from the large curvature to the pylo-
rus; or over a large extent; or we find the entire surface converted into
a black carbonaceous mass, of several lines in thickness, distended by
sanguinolent fluid, and consequently presenting a tumefied appearance.
The muscular coat is altered in the manner peculiar to the third degree,
and we therefore often find the parietes of the stomach perforated.

The acid affects the neighboring organs through the membranes, and
thus either coagulates or tans the contained fluids, fuses the tissues into
a gelatinous mass, or carbonifies them; the discoloration produced is
always very marked. In many cases, not only the blood of the neigh-
boring bloodvessels, but also of the larger trunks, and even of the
aorta, is changed into pultaceous, pitchy, greasy, black cylinders. Beyond
the stomach, and especially in the duodenum, and at the commencement
of the jejunum, the effect of the lowest degree is exhibited in coagulation
of the intestinal mucus, and of the chyle, in corrugation and opacity of
the epithelium, in the tanned state of the mucous membrane, and the
dark injection of its vessels. The consequences and results are modified
according to the intensity of the operating influence.

The highest degrees, in which, generally, a very extensive surface is
involved, produce a rapidly fatal termination. The lowest degrees are
followed by exudative inflammation; the mortified epithelium sloughs,
and being replaced by a new formation as soon as the reaction has abated,
recovery ensues.

In all the higher degrees we have reactive inflammation in the healthy

tissue, which effects the rejection of the superincumbent mortified tissue by passing into suppuration. As the inflammatory and suppurating processes diminish, the tissues contract, cicatrices form, and a cure results; or suppuration is protracted, causing a late recovery, or ending fatally in œsophageal phthisis.

According to the depth to which the tissues are destroyed, the loss of substance is repaired under a formation of strictures, that vary in size and consistency.

If the mortification be limited by the submucous cellular tissue, we shall find the latter condensed over the pale, ashy, muscular coat, which now resembles the elastic tissue, into a serous, or fibro-serous, tissue, replacing the mucous membrane to a considerable extent. This tissue forms, at some places, projecting ridges, or valvular, and even annular, duplications towards the œsophagus; and we thus have a peculiar membranous stricture of the latter produced, not unlike the strictures found in dysentery.

If the muscular coat itself is involved, it is partially or entirely destroyed, and the walls of the œsophagus are converted into a fibro-cellular firm tissue, which contracts, and thus produces the most important and most resisting strictures.

These strictures are formed chiefly, though not exclusively, at the lower section of the pharynx, posterior to the cricoid cartilage, and in the vicinity of the cardiac portion. We also not unfrequently see, besides these strictures, solitary insulated remains of the mucous membrane on the inner surface of the œsophagus, in consequence of the contraction of the new tissues. They have a shrivelled appearance, and are in part detached, or form transverse bands.

A torpid suppurative process is commonly the result of a more profound injury, and is seen in the shape of abscesses and sinuses of the muscular coat, and of the condensed cellular sheath of the œsophagus. When it ceases, it invariably leaves very considerable contraction of the tissues and strictures of the kind last described; ulcerative perforation of adjoining passages (trachea, bronchi) may follow; death frequently ensues from phthisis, or by exhaustion from dysphagia.

The same occurrences may, though less frequently, be observed in the membranes of the stomach.

The operation of arsenic is limited to the mucous membrane of the stomach, but it frequently produces no local effect; and this is particularly the case where the symptoms of poisoning and death follow rapidly after the introduction of small quantities. When present, it is an exudative inflammatory process, accompanied by softening and sloughing. At one or more points, to which a white pulverulent substance (arsenic) happens to attach itself to a larger amount, the mucous membrane appears plicated and tumefied, reddened, invested by a detached epithelium, and a tawny exudation; its tissue is softened, pultaceous; and at the spot where the white grains of arsenic are attached, it is converted into a yellowish or greenish-brown slough. Between these solitary foci, from which reddened folds of the mucous membrane proceed, the inner surface of the stomach presents at many parts a perfectly normal structure.

2. *Ulcerative processes.*—The ulcerative loss of substance which results

from one or the other of the processes we have hitherto considered, requires no separate examination, as it presents nothing characteristic. There are other ulcerative affections of the stomach which appear of more importance. Those connected with tubercular and cancerous affections we shall examine under the head of tubercle and carcinoma. At present we consider the following :

a. The perforating ulcer of the stomach.—There is one kind of ulcer that occurs in the stomach, which, both on account of its frequency, and on account of the extreme pain it causes, as well as on account of the numerous and enigmatical symptoms that accompany it, deserves every attention,—an ulcer, termed by Cruveilhier the simple chronic ulcer of the stomach, and which we would call the perforating gastric ulcer, from its prevailing tendency to perforate the parietes of the stomach.

In a well-defined case there is, in the region of the pylorus, a circular orifice of from three to six lines in diameter, with a sharp peritoneal edge, as if a round piece of the gastric parietes had been punched out. When viewed from within, the loss of substance on the internal membranes of the stomach, and especially on the mucous layer, appears more considerable, so that the edges of the hole seem bevelled off from within outwards. There is no further morbid appearance beyond a thickening of the parietes in the immediate neighborhood of the ulcer, and a tumefaction of the gastric mucous membrane.

The pyloric half of the stomach is the seat of the ulcer ; it is most frequently found in the middle zone of this portion ; it is oftener seen at the posterior than at the anterior surface, almost always near to, and frequently at, the lesser curvature ; and it occurs, in extremely rare cases only, at the fundus. This affection may also appear beyond the stomach in the upper transverse portion of the duodenum, but it does not occur in the remaining portion of the intestinal canal.

The size of the ulcer varies from that of a sixpence to that of half-a-crown, and even to that of a cheese-plate.

Its shape is commonly circular, but, in exceptional cases, it is from the beginning of an irregular form, though the circular form with which it commences frequently disappears subsequently. Ulcers of great extent approach the elliptical shape ; but, on further extension, this too is lost, and they become irregular in consequence of the formation of sinuses varying in depth. The extension of the ulcer in the transverse diameter of the stomach, so as to form a zonular ulcer, is singular, on account of the deformity of the stomach which follows. The original form of the ulcer is also lost, when two ulcers coalesce so as to form a single one. In these cases we may for a long time be able to point out the boundaries of each, represented by a ridge of cellular tissue, but this, too, will disappear, and they then both have the same common base.

In the majority of cases there is only a single ulcer, but frequently there are two or three, occasionally four or five, and these are then commonly placed above or near to one another at the posterior surface of the stomach, or at the lesser curvature. It is very rarely the case that one occurs at the posterior, and the other at the anterior surface of the stomach, or that two ulcers are formed opposite to one another in the duodenum.

It has not been clearly ascertained in what shape the malady takes its origin, and in what manner the further development is effected. It is probable that it commences with an acute, circumscribed, red softening (hemorrhagic erosion), or with a circumscribed sloughing of the mucous membrane; it is still more probable that the ulcer increases in this manner, the tissues at the base of the ulcer sloughing and exfoliating layer by layer. We have observed this occurrence in a few solitary instances, and we would therefore view the process as offering a valuable analogy to sloughing of the lungs (gangræna pulmonalis); on the other hand, we cannot admit that view to be well grounded, which explains the loss of substance in question solely by the absorptive process; the callosities of the surrounding tissues, and the well-marked reaction at the base, are in themselves sufficiently strong arguments against it.

The ulcer attacks the deeper-seated parts in a peculiar manner, when it presents the perfectly round form. The loss of substance is more extensive in the mucous membrane; if the muscular coat has been attacked and destroyed, we find a smaller ring with sharp edges, and the ulcer thus obtains a peculiar scarped appearance. If, finally, the peritoneum is perforated, this point will occupy the centre of the circle; the serous membrane will be converted into a yellow slough, and it will tear, or be voided.

This process may run an acute course; but it is commonly chronic; occasionally it comes to a standstill, and then again exacerbates in an acute or chronic form. A cure may result at any of the stages, as proved by the various cicatrices frequently observed on the inner surface of the stomach. Even actual perforation of the stomach is frequently rendered innocuous by the adhesion of neighboring organs, and complete cicatrization may follow.

Loss of substance in the mucous membrane alone is repaired by a condensation of the submucous cellular tissue into a fibro-cellular tissue, which causes the edges of the mucous membrane to approach one another, and is finally blended with it and the muscular coat. A radiated, asteroid scar, varying in size, remains.

When the ulcerative process has involved the muscular coat, and has penetrated beyond it, the muscular fibres that edge the ulcer retract beyond the mucous membrane, the subserous cellular tissue and the peritoneum shrivel up, the walls of the stomach forming the bases of the ulcer, and now only consisting of these two layers, are doubled inwards, the divided portions of the mucous membrane are thus brought together, and a union is gradually effected. We then find corded cicatrices, which shorten the stomach in its transverse diameter, or form annular contractions proportionate to the extent of substance destroyed or to their position. The pylorus is particularly liable to a diminution of its calibre.

Perforation and its temporary or permanent cure, demands a more minute exposition.

If it takes place at a portion of the stomach, which, like the greater part of the anterior gastric parietes, but rarely enters into a protective adhesion with neighboring tissues, perforation allows the contents of the stomach to pass freely into the peritoneal cavity, and fatal peritonitis follows.

This result is frequently prevented. Whilst we find the tolerably uniform irritation within, giving rise to hypertrophy of the mucous membrane and to callosity of the base of the ulcer and its circumference, we see at the corresponding points of the peritoneal surface, cellular adhesions, or a more intimate union between the stomach and the reverted omentum, the left hepatic lobe, or the pancreas, produced by repeated, circumscribed, inflammatory attacks. The cellular adhesions which have been effected between the stomach and the omentum, and are sometimes found to unite the former with the left lobe of the liver, are not sufficient to prevent a fatal issue when perforation occurs, for as soon as this event has taken place, the adhesions inflame,—this, and the forcible expulsion of the gastric contents, loosens and tears them, and thus the inflammation spreads to the peritoneum, both by continuity of tissue, and by direct contact. The perforation will be rendered more permanently innocuous by intimate adhesion, viz., by the agglutination of a fibro-cartilaginous exudation; as this tissue offers to the contents of the stomach, both from its density and its thickness, a sufficiently firm resistance. This frequently occurs between the small curvature, or the anterior surface of the stomach, and the concavity of the left lobe of the liver, and very frequently between the posterior gastric surface, the pancreas, and the adjoining lymphatic glands; but very rarely between the posterior surface of the stomach and the spleen, after the latter has been dragged into that position, or between the stomach and the diaphragm (Abercrombie, Chardel). In such cases, after the external membranous layers have been destroyed to an extent proportionate to the loss of substance, the mucous membrane is invariably doubled back over the edge of the perforating ulcer, and impinges upon the pseudo-membranous agglutinating tissue external to the stomach; thus the orifice in the gastric parietes is never filled up by the superimposed tissue in such a manner as to be flush with the inner surface of the stomach, or even to project beyond it into the cavity of the latter.

In favorable but rare cases the pseudo-membranous tissue contracts and draws the edges of the orifices together, so as to produce a firm, callous cicatrix.

In other instances this does not occur; the cavity, though covered in as described, remains, and particularly when adjoining the pylorus, in consequence of the *vis a tergo* of the gastric contents, enlarges into a lateral sinus, which is lined by the false membrane.

Although in the majority of cases a free opening of the stomach is thus prevented, we may even here find exceptions; the soldering tissue may itself gradually be consumed, the adjoining organ is laid bare, and becomes exposed to an extension of the process. Thus we have seen one case in which the adjacent diaphragm, which had formed a plug, was perforated from the stomach, and the base of the adhering lung was attacked.

In the progress of the perforating gastric ulcer a very important occurrence frequently supervenes, viz., hemorrhage, which often kills on the first, but more frequently after repeated attacks. So long as the ulcer has not perforated the walls of the stomach, the loss of blood is inconsiderable, as the process involves only the small vessels of the mem-

branes, which are easily plugged up. But as soon as the ulcer has penetrated through the gastric parietes, it meets with larger vessels in and beyond the pseudo-membranous layer, or with the vascular system of the obturating organ. Thus, the trunks of the splenic, the coronary, the pyloric, the gastro-epiploic, the gastro-duodenal arteries and their branches, and more especially those going to the pancreas, are corroded and opened, and exhausting and fatal hemorrhages ensue.

Bloodvessels are not alone involved in the destructive process, but other canals also, and we instance the pancreatic ducts, which, in the case to which we allude, open upon the base of the ulcer, and by forming pancreatic fistulæ, oppose the complete consolidation of the imperfect cicatrix.

The ulcer not only proves fatal by perforation, with consequent peritonitis and hemorrhage, but also, though rarely, by exhaustion from dyspepsia and harassing cardialgia. It is invariably accompanied by chronic catarrh and blennorrhœa of the gastric mucous membrane; it heals as we have remarked, very frequently, but it as often recurs. The cure of large ulcers is followed by considerable deformities of the stomach, and more especially by shortening of the posterior wall and the lesser curvature, or by annular structures. The disease occurs chiefly at the period of puberty, and very often, particularly in the female sex, as early as the 15th year.

The perforating gastric ulcer is in no way connected with gastritis and cancer, though it is often mistaken for these affections; but it is important to know, though it be for the mere cadaveric diagnosis, that in rare cases it may be complicated with cancer; yet it always retains its peculiar characters so as to be distinguishable in the midst of the cancerous growth and devastation.

(b.) *Hemorrhagic erosion of the gastric mucous membrane.*—Very frequent opportunities are presented to us of observing loss of substance accompanied by bleeding, in the mucous membrane of the stomach. There are round or roundish spots of the size of a pin's head or a pea, or narrow, elongated streaks, at which the mucous membrane appears dark red, lax, soft, bleeding, and presenting a depression in consequence of loss of substance or slight erosion.

Commonly a dirty brown coagulum is attached to the point, and the nature of the derangement only becomes evident after the coagulum has been removed. Sometimes this loss of substance involves the entire thickness of the mucous membrane and the submucous cellular tissue, and produces an appearance of small, round, or striated ulcers.

This process is invariably accompanied by hemorrhage; the gastric mucus, which generally is present in considerable quantity, presents streaks of discolored blood, proportionate to the number of diseased points, or it shows a copious admixture of brown flocculi or debris, or we find an accumulation of fluid in the stomach, resembling coffee-grounds. The entire mucous membrane is found in a condition of recent or inveterate blennorrhœa and catarrh, and in the vicinity of the erosions it is often tumefied so as to form a vallated circumference.

The number of these erosions varies; it not unfrequently happens that the stomach, with the exception of the fundus, is closely studded with

them, and is marked with red or brown spots, according to the color of the adherent coagula.

They occur at every period of life—they are seen even in the infant, and they are found chiefly at the pyloric portion, i. e. in that part which is the chief seat of the catarrhal process. The follicles, or the glandular apparatus of the gastric mucous membrane (Cruveilhier's gastritis folliculosa), appear to be their occasional nidus.

This inflammation and erosion undoubtedly occur sometimes as an idiopathic affection. They are more commonly developed consequent, or attendant upon the most diverse, acute, and chronic diseases, so that no definite conclusion as to the real nature of the process, and as to its connection with other affections, has yet been arrived at. An acquaintance with the fact is of considerable importance, though it only serves to assure us that the disease is idiopathic, and in no way allied to the erosion produced by caustic substances.

3. *Softening of the Stomach.*—We must distinguish *two* primary forms of softening, which present essential differences in numerous points; both, however, are to be carefully distinguished from cadaveric softening, the self-digestion of the stomach.

The one, a disease of infant life, is called *gelatinous softening.* It appears to be a metamorphosis—a softening—of the mucous membrane of the fundus, which extends to the muscular coat and the peritoneum, converting them and the intervening interstitial cellular tissue, into a grayish or grayish-red transparent jelly, with a yellowish tinge, through which single dark-brown streaks, the broken-down bloodvessels, are observed to pass. Inasmuch as the softened inner strata occasionally become detached, the fundus of the stomach may be found to consist of nothing else but thin, gauze-like, friable portions of the peritoneum.

The softened portion of the stomach tears at the slightest touch; it dissolves between the fingers, and perhaps in rare cases these rents occur during life, but probably oftener after death, giving rise to effusion of the gastric contents into the abdominal cavity.

The process is not, however, limited to the stomach, but frequently extends to the neighboring tissues, and chiefly to muscular organs, and especially to the diaphragm. Here, too, perforation is the final result, and with it there is effusion of the gastric contents into the left pleura.

Gelatinous softening of the stomach commonly runs a subacute course: general anæmia, which is particularly apparent throughout the intestinal canal, and general collapse and wasting, which are chiefly evident in the muscular tissue, are constant accompaniments of this disease. It is frequently founded upon a demonstrable affection of the brain, principally hypertrophy, or hydrocephalus: and this fact renders it probable that there is a similar causative nexus in those cases also, in which no visible anomalies have been hitherto detected in the infantine brain. Perhaps the proximate cause may be looked for in diseased innervation of the stomach, owing to a morbid condition of the vagus, and to extreme acidification of the gastric juice.

Nevertheless, the *quæstio vexato* as to the origin of the affection in irritation or inflammation remains. If we consider, in addition to the above remarks, the uniform localization of the disease, that in none of its

stages it presents, either at the point of the softening or in its vicinity, hyperæmia, injection, or reddening, and that we are still less able to demonstrate upon the inner surface of the stomach, or in the tissue of its coats, the products of inflammation, we are constrained to infer the non-inflammatory nature of the affection. This conclusion gives a key to the various kinds of softening that occur at advanced periods of life under similar circumstances, viz., in cerebral affections.

A second form, in which softening of the stomach takes place, is distinguished by an absence of pallor in the softened tissues, or rather by their color. The parietes of the stomach are converted into a more or less saturated dark-brown, or blackish pulp.

It occurs under two different circumstances, though in both the process has an acute character, and in both the color of the softening tissues is produced by an alteration of the blood contained in them, by an acid. They differ essentially in their genetic relations.

In the first instance, it occurs, both in children and adults as a sequela of acute affections of the brain and its membranes, and more especially of tubercular meningitis at the base of the brain. It is the same process as gelatinous softening of the stomach, and the theory to which we have alluded is the more applicable the more fully the affection at the base of the cerebrum is developed. But the development takes place with the greater rapidity, the less the acute disease of the brain has induced that degree of anæmia which commonly prevails in gelatinous softening ; and, the tissue being still more or less injected, the superabundant acid acting upon the contained blood, produces the characteristic discoloration. The solitary fuliginous streaks above alluded to, as occurring in the pale, jelly-like membranes of the stomach, are analogous to this condition. Besides, it not unfrequently presents itself in those cases of pulmonary paralysis which are probably caused by a reflex action of the œsophageal and gastric branches of the vagus.

In the second instance, the softening occurs, unconnected with the etiological relations we have hitherto discussed, under totally different circumstances. We now speak of it as a sequela of certain cachexiæ, which were either originally acute, or became so under the influence of certain circumstances, viz., the exanthematic, the croupy, the typhoid in the widest senses, pyæmia, acute tuberculosis, acute cancer—it is then to be viewed as a fatal degeneration of these diseases. This form is developed from a congestion in the capillary network of the gastric membranes, and particularly of the mucous membrane of the fundus, which is generally accompanied by a more or less congested state of the spleen. It probably arises from the state of the blood itself which accumulates with an excess of acid in the vascular system of the fundus, and of the spleen. This too is the cause of the generally rapid course of the affection, the dark color of the softened tissues and their frequent perforation. It commences with a dark brown or black discoloration of the mucous membrane at the fundus, which is soon converted into a black pulp that may easily be detached. If it be removed or if it separate spontaneously, a pale, bluish-white, submucous cellular tissue is exhibited, in which vessels ramify whose coats are disorganized, and which contain a black (carbonified) granular coagulum. The subjacent muscular coat is pale and

thin, the peritoneum dull, and of a dirty gray color. The process extends from the mucous membrane to the subjacent tissues; they are converted into a black, grumous pulp, and thus more or less extensive perforations result, which are bounded by a furred margin. Here, too, the diaphragm is frequently involved, and softening and perforation of this organ follow. The stomach is found to contain large quantities of fluid resembling coffee-grounds or ink, which is often vomited during life—there is an admixture of more or less of the softened tissues, and of their fat, which floats in the mass in the shape of oil-globules. This fluid originates in the sanguineous effusion which takes place at the commencement of the disease; the latter proceeding from the vascular system, and first affecting the coats of the vessels. On the occurrence of perforation, the fluid is extravasated into the peritoneum, and into the left pleura; and it here gives rise to a similar process in the serous membrane, accompanied by the evolution of gas.

In rare cases this process only takes place at solitary, circumscribed spots, and does not then appear to run its course so rapidly. The mucous membrane disappears at these points, with the exception of a very thin, gauze-like, discolored layer, to the edges of which are attached a few jagged remains of the former.

The fundus is the seat of all the softening processes of the stomach—from here they extend to the large curvature of the stomach, in which respect they differ from the gastric diseases that we have already considered, or that we are about to examine, such as catarrhs, follicular erosions, the perforating ulcer, hypertrophy, cancer. It is there too that we find the perforations; and it is only in very rare cases that we see softening at the large curvature precede the development of softening at the fundus.

Softening, and especially the last-named form, occurs in company with softening of the fundus at the œsophagus. The lower third of this tube is liable to be attacked, and the side which is directed towards the left side of the thorax is chiefly so, as the perforations almost invariably occur here, producing effusions into the left half of the thorax, after the cellular sheath of the œsophagus and the mediastinum have been absorbed.

The softening is never distinctly circumscribed, but is shaded off gradually into the surrounding tissues. It is a fact of considerable importance, that softening may take place after death from the operation of cadaveric, chemical changes, which closely resemble the processes we have just described. It is not always easy to decide between this self-digestion and morbid softening; nay, it is a matter of impossibility for the conscientious pathologist, unless he take the previous disease and the mode of death into consideration.

The following circumstances may, however, serve to characterize cadaveric softening:

a. The absence of all symptoms during life which indicated softening, or the morbid processes that gave rise to it.

b. Sudden death, from natural or other causes, during the digestive act, whilst the stomach is filled with chyme, without previous illness.

c. Limitation of the softening to the mucous membranes, and especially to the projecting folds, so as to form streaks.

d. And at the same time its extension beyond the ordinary boundaries of morbid softening—its development being most remarkable at those points at which there is a stagnation of the greatest quantity of the gastric contents.

4. *Heterologous formation. a. Anomalous occurrence of fatty tissue, of lipomatous tumors between the gastric membranes, and chiefly in the submucous cellular tissue.*—These growths project into the cavity of the stomach, being either attached by a neck or sessile, and being invested by mucous membrane. Occasionally they pass through the fasciculi of the muscular coat, and present similar tumors under the peritoneum.

b. Anomalous fibrous and fibro-cartilaginous tissue—appears chiefly in the vicinity of the cardiac orifice and the lesser curvature, and assumes the shape of flattish, roundish, whitish, tough concretions in the cellular tissue, which are movable and of the size of a lentil or pea.

c. Erectile tissue—is either developed at the free end of polypi, or the mucous membrane degenerates into it on a larger surface, upon which the erectile tumor is attached by a broad base, or only by a very short neck or stalk. It is the common seat of encephaloid infiltration.

d. Tubercle and tubercular ulceration of the stomach—are a very rare occurrence, and primary tuberculosis of the stomach is almost unknown. It commonly occurs as a result of intestinal tuberculosis which has advanced to an extreme degree; the tubercular ulcers extend from the ileum through the jejunum and duodenum into the stomach. They here are generally limited to the pyloric portion, but sometimes extend to the fundus. The remarks we shall have to make on intestinal tubercle apply also to the original seat of tubercle and the character of the tubercular ulcer in the stomach. In the former the mesenteric, in the latter, the lymphatic glands are the seat of tubercular affections, and we may use this as an aid to the diagnosis of a gastric ulcer, the characters of which may not otherwise be sufficiently defined.

e. Carcinoma.—Carcinomatous diseases affect the stomach very frequently, and carcinoma of the stomach is moreover the most common carcinomatous disease of the digestive tube. It must be carefully distinguished, as we shall have occasion to explain more fully, from mere hypertrophy, the non-malignant thickening of the gastric membranes, with which it is sometimes confounded.

We find all the different species of carcinoma, the fibrous, the medullary, the areolar, occurring at this point; though in various degrees of frequency. Fibrous cancer is the most common, the pure genuine medullary cancer less so, and the areolar variety is very rare. Often enough we find the first two, and sometimes all three, occurring in primary, but more particularly in secondary, combination.

a. Fibrous cancer appears as thickening of the submucous cellular stratum, which congeals into a resisting, whitish, fibro-lardaceous mass, and unites intimately with the mucous and the muscular coats. The latter becomes pale, and gradually undergoes a change which is characteristic of all kinds of cancer. It increases in thickness, and at the same time degenerates into a pale yellowish-red areolar tissue, the interstices

of which are filled up by a slightly translucent and apparently crystalline substance. The increase of the muscular coat is uniform, whereas that of the submucous cellular tissue is commonly irregular, and we thus see lobulated protuberances formed on the inner surface of the stomach.

Fibrous cancer is the one most easily and most frequently confounded with hypertrophy of the gastric coats. The distinguishing signs are, the preponderating increase of substance in the submucous cellular tissue and its want of uniformity, the accompanying cartilaginous hardness and closeness of texture, the fusion with the mucous and muscular coats, and particularly the alteration in the muscular tissue just described (John Müller).

The mucous membrane itself undergoes further peculiar changes. It sometimes degenerates into an areolar cancerous tissue, which discharges large quantities of a gelatinous mucous fluid; or it is converted into erectile tissue, as a fungoid growth, which becomes the seat of encephaloid infiltration, suppurates, and partially exposes the submucous scirrhous cellular tissue; or lastly, it most frequently becomes the seat of a sloe-black softening with hemorrhage, and we thus find the scirrhous submucous cellular tissue invested by a thin, gauze-like black remnant of the mucous membrane, or it is quite denuded, merely retaining here and there a few solitary black convolutions of vessels at its surface.

The scirrhus, too, at once becomes the seat of various metamorphoses. It may, after it has been denuded of its mucous membrane, become gangrenous in large patches or in round circumscribed spots, the tissue exfoliating by layers, so as to give rise to deep, smooth excavations in the crude cancer; or it may become developed into a more highly-organized carcinomatous formation, such as medullary sarcoma, accompanied by bleeding fungoid tissue; this is soon destroyed by a suppurative process, leaving an ulcer which is surrounded by an elevated lardaceous margin.

β. Medullary cancer of the stomach occurs independently of its secondary appearance in the metamorphosis of fibrous cancer, primarily in various forms:

αα. In the shape of soft and even liquid, milky, medullary, infiltration of the erectile tissue, into which the mucous membrane has degenerated, the other coats remaining normal (vide p. 43).

ββ. As a lardaceous, medullary degeneration of the submucous cellular stratum to a greater extent.

γγ. As knotted tumors between the gastric coats, and here too chiefly in the submucous cellular tissue.

Medullary carcinoma is distinguished in this form also, by its extensive growth, and by its rapid metamorphosis, accompanied by vascular fungoid degeneration.

γ. Areolar cancer presents, in the degeneration of the mucous and submucous cellular tissues, the characters generally peculiar to this form.

We often, as has been remarked, find these varieties of cancer occurring simultaneously; in the stage of metamorphosis in which more particularly a consecutive complication is seen, the fibrous cancer at the base gives rise to an areolar cancer, from which, in its turn, medullary cancer shoots up in the shape of a peripheral erectile growth.

The stomach is either the primary or the secondary seat of disease. In the former, the most usual case, the cancerous degeneration extends from the stomach to other organs, attacking the lymphatic glands which are contiguous to the head of the pancreas and the biliary ducts, the pancreas itself, the glands of the lumbar plexus, and, finally the fibrous investments of the vertebral column, the liver, the transverse colon, the omentum, &c. In the latter case, which is of much less frequent occurrence, the stomach is secondarily attacked, the morbid affection commencing in neighboring tissues, and particularly in the conglomerations of lymphatic glands, from which it extends to circumscribed portions of its posterior parietes. In this variety, the cancerous ulcer may proceed beyond the stomach, establishing communications with the transverse colon or with other portions of the intestine, and it may even force its way outwards after a previous union of the stomach and the abdominal parietes has been effected, and the latter have been destroyed.

The pylorus, indifferently at all parts of its circumference, is known to be the chief seat of primary fibrous and areolar cancer of the stomach. From this point the degeneration extends chiefly along the lesser curvature over the pyloric half of the stomach ; in many, though rarer cases, it affects the entire stomach, attacking the fundus last, which however generally remains partially free. The parietes of the stomach may attain an inch in thickness, being rigid and generally tuberculated on their inner surface ; the cavity of the stomach will at the same time be diminished in size. The cardiac orifice of the stomach is rarely the seat of cancerous degeneration, and it is singular that cancer of the pylorus is accurately bounded by the pyloric ring, and never extends to the duodenum ; whereas when cancer occurs at the cardia, excepting, of course, those cases in which it descends from the œsophagus, it invariably involves a portion of the latter.

The scirrhous pylorus is commonly bound down by the degeneration of the tissues that lie behind it ; but exceptions occur which require the more to be known, as they materially affect the diagnosis. The degenerated pylorus may remain unattached, and will then, owing to its increase in weight, descend to a lower region of the abdomen, even down to the symphysis pubis, causing a hard, very movable tumor, which easily gives rise to mistakes.

In proportion as the parietes increase in size and thickness, the stenoses of the pyloric channel will be more or less considerable ; nodose protuberances, uneven contraction of the tissues, and corrugation of the parietes, give rise to inflections presenting a more or less acute angle. The greater the stenoses, and the more the cancerous degeneration is limited to the pylorus, the more considerable will be the dilatation of the stomach, which sometimes reaches an enormous size, and presents a more or less hypertrophied state of its muscular coat.

It is very frequently found to contain the well-known chocolate-colored fluid resembling coffee-grounds, the origin of which is apparent from the various conditions of the inner gastric surface we have above examined.

Cancer of the stomach in most instances is uncomplicated, but it is also found coexistent with cancer of the liver, of the lumbar glands, of

the intestine, and especially of the rectum, of the uterus, the peritonium, the ovary, &c.

§ 7. *Anomalous Contents of the Stomach.*—Among the anomalous contents of the stomach, we class, first, the secretions of the mucous membrane, which, both as regards quantity and quality, in various ways depart from their healthy condition ; secondly, the products of different morbid processes which occur either in the stomach or external to it ; thirdly, foreign bodies which have been introduced into it from without in a variety of ways.

To the first belong large collections of gas, of very acid gastric juice, as we find occurring in chronic gastritis and many other morbid metamorphoses of the gastric membranes, the absence, but more frequently the excess, of a white, milky, opaque, and purulent, or of a transparent, viscid, gelatinous, glassy mucus, such as we find in chronic catarrhs, or in a blennorrhoic condition of the gastric mucous membrane.

To the second belong the products of exudative processes, and of ulcers in the stomach itself, such as plastic, viscid mucous, fibrinous exudation, pus, ichor. The latter may also be introduced from without, from abscesses of contiguous organs, the liver, the spleen, the pancreas, the lymphatic glands, from ulcers of the œsophagus, and even from abscesses of the vertebræ.

Blood occurs in varying quantities ; when found to a large amount either in a coagulated or fluid condition, it commonly has its source in rupture of varicose veins of the œsophagus or stomach, in rupture of an aneurism communicating with those cavities, or in corrosion of arteries lying at the base of a perforating gastric or duodenal ulcer. Occasionally, too, the capillary bleedings which accompanying follicular inflammation and erosion, degenerate into such exhausting hemorrhages.

Blood may also occur as a reddish-brown, or black pulverulent substance, either mixed up with the contents of the stomach, and especially with the mucous secretion, in the shape of streaks or flocculi, or attached to the mucous membrane, and more especially to the bleeding portions.

Or it may occur as a chocolate-colored, coffee-ground-like or inky matter, and that will be the case under all circumstances that give rise to gastric hemorrhage, if the blood has been retained in the stomach for a certain period, and submitted to the action of the gastric juice. It is evident that this will chiefly be the case in passive hemorrhages. We gather from the preceding observations that the following are the cases in which the contents of the stomach present this appearance, and in which there will be vomiting of black matter during life :

a. In slow hemorrhage from a perforating ulcer of the stomach ;

b. In capillary hemorrhage accompanying hemorrhagic erosion of the gastric mucous membrane and their follicles ;

c. In softening ;

d. In the hemorrhages that accompany cancer of the stomach.

In rare cases we find blood in the stomach without being able to trace a distinct cause of the hemorrhage, either in or out of the organ ; the parietes of the stomach are either found to be in a state of complete anæmia, or occasionally single, red, injected portions of the mucous

membrane are visible, which bleed on the application of slight pressure from below, by which the congestion is increased. There is no doubt that, in such cases, hyperæmiæ of various kinds precede, and blood at once transudes through the vascular coats; the greater the impulse of the blood, the laxer the tissue and the vascular coats, and the thinner the blood itself is, the easier will this be brought about.

The blood which is found in the stomach is not only, as we have remarked, frequently the result of extravasation which has taken place external to the stomach, but it may even have been extravasated external to the œsophagus and intestinal canal. Thus it is often swallowed in large quantities during hemorrhages of the respiratory mucous membrane.

Finally, there may be bile, biliary calculi, fecal matter, and lumbrici, in the stomach.

To the third class belong the most various foreign bodies which have been swallowed accidentally, or in consequence of morbid appetites; in the latter case, chiefly seen in lunatics, they are taken in large quantities, and with evident selection. We may enumerate flints, clay, indigestible vegetables, grass, and straw, waste pieces of clothing; metallic substances, as coins, bullets, iron nails, pins, &c. They give rise to various lesions, to perforation of the stomach, or at least, to irritation and inflammation, with subsequent ulceration of the mucous membrane.

SECT. V.—ABNORMITIES OF THE INTESTINAL CANAL.

§ 1. *Defective and excessive Formation.*—A complete absence of the intestinal canal when an abdominal cavity existed, has probably never been observed. It is frequently defective; at times it is a short tube of uniform calibre, attached to a flat narrow strip of mesentery, or it consists of several detached portions of intestine which are strung together on a very defective fold of the peritoneum.

We must here mention the blind termination of the intestine at different points of its course, there being either a fresh acuminated commencement lower down, or an absence of the remaining portion. The most frequent anomaly is the more or less extensive deficiency of the rectum with a consequent atresia ani. The latter abnormities demand the formation of an artificial anus at the natural situation, or at some other suitable part, if they occur in individuals who are otherwise capable of sustaining life.

Defective formation may occur in the shape of tissue, of irregular communication of the intestinal tube, as in the case of the latter discharging at the navel, into the cavity of the urinary or small sexual organs (cloaca); it then is commonly the result of an arrest of development.

Excess of development, with the exception of the various degrees of biventral monstrosities, is probably nothing but a deceptive appearance; the repetition of some of its segments, and the presence of larger or smaller blind appendices, which open outwards or into the intestine, and more especially the so-called diverticula, are almost invariably to be considered as arrests of formation.

The latter, the congenital diverticula, Meckel's diverticulum verum,

deserve a special consideration. It is a dilatation of the small intestine, representing a hollow appendix, which consists of all the intestinal membranes, and is placed at from eighteen to twenty-four inches from the cæcal valve; although we do not quite assent to Meckel's view, that it is a remnant of the umbilical canal, it evidently has its origin in the development of the intestine in the umbilical vesicle. We accordingly always find it solitary and attached at the above-mentioned spot; it varies in length from five to six inches; it sometimes is wider, sometimes narrower, than the intestine itself; it is frequently contracted at intervals, of a conical or cylindrical shape, and terminating in a round, clubbed, or lobulated expansion. It either projects at right angles from the convex surface of the intestine, hanging unattached in the abdomen, or it passes off at an acute angle from the concave surface of the intestine near the mesenteric insertion, being attached to the latter by a falciform process of the peritoneum. In this case it is often placed parallel to the intestine. Occasionally a ligamentous cord, the remains of the omphalo-meseraic vessels, is found at its free extremity, and as this may, by its adhesion to various points of the peritoneal cavity, give rise to internal hernia (strangulation of the intestine), it receives importance in a pathognomonic point of view.

The following case, in which this appendix was abortive, may be interesting: In the corpse of a young man, the small intestine was found enlarged at the above-mentioned spot, to the extent of several inches, the peritoneum and the adjoining laminæ of the mesentery were white and opaque, studded with tendinous patches, and a tolerably long ligamentous cord, the remains of the bloodvessels, was found depending from a rounded embossed dilatation.

We may finally observe that the entire intestinal canal or portions of it, are found in some individuals inordinately long or short; no fixed rule has, however, been established with regard to the relation among the parts themselves, to the stomach, the organs of mastication, &c.

§ 2. *Abnormities of Size.*—The congenital malformations belonging to this section, are the anomalies which we have described above, when speaking of the length of the intestine, and the true diverticulum.

The acquired malformations, as distinguished from the former, have reference to the calibre of the intestine, and are either dilatations or contractions.

The former occur either as uniform dilatations of the tube, or a lateral extension.

Uniform dilatation is the result of atony, or paralysis, consequent upon concussion, habitual repletion, peritonitis, rheumatism, typhus, dysentery, cholera, overstimulation by injections and purgatives, and the like; or it is the immediate consequence of disease in the nervous centres; or the dilatations may be developed as dilatations of an active character, i. e. with hypertrophy of the muscular coat above a constriction. In accordance with their etiological relations, they occur chiefly in the colon.

The lateral dilatation of the intestine occurs in a form resembling a diverticulum, constituting the false, in contradistinction to the true di-

verticulum; it is a hernia of the intestinal mucous membrane, resulting from the separation of the fibres of the muscular coat. It differs in every one of its characters from the true diverticulum:

1st. False diverticula consist solely of mucous membrane and peritoneum;

2d. They occur at the duodenum, in the entire course of the small and large intestines;

3d. They are found in considerable numbers;

4th. They occur from the size of a pea to that of a walnut, in the shape of round, baggy pouches of the mucous membrane;

5th. They form, more especially in the colon, nipple-shaped appendages, which occasionally are grouped together in bunches; when occurring in the small intestine, they are commonly developed on its concave side, and are therefore placed between the layers of the peritoneum; when in the colon, the fæces are retained by them, and dry up into stony concretions.

Contraction of the calibre of the intestine at a small portion, or in a greater extent, is the effect of the pressure or traction exerted by large morbid growths, by hypertrophied contiguous organs, the impregnated uterus, uterine fibroid tumors, dropsy or cancerous degeneration of the ovary, &c.; it is brought on by incarceration or traction of the intestine in external and internal herniæ, by invagination, by adhesion of the intestine, accompanied by angular inflection at the point of adhesion; by compression of a large portion of intestine into a small space, in consequence of a firm adhesions between the coats and the peritoneum; it is produced by disease of the tissues, and more especially by cancer (cancerous stricture), by cicatrization of tubercular ulcers, by the healing of loss of substance in dysentery, by catarrhal suppuration, by the scar following a gangrenous slough, or by simple hypertrophy. The passage of the intestine is moreover interfered with or entirely obstructed, by tumors which project into the intestinal cavity, and it is variously affected by foreign bodies.

We have lastly to remark, that we find various states of contraction and vacuity of the intestine coexistent with its blind termination, with an artificial anus, or with stricture.

In a different point of view we must here cite the anomalies which occur in the dimensions of the intestinal parietes: they appear in the shape of augmentation or diminution, i. e. thickening or thinning. Thickening is found to accompany or result from textural diseases, under which head this form will be considered; but it also presents itself as simple hypertrophy.

This either affects the mucous membrane and the muscular coat separately, or both simultaneously, together with the intervening cellular tissue. In the first and last cases it is the result of a repeated and habitual state of irritation of the intestinal mucous membrane, which, in accordance with a uniform law, after a certain duration gives rise to hypertrophy of the muscular coat, and an increase in the density and quantity of the intervening cellular layer. The hypertrophy of the mucous membrane is presented to us in a very characteristic shape in polypus of the intestine; this growth is peculiar to the colon, and chiefly

to its terminal portion, being often remarkable for its length, its frequent repetition, and the massive cauliflower-like development of its free extremity. When hypertrophy exclusively or mainly affects the muscular coat, it generally results from excessive innervation accompanying habitual spasm of the intestine, or from extreme excitement of its muscular activity induced by repeated or continual repletion, as we see following a stricture.

Excessive thinning of the intestinal mucous membrane, presenting an appearance which resembles that of serous membranes, occurs chiefly in the colon after the protracted serous diarrhœas which accompany consumptive diseases; the tissues are there found in an anæmic and pallid condition, without exhibiting any conspicuous anomaly in consistency.

Atrophy of the muscular and mucous coats of the intestine is often seen in connection with tabes universalis, though rarely as it appears, dependent upon idiopathic torpor of the abdominal ganglia; it is found coexistent with a wasting of the mesenteric glands in hypochondriac and melancholic affections, or as a signal of certain acute processes, as for instance, of typhus, or as a consequence of slow poisoning by lead. The thinning which, coupled with relaxation and friability of the intestinal membranes, occasionally exists simultaneously with an accumulation of fat in the mesenteries and omentum, is still more remarkable. The excessive production of fecal matter (copropoesis excedens) which frequently accompanies these two conditions, is important in reference to their pathogeny.

The follicular apparatus frequently becomes atrophied at an advanced age, but it may be similarly affected in consequence of acute diseases, such as ileo-typhus. Berres has demonstrated senile atrophy in the intestinal villi.

§ 3. *Deviations of Position.*—The intestinal canal may be irregular in position, either being placed altogether external to the abdominal cavity, or by its relations and its disposition within the cavity being irregular.

In the first class we reckon the following congenital irregularities: protrusion of the intestines, external to the abdomen, from absence of the parietes, or from fissure at or near the median line (eventration, omphalocele, congenital umbilical hernia); congenital inguinal hernia; thoracic hernia from partial or total absence of the diaphragm, the left side of the latter being chiefly liable to this malformation. To the acquired irregularities belong prolapsus of the intestines, resulting from penetrating wounds of the abdomen, wounds or rupture of the diaphragm, and the different forms of ordinary hernia.

In the second class we reckon, as a congenital deviation, the lateral transposition which is likely at the same time to involve secondarily, not only the other abdominal, but also the thoracic viscera; the various changes of position, produced by diffused or circumscribed fluid effusions or accumulations, by hypertrophied viscera, or by morbid growths; the spontaneous descent of the transverse colon into the hypogastric region, of the small intestine into the pelvic cavity; external and so-called internal hernia; invagination and prolapsus ani, the two being identical in

character and causation; the changes of position which the intestine experiences in consequence of the cellular or fibro-cellular adhesions that it forms with the parietes, and that unite the coils to one another.

For external hernia we refer the reader to surgical works; we shall here examine only the relations of internal hernia, invagination, prolapsus ani, and the change of position produced in the intestines by adhesion.

1. *Internal hernia.*[1]—We define internal hernia, in contradistinction to external hernia, as a change of position in the intestine leading to incarceration, which occurs in the abdominal cavity without the formation of a hernial sac, and which is therefore not accessible to the usual mode of examination applicable to external hernia. Certain cases in which the intestine is placed or incarcerated in congenital folds or pouches of the peritoneum, such as we occasionally see in the hypogastric region, are to be viewed as transition forms between internal and external hernia (vid. Peritoneum, p. 24). The former are also termed *incarceratio, strangulatio interna.*

They may be subdivided in the following manner:

a. Incarceration is the result of the simple pressure, which is exerted upon one or more points of the intestinal tube, by a portion of the intestine or by the mesentery, resting upon the former. It is a matter of course that this simple compression of a portion of the intestine, can only be effected in the direction of the resisting posterior walls of the abdomen, and at its lower segment; inasmuch as the occurrence of a similar relation anteriorly is inconceivable, on account of the smoothness and yielding nature of the parts. Experience confirms the fact that the small intestine, from repletion or increase of volume, is particularly prone to occupy abnormal positions; it is very liable to descend, and with its lengthy and frequently hypertrophied mesentery, fall and weigh upon the colon or the rectum, and to compress their walls.

These incarcerations of the intestine commonly occur at an advanced age, at which a descent of the intestines to a lower region of the abdomen and into the pelvic cavity, prolapsus of the pelvic viscera and large herniæ, which may be viewed as analogous conditions, are very frequent.

A long, flabby mesentery predisposes to the complaint; especially when, by traction, it has been converted into a pedicle or cord. Repletion of the intestine above a stricture, accompanied by atony, or the dislocation of the intestine in large herniæ (inguinal and scrotal herniæ), is likely to produce this effect.

b. Incarceration may be the consequence of a rotatory movement, and of this there are three varieties:

a. A portion of intestine may have become twisted upon its own axis, and we then find that even semi-rotation causes such an approximation of its parietes, that they touch and close up the passage. This can probably only occur in the colon, and according to the cases on record, only in the colon ascendens. Accumulations of gas, and unequal filling of different portions of the intestine, appear, as far as we are able to judge from the few cases which have been noticed, to be the cause. Such an

[1] Vide Oestr. Jahrb. x. 4.

occurrence is scarcely conceivable in the small intestine, on account of the uniformity of its calibre, the absence of angular flexures, and its loose position, as every rotation of one portion upon its axis would be counterbalanced by the rotation of the next segment.

β. The mesentery may be the axis, and the intestine will then be rolled up upon the former, i. e., the entire mesentery, or a portion of it, is twisted into a cone, and in proportion to the number of its rotations, more or less of the intestine will be dragged after it. In this case we must take into consideration the traction and the pressure, which the intestine suffers at the acute angle, which the dependent mesenteric cone forms with the base whence its point rises. This variety can scarcely occur anywhere but in the small intestine and its mesentery.

γ. One portion of the intestine, either single or double—a coil—may afford the axis round which another portion with its mesentery is thrown, so as to be throughout in contact with the circumference of the axis, and thus to compress it like a ferrule. This variety is evidently a higher degree of the first in which a portion of intestine is merely compressed from before backwards, and, as it were, flattened down. A coil of small intestine, the sigmoid flexure, or the cæcum, may form the axis.

The last two varieties occur like the first, chiefly at an advanced period of life. In early life a predisposition to the affection may be caused by a congenital malformation of the mesentery, by large herniæ, or by small herniæ when there is adhesion of the intestine.

This predisposition consists, first, as in the incarcerations of the first variety—in a congenital or acquired long, loose, and flabby mesentery, by which a rotation of the intestine round the mesentery or another portion of intestine is rendered possible; and secondly, in an enlargement of the abdominal, and especially of the pelvic, cavity.

c. The incarceration of the intestine may be effected by peculiar structures, which either belong to the normal condition, or are congenital malformations, or are, in part at least, the products of previous morbid processes. We allude to genuine incarcerations of the intestine in various annular spaces or fissures, of which we cite the following:

α. The fissure of Winslow, in which we once found a large portion of small intestine strangulated;

β. An intestinal diverticulum (verum), which is directly or indirectly, by means of an obsolete vascular cord, attached to a certain portion of the peritoneum;

γ. Adhesions of the free end of the cæcum, or of the vermiform process;

δ. Holes or fissures (congenital or acquired) in the mesentery;

ε. Malformations of the omentum, forming rounded or flattened cords and bands which are attached to the peritoneum, or furcated fissures of the omentum;

ζ. Pseudo-membranous formations, as the result of previous exudative processes, in the shape of cellular or ligamentous cords, bands, or plates, which pass from one part of the intestine or the mesentery to another, from the intestine to the abdominal parietes, the omentum or an organ of the abdominal and pelvic cavity, or from one of these to the abdominal parietes, or between the organs themselves.

It is most frequently a portion of the small intestine which is incarcerated in these structures; only the more movable portions of the colon, the cæcum and the sigmoid flexure, are likely to become involved.

These varieties of incarceration are very common, and, as compared with the others, the most frequent.

They occur at every period of life. The female sex is more prone to them than the male, as the omentum, the diverticula that may be present, and pseudo-membranous formations, are not only frequently attached to the internal sexual organs of the female, but the latter are themselves liable to give rise to new growths.

The consequence of internal hernia is a distension of the intestine above the compressed or strangulated portion, peritoneal inflammation, paralysis, and ileus; the incarcerated portion in hernia of the third variety is from the strangulation of its mesenteric vessels peculiarly liable to congestion and gangrene.

This affection, when diagnosed, most imperatively requires an operative proceeding, for the purpose of disentangling and arranging the intestines, and for division of the strangulating structures with or without the knife.

2. *Invagination of the Intestine.*—Invagination or intussusception,[1] incorrectly termed volvulus, consists in the inversion of a portion of intestine into the cavity of the adjoining upper or lower portion.

We frequently find intussusception in the corpses of children and adults, but in the majority of these cases it is produced during the last moments of life, during the death-struggle. It is the result of an unequal irritability of the intestine, and the consequent irregularity of its movements, and it is therefore frequent in diseases characterized by torpor of the cerebro-spinal system, and in the mortal agony proceeding from them; whereas it rarely or never occurs in diseases accompanied by, or ending with, abdominal paralysis such as cholera, typhus, general peritonitis, &c. In this case we find no traces of reaction, the parts are easily restored to their proper relations, the inversions are found occurring simultaneously at several points, though only in the small intestine, and the inversion may take place downwards, and at the same time, but rarely, upwards.

Another form of invagination, which, once formed, presents itself as an idiopathic, dangerous, and often fatal disease of the intestinal tube, is of extreme importance, and will be the subject of the following remarks.

Every intussusception consists of three layers of intestine: of these, reckoning from without inwards, the first and second present their mucous, the second and third their peritoneal, surfaces to one another. The canal of the intussusception or volvulus passes through the latter. In order to facilitate comprehension, and in accordance with fact, we term the external layer of the intestine the sheath of the volvulus, or the intussuscipient portion, the innermost layer the entering tube, the middle one the receding or inverted tube, and the last two together, the intussuscepted portion, or the volvulus properly so-called. It follows that isonomic layers are always opposed to one another; and we shall

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[1] Oestr. Jahrb. xiv. 4.

find this to be the case even when the intussusception is double, and consists of five superimposed layers.

Between the entering and inverted tube we find a portion of mesentery, of corresponding size, and of an arcuate form. It is folded up so as to represent a cone, the apex of which lies at the free termination of the volvulus, with its base in the sheath, and at the entrance to the invagination.

This portion of mesentery is always in a state of tension, which chiefly affects the part belonging to the inverted tube, and has a singular influence upon the form of the volvulus. It is the cause of the following circumstances:

Firstly; that the volvulus does not lie parallel to its sheath but always offers a greater curvature than the latter, the inverted tube being compressed in its concavity into tense transverse folds.

Secondly; that the orifice of the volvulus does not lie in the axis or in the centre of the sheath, but external to it; and that, following the traction exerted upon it by the mesenteric fold that belongs to the inverted intestine, it is directed towards the mesenteric wall of the sheath; that it is not circular, but represents a fissure. This affords a diagnostic sign for the examination of intussusceptions of the rectum, which are within the reach of manual exploration.

Intussusceptions occur with equal frequency in the colon and small intestine; but several cases which have been described as occurring in the former are remarkable on account of the magnitude they attained. In these cases the sheath contains a very long portion of the colon and ileum; both may be inverted two or three times, and the intussuscepted part advances to the vicinity of the anus.

An inversion of the intestine from above downwards is the most usual occurrence. Post-mortem examinations have, with very rare exceptions, proved this to be the case; and it is but fair to assume the same in those cases in which, after urgent symptoms of danger, larger or smaller portions of intestine were discharged, and the patients recovered.

We naturally ask how the intussusception is brought about, and how its enlargement is effected?

The cause is to be found either in the contraction and movability of a piece of the intestine, on which account it passes into the adjoining and more capacious tube; or in the extreme expansion or relaxation of a segment of intestine, which gives rise to an inversion of the adjoining narrower and more innervated portion. In every case the volvulus is formed at the expense of the external layer of the intestine or sheath. For we find that the entering portion, as it enters and advances (increase of the volvulus), is not reverted at its free termination to form the receding tube, but that the latter is formed by the inversion of the sheath at the entrance of the volvulus.[1]

Whether the intussusception takes place in one way or the other, the volvulus is not immediately subjected, as is commonly thought, to an-

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[1] [In other words, the volvulus increases at the expense of the inferior portion of the intestine.—Ed.]

nular strangulation. In the first instance, the mesentery of the volvulus and its vessels suffer tension and compression at their entrance into the sheath; and, in consequence, we have in the volvulus an obstruction to the circulation, with swelling and intense redness, in short, violent inflammation, which gives rise to sero-sanguineous infiltration of the tissues, plastic effusion on the contiguous serous surfaces of the entering and receding tube, and upon the mucous membrane of the latter. The inverted portion is invariably the one that suffers most; the inflammation of the entering tube is less considerable, and it is characteristic, that even when the inflammation of the volvulus runs high, its mucous membrane remains pale; the sheath of the volvulus also is but slightly affected in small intussusceptions, with the exception of the peritonitis at the point where it enters. In large invaginations of the intestine, however, the sheath is more deeply involved in the inflammatory affection on account of the tension of the mesentery and the strangulation of the vessels.

In consequence of the tumefaction that results from the inflammation of the volvulus, we find, as a secondary occurrence, the formation of a true annular incarceration, either at the entrance, or in rare cases, at other points.

During this period, in which the volvulus becomes fixed, in consequence of the tumefaction, the incarceration, and even the adhesion of the contiguous serous surfaces, which is brought about by plastic exudation, it gradually or periodically enlarges to an enormous extent; the peristaltic action, and the increased accumulation of the intestinal contents, forcing the volvulus, the sheath of which continues to be progressively inverted, lower down. We are thus led to distinguish between a primary and a consecutive form.

If the intussusception does not prove fatal by the peritonitis which extends upwards from the serous surfaces of the entering and receding tube, with symptoms of strangulation, or by gangrene of the volvulus, it may have other more or less favorable terminations.

a. The most favorable issue, although purchased at the greatest risk of life, is gangrene and discharge of the volvulus and its mesenteric portion, subsequent to a complete adhesion between the entering and receding tube at the entrance into the sheath. At the spot where the separation has taken place, we find, in the corpses of individuals who had been thus affected, an annular swelling, which more or less interferes with the calibre of the intestine, and adhesions with the contiguous peritoneal surface, and more particularly of the mesentery.

b. In rare cases, in which the incarceration has been developed at an unusual point, only a partial sloughing of the volvulus takes place, and the portion which lies above the strangulation is retained. Under these circumstances, the latter forms a conical plug with a narrow channel, and projects into the cavity of its sheath, surrounded by a thick fringe of mucous membrane.

c. Occasionally the inflammatory action which has taken place in the volvulus abates, after having caused adhesion between the entering and receding tube, and the volvulus is retained.

The process described under *a*, generally leaves a sufficient passage,

and consequently ends in a permanent cure, which cannot be predicated of the other two events. In the latter, a chronic state of hyperæmia and inflammatory intumescence remain, with a liability to exacerbations. General intestinal inflammation not unfrequently follows. The channel of the intestine does not suffice for the removal of its contents, and the volvulus, or the remainder of the volvulus, are moreover the cause of a consecutive increase of the intussusception.

Invaginations occur at all ages. Diarrhœa is the chief predisposing cause, and the most rational therapeutic proceeding consists, according as the inversion has taken place upwards or downwards, in an early injection or exhaustion of air by means of a syringe. To be effective, this must be done before the volvulus has formed adhesions.

Intussusception has an analogue in prolapsus ani.

3. *Prolapsus ani.*—Prolapsus ani is a volvulus without a sheath, and it is characterized by an inversion of the internal portion of the intestinal tubing. It represents a sausage-shaped or pyriform tumor, which is contracted at the anus, so as to form a pedicle, and at the free extremity there is, in trifling cases, a round central opening, which in larger prolapsus assumes an eccentric position, and, following the traction exerted upon it by the mesorectum, recedes so as to present a mere fissure. The external mucous layer is the seat of inflammation and swelling, which partly proceeds from mechanical hyperæmia, partly from irritation produced by the atmosphere. It is the result of violent and lasting diarrhœa in children, or of blennorrhœa of the rectum in adults and old people.

4. *Altered position of the intestine consequent upon adhesions.*—These changes of position[1] vary according to the point of adhesion, and assume very different forms. They are of importance, as they sometimes offer impediments to the propulsion of the contents of the intestine; but this is not in a ratio with the extent or degree of adhesion, but bears a direct relation to the degree of dislocation produced in one or more coils of intestine. We are now alluding to the adhesions produced by cellular or fibro-cellular tissue, the remains of an entirely extinct exudative process, since we find that similar adhesions, when accompanied by heterologous formations, and especially by peritoneal tuberculosis, rarely produce constipation, but almost invariably give rise to diarrhœa.

We therefore speak of the following forms:

a. Partial circumscribed adhesions of the intestine, with the abdominal parietes, with a second, less movable portion of the tube, with the mesentery, with the internal female sexual organs, &c., causing an angular inflection of the intestine. The inflection will be the more considerable, the more the adhesion is limited, and the more remote the latter is from the normal position of the intestine.

When occurring at the colon, the dislocation may be induced by direct adhesions of the less attached portions, or indirectly by the adhesions of the omentum, especially when the latter is shortened, or when it lies in the sac of an inguinal or femoral hernia.

b. Extensive adhesions among the coils or the mesenteries, that often affect the entire small intestine, in such a manner as to twist and bend

[1] Oestr. Jahrb. xviii. 1.

them, and to produce external valvular duplicatures of the intestinal coats at the projecting angles. This form of adhesion is not unfrequently developed in intestinal segments which have long been included in large hernial sacs, in which case it is limited in extent. A remarkable instance of this was offered in a case of fatal constipation, where a portion of the ileum, twenty-four inches in length, was found inclosed in a cartilaginous sheath of peritoneum of four inches.

§ 4. *Solutions of Continuity.*—These are the effect of penetrating injuries produced by cutting instruments or firearms, or they may be the result of concussions affecting the entire trunk, as in the case of a fall from a considerable height, or a small portion of the abdomen only, as from compression, in being driven over, &c., in either instance giving rise to rupture or laceration of the intestine; or they may be the consequence of ulcerative processes that proceed from without inwards, or *vice versâ*, in the shape of perforating intestinal ulcer.

The danger of these lesions bears a direct relation to their extent, and in the last case, also to the rapidity with which the morbid state is developed.

We must finally adduce those perforations of the intestine which are the combined result of mechanical injury, and of an ulcerative process brought on by foreign bodies that have been introduced into the canal; the putrefaction of the intestine, in consequence of sloughing gangrene at or near the spot; and the spontaneous and incurable ruptures of the intestine which follow its excessive distension above a stricture, and are commonly accompanied by circumscribed sloughing of the mucous membrane, or which are the consequence of complete softening of the coats.

Unless the injury affects the coats of the intestine in a very slanting direction, we find that in wounds produced by cutting or stabbing, sloughing or ulceration, the mucous membrane projects over the peritoneal surface in the shape of a tumid fold. In the case of ulcerative perforation, this will not take place until the destruction of the external and internal plates coincide.

In those cases in which a fatal termination is not induced by an escape of fecal matter into the abdominal cavity, giving rise to general peritonitis, nature adopts the following process:

After a mechanical injury has been inflicted, we find that in the vicinity of the orifice, plastic exudation immediately agglutinates the perforated coil to an adjoining surface, which temporarily closes up the hole; in ulcerative processes the perforation is generally anticipated by the inflammatory action of the peritoneum throwing out a guard of lymph.

This agglutination, when following injuries to the intestines that occupy the umbilical region, rarely unites them with the abdominal parietes, except by the intervention of the omentum, which protrudes into the opening of the abdominal walls; it commonly unites them to a neighboring coil. The small intestine that lies in the inguinal region, the colon, a portion of intestine included in a hernial sac, are in close proximity to parietal regions allowing agglutination, and we there find the lymph converted into cellular tissue.

The opening in the intestine communicates with the external surface

of the body by means of the agglutinating medium. After ulcerative or gangrenous perforation has occurred, the extravasated intestinal contents give rise to and maintain inflammation and ulceration; and thus perforation of the abdominal parietes or of the adjoining intestinal coil is induced. In the first two cases an abnormal opening of the intestine outwards is formed, which, according to its size, and in proportion as it suffices for the discharge of feculent matter, receives the name of fistula stercoralis or anus artificialis. In the latter case an abnormal communication is established between two portions of intestine (fistula bimucosa), and then we have a condition which presents a variety of complications.

These results may not take place; the minute intestinal orifice which results from ulcerative or gangrenous perforations, not sufficing to induce the secondary destruction of the adjoining abdominal or intestinal parietes, the agglutinating tissue is converted into a rounded extended cord, into which the perforated intestine sends a funnel-shaped prolongation of its mucous membrane, and the intestine itself is thus less firmly attached. Continued traction gradually closes up this funnel-shaped cavity, the cord becomes solid, and the mucous membrane cicatrizes over it, generally leaving a pouch at the spot. At a later period the cord may become detached, and it then shrivels up into a cellulo-fibrous nodule lying above the cicatrix of the mucous membrane.

The cure of fistula stercoralis is established in a similar manner. The intestinal opening communicates by means of a layer of organized lymph, with the external surface of the abdomen. The exudation gradually becomes distended so as to form a hollow cord, which, to a certain extent, is lined by the mucous membrane of the perforated intestine; continued traction lengthens out the cord, its channel diminishes at the same time and finally closes. The immediate consequence is the healing up of the external fistulous opening, and in the same manner cicatrization of the intestinal orifice may be effected.

§ 5. *Diseases of the Tissues.*—The muscular coat is scarcely ever proved (by cadaveric examination), to be primarily affected; the disease almost invariably arises in the mucous and the submucous cellular tissue, and involves the former secondarily; we are, therefore, the more limited to a consideration of the affections of the mucous and the submucous cellular tissues, as they demand a minute investigation on account of their extreme importance.

We may infer the general importance of this branch of pathology from the rank the mucous membrane occupies in the domestic economy, from the consequent frequency, and the variety in the forms of its idiopathic affections, but more especially from the frequency of the secondary complications to which it is subject, from the numerous relations which it bears to other systems and organs, and the fluids at large.

We introduce the subject of inflammation by a preliminary consideration of the hyperæmic and anæmic states of the mucous membrane.

1. *Hyperæmia, Anæmia.*—*a.* Hyperæmia is the result of active congestion, arising from idiopathic, sympathetic, or metastatic irritation, or it presents itself in the passive form as the precursor of asthenic inflammation, in consequence of a paralyzed state of organic innervation; or it

may be purely mechanical, arising from obstruction to the circulation by pressure, incarceration of the intestine and its mesentery, contraction of the large vessels and the heart, impermeability of the lungs, &c., in which case it affects the veins chiefly. In consequence of the vascular injection, the mucous membrane of the intestine offers various degrees of redness, or there are slight extravasations or ecchymoses; or, as is particularly seen in the last two cases of hyperæmia, the mucous membrane, or even the entire membranes of the intestine, may present a uniform reddish-black color, the tissue being saturated with blood, and no injection of bloodvessels being distinguishable; the larger vessels, and particularly the venous trunks are distended even as far as the mesenteries, and overcharged with blood (*apoplexia intestini*). In either case hemorrhage may take place into the cavity of the intestine.

b. Anæmia of the intestinal mucous membrane occurs in connection with an atrophic state of the intestinal coats, and accompanying tabes universalis and general anæmia. It is often seen as a sequel of a rapid consumption of the vital fluids from excessive diarrhœa and exhausting discharges, and it appears in a very marked form in the gelatinous softening of the stomach and of the intestine in children. The intestine presents the color of tissue that has been rendered pale by maceration; or it may have the peculiar yellowish pallor of wax.

2. *Inflammations of the intestinal mucous membrane.*—We are acquainted with a catarrhal (erythematous) and a croupy form of inflammation, and, on account of the prevalence of a dyscrasic type, we may consider the typhous and the dysenteric processes occurring in the intestinal mucous membrane as allied to the latter.

a. Catarrhal inflammation—or in a lower degree mere catarrhal irritation, catarrh—presents itself as genuine entero-catarrhus, with a discharge of a thin muco-serous secretion in catarrhal diarrhœas, namely, as a consequence of suppressed cutaneous exhalation. It may result from mechanical or chemical irritation of the intestinal mucous membrane by foreign bodies or stagnating fecal matter; it may also be developed in consequence of congestive or venous stasis in the portal system; or, lastly, it frequently shows a specific, contagious property (exanthematic, typhoid catarrhs), and appears as a precursor of these processes or associated with them, and in the vicinity of morbid growths.

Catarrh is either acute or chronic, and it either attacks the mucous membrane uniformly, or is developed mainly in the villi and follicles.

The anatomical signs of the acute form are, more or less intense redness and injection of the mucous membrane, affecting its entire surface, or appearing as punctiform reddening from affection of the villi, or as a vascular halo surrounding the follicles; relaxation of the tissue, and intumescence of the mucous membrane, equally affecting the entire substance, or only the villi and the follicles, opacity of the mucous membrane and its epithelium from infiltration of the former, and softening of the latter; friability and softening of the mucous membrane. The submucous cellular tissue is injected, relaxed, and infiltrated with a watery, opaque fluid; the secretion is at first copious and serous; as the affection increases in intensity, the former diminishes in amount, becomes opaque, viscid, and puriform.

Chronic inflammation is characterized, in addition to the above signs, by a dark, rusty, livid discoloration, which in severe cases appears to pervade the entire mucous membrane; by a tumid state of the mucous membrane and its follicles, accompanied by increased density of the tissue, copious secretion of an opaque, grayish-white, or yellow puriform mucus.

Acute inflammation frequently passes into resolution, but it often recurs, and may, if the predisposing cause is not got rid of, become habitual or chronic. Chronic inflammation rarely admits of a complete cure. It is generally followed by a blennorrhoic condition; and we thus find, in well-marked cases, a permanent dilatation of the vessels established, with the following alteration in the tissues:

A brown, slate-colored, or bluish-black discoloration of the mucous membrane (deposit of pigment,) involving its entire thickness, or the villi or the follicles only;

Increase of substance, or permanent tumefaction of the mucous membrane, its follicles, or villi, with increased density and consistence (hypertrophy), giving rise in higher degrees to elongation of the membrane, and formation of folds and polypi;

Hypertrophy of the submucous cellular tissue and the muscular coat;

Profuse secretion of a grayish-white and milky, or of a transparent gelatinous and viscid mucus (pituita vitrea).

Catarrhal inflammation occasionally passes into suppuration and ulceration. This is found to occur in consequence of frequent relapses of acute inflammation, but more particularly when an acute attack supervenes upon an existing chronic affection, or invades a blennorrhoic mucous membrane. The mucous membrane is converted into a dark-red, granulated and friable tissue, on the surface of and within which suppuration is established. This penetrates to the deeper tissues, and thus gives rise to abscesses, which open internally; in either case loss of substance is entailed, which increases with more or less rapidity; ulcers follow, which are surrounded by tumid, irregular, sinuous, undermined edges, having a granulating base, and extending into the surrounding cellular tissue or into the hypertrophied muscular coat. The suppuration may even pass through the latter by means of sinuses, in the vicinity of which the mucous membrane presents the above-described appearances, or is blennorrhoic, and often covered with polypous excrescences. This process is invariably accompanied by corrugation and slaty or bluish-black discoloration of the intestinal coats. Catarrhal phthisis thus occasions a contraction of the intestinal canal, which becomes more considerable after the cure of the former. Cicatrization is effected by a dense, resisting cellulo-fibrous tissue, which compresses the mucous membrane in the vicinity of the loss of substance, or the solitary insular remnants of the mucous membrane, into plicated polypous tumours.

The seat and extent of the catarrhal inflammation and of the blennorrhœa, differ according to the cause. They are frequently spread more or less uniformly over the entire intestinal tract; they are often limited to a certain portion of the colon or the small intestine, where they occupy large spaces; or they may occur in one or several small circumscribed spots, in consequence of local irritation. These affections are peculiarly

liable to recur as long as the predisposing cause continues; they exacerbate from time to time if chronic, and lead to suppuration. They are not common in the small intestine, their usual seat being certain portions of the large intestine, viz. the cæcum and rectum.

A peculiar disease that we must here advert to is ulcerative inflammation of the follicles of the colon, such as we find in lientery, brought on by tedious diarrhœas. An ulcer results, which is distinguished from the catarrhal ulcer just described, by the shape which it derives from the follicle, and still more by the total absence of reaction, which is brought on by the excessive destruction of tissue, and which produces an atonic and relaxed state of the tissues at the base.

In this disease, which in the dead subject is commonly not observed until it has committed extensive ravages, the follicles are at first tumefied in various degrees, and consequently project as smaller or larger round, conical nodules on the internal surface of the intestine, being surrounded by a dark-red vascular halo. Ulceration now ensues in the interior of the follicle, the small abscess penetrates the mucous membrane within the vascular halo, and a fringed ulcerated opening, of the size of a millet seed appears, which leads to a small follicular abscess with red spongy walls. The ulceration continues, and the follicle is eaten away. The mucous membrane that loosely surrounds the enlarged orifice of the abscess, overlays the exposed submucous tissue. In most cases the hyperæmia of this edge diminishes in consequence of the exhaustion brought on by the discharges; it becomes pale, or is discolored by a deposition of black pigment in its tissue, which gives rise to a slaty appearance. The ulcer is of the size of a pea or a lentil, round or oval, the mucous membrane at the circumference is pale, slate-colored, livid, and much relaxed, the cellular tissue at the base is dull white, anæmic, sanguineous or dark blue. A flabby typhous ulcer of the colon is the only thing that might render the diagnosis uncertain.

At this period a secondary destruction of the intestinal mucous membrane commences, which proceeds with great rapidity. The original follicular ulcer enlarges in every direction, forming sinuses and exposing the pale, lax, muscular coat at its base. Several ulcers coalesce, and we thus frequently find the mucous membrane and its cellular substratum destroyed to a considerable extent and the remaining portion of the mucous membrane pale or slate-colored; there is general anæmia and tabes; and the contents of the intestinal canal consist of the half-digested food mixed up with reddish, semifluid, grumous matters.

We may state it as a rule, that the lower down the original, as well as the secondary process takes place, the more fully they are developed. Hence the most extensive destruction is found to occur in the sigmoid flexure and the rectum. It is always confined to the colon. Occasionally the disease runs a still more rapid course, as in infants at the breast; and it is then accompanied by catarrhal irritation of the small intestine.

On account of the alvine discharges, which are invariably associated with this ulcer, the affection may not inappropriately be termed ulcerative diarrhœa.

b. Exudative processes of the intestinal mucous membrane.—Under this head we include all those products of serous, albuminous, pasty,

fibrinous, puriform, and purulent exudation occurring on the mucous membrane, which are more or less profuse, and are preceded by slight redness and congestion. Maceration and solution of the epithelium, relaxation, and infiltration of the mucous and submucous tissues, fusion (as it were self-secretion) and gradual disappearance of the mucous membrane and its follicles, take place at the same time. The mucous membrane is softened and tumid, it is infiltrated with the exuded matter, variously reddened and injected, or pale, or of a dirty gray or tawny color. In proportion to the degree of vascularity and the quality of the exudation, it is more or less pultaceous, and attenuated or entirely destroyed.

We here also adduce the process that takes place on the intestinal mucous membrane in cholera, the acute pituitous condition of the mucous membrane (Eisenmann's pyrotic process), genuine croup, puriform and purulent diarrhœas, &c.

These processes probably always involve a large tract of intestine, and are the expression of a constitutional affection, which itself may either be primary or secondary; in the latter case it represents a degeneration or an anomalous form of the original disease. The not unfrequent degenerations of specific cachexia, such as typhus, the exanthemata (particularly variola and scarlatina), acute tubercle and cancer, which were originally acute, or have become so under certain conditions, may thus present the type of the process just described.

c. The Typhous Process.—The first duty of the pathological anatomist in this case, is to institute a comprehensive investigation of the local typhous processes, and we offer the results derived from the observation of normal ileo-typhus as it is presented in the indigenous form, which is commonly very defined in its localization. On account of the importance of the subject, we shall add a summary of the changes that occur in other systems and organs in typhus, as well as a synopsis of the more important anomalies of the typhous process, that we are at present acquainted with.

The Typhous Process in the Mucous Membrane of the Small Intestine.

The typhous process of the small intestine presents four stages:
The congestive stage.
The stage of deposition of the typhous product,—of typhous infiltration; the crude stage of the deposit.
The stage of softening and rejection of the typhous deposit.
The stage of the genuine typhous ulcer.

In the first stage, which corresponds to the period of irritation with a predominance of catarrhal and gastric symptoms, we observe on the mucous membrane of the small intestine, dilatation and stasis in the venous system, with swelling, and a peculiar succulence of the mucous membrane, accompanied by opacity and slaty discoloration. The swelling of the villous layer is particularly distinct. This condition affects, more or less, the entire mucous membrane of the small intestine, but it deve-

lopes itself more strongly at some parts than at others, and there generally appears to be a gradual increase from above downwards, as far as the cæcal valve. The inner surface of the intestine is invested by a thick layer of dirty, yellow, gelatinous mucus.

The mesenteric glands are slightly swollen, their bloodvessels are injected, the tissue itself is elastic, soft and dark-colored.

In the second stage the congestion is diminished; the injection and reddening, and even the swelling of the mucous membrane, retract within circumscribed spaces which correspond with Peyer's agminated glands, or occasionally with solitary follicles. Rounded or more commonly elliptical tumefactions (plaques), varying in thickness from half a line to three lines, are formed, which result from the deposition of a peculiar substance in the tissue of the Peyerian plexus and of the submucous cellular tissue. They are surrounded by a vascular wreath which stops short at their circumference, and by a marginal plane which rises abruptly, or is contracted, so as to appear pediculated. In the latter case they the more resemble flat sessile fungi, as they often present an umbilical indentation at their centre. According to the amount of matter accumulated, the mucous membrane is more or less tense, being intimately blended with the deposit, as this again is firmly and immovably attached to the muscular coat of the intestines.

The typhous patches offer a gray or tawny discoloration, which is perceptible through the mucous membrane as well as through the two external coats of the intestine, and they are hard and resilient: when the discoloration is darker, and more of a bluish-red tint, they are softer and more compressible. They appear, when seen through the peritoneum, as insulated spots; they may be generally recognized by the varicose condition of the peritoneal vessels, and they are perceptible to the touch through the tumefaction on the external surface of the intestine.

The lower third of the small intestine is the common seat of typhous infiltration, and the typhous spots are placed at the side opposite to the insertion of the mesentery; they increase in number towards the cæcal valve. They vary in size from that of a sixpence to half-a-crown; towards the terminal portion of the small intestine, in correspondence with the extensive glandular apparatus that exists here, they occupy a space of several inches, and end upon the ileal surface of the valve. Near and between the patches we find single, round, nodulated tumors of the size of a hempseed or pea, surrounded by a similar vascular wreath; these represent the typhous infiltration of a solitary follicle.

On minute examination of the morbid product, it proves to be deposited under the mucous membrane and in the submucous tissue, without involving the muscular coat. It presents a substance of more or less density, of a pale-red color and fibro-lardaceous texture; it is occasionally traversed by streaks of blood. The deposit very rarely extends beyond the follicular apparatus.

The swelling of the mesenteric glands also increases; they are of the size of a bean or hazelnut, blue or bluish-red, tolerably firm, and apparently infiltrated with a lardaceous mass.

The commencement of the third stage is marked by a return of violent congestion to the small intestine. The vessels, and especially the veins

of the mesentery and their ramifications between the intestinal coats, are filled with dark-purple and viscid blood. The mucous membrane again swells, the villi in particular tumefy, and on pressure, exude a grayish-white opaque serum.

The most remarkable change is now effected in the typhous patches and in the mesenteric glands; they soften. The patches become more tumefied, and if the softening process does not affect them uniformly, they acquire an uneven tuberculated surface. The deposit is converted into a grayish-red medullary mass; this may, from the imbibition of bile, be at once metamorphosed into a dirty-yellow or brown slough, involving the investing mucous membrane. The slough shrivels up in a vertical and lateral direction, becoming loose at the edges and pultaceous, splitting in various directions, and detaching itself from the lowest stratum of submucous cellular tissue, by which means it is wholly or in part discharged; or the morbid product degenerates, when the epidemy is of very intense character, into a loose, vascular, fungous growth, which is traversed by streaks of extravasated blood, or is entirely saturated with blood; it is the chief source of profuse intestinal hemorrhages, and is generally discharged piecemeal without antecedent sloughing.

This metamorphosis sometimes attacks the entire patch, sometimes it only affects single portions or separate folliculi; in the latter case the remainder of the patch passes through a retrograde metamorphosis; absorption causes it to collapse, and a lax, succulent, plicated tumefaction of the glandular plexus remains. Accordingly, the above-mentioned slough is in the former instance embraced by the mucous membrane, which invests the marginal surface of the typhous patch; in the latter, by the retrograde[1] portion (retrograder antheil) of the glandular plexus.

A similar metamorphosis takes place in the tumefaction of the solitary glands, though it appears to commence later, and to advance less rapidly; the sloughs are small and rounded, and seem generally to undergo the retrograde process.

This metamorphosis commences in the neighborhood of the cæcal valve, and is commonly in advance of that which takes place at the upper part of the ileum.

The intestine is more or less distended with gas (meteorismus); it also contains yellow or brownish muco-gelatinous or biliary matters, mixed up with grumous, furred particles; it always occupies a low position in the hypogastrium, and even sinks into the pelvic cavity. The cæcum is very often found to contain the trichocephalus dispar in larger or smaller numbers.

The mesenteric glands, which almost invariably pass through the stages of the metamorphosis with less rapidity than the typhous products in the intestine, now attain their largest bulk; they reach the size of pigeons' eggs; and, in the vicinity of the cæcal valve, even of hens' eggs, and form a tuberculated chain which extends in a slanting direction from the terminal portion of the ileum to the lumbar plexus. Their

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[1] [The terms "retrograde" and "retrogression" are intended to designate the return of a diseased part to its normal condition by absorption of the deposit, or otherwise. They have been adopted from the absence of any terms which exactly convey the author's meaning. —Ed.]

color is blue or bluish-red; they are much congested, and the vessels, spread out in the cellular capsule of the gland, present a vascular network which is perceptible through the mesenteric laminæ. Their substance is firm, but they are soon converted into a grayish-red, lax, medullary matter, in which we frequently discover extensive extravasations of blood; they then become soft and elastic, or even present distinct fluctuation.

Fourth stage. After the morbid product has been detached, a cavity remains on the internal surface of the intestine, which represents the true typhous ulcer.

If the entire morbid growth is removed at once, that portion of the intestinal mucous membrane which invested the marginal surface of the heterologous product sinks down upon the ulcer, and thus forms a mucous fringe, which varies in width and extent according to the previous elevation (thickness) of the morbid growth; and from being at first dark-red, subsequently assumes a blackish-blue or slate-gray color. If the morbid growth has only been partially detached, the remaining portion of the patch becoming retrograde, we find the smaller ulcerated surface equally surrounded by a margin of glandular tissue.

In the former case the base of the ulcer corresponds in form and size to the previous infiltration (plaque); varying in size, it is either round or, more frequently, elliptic; the latter shape prevails at the terminal portion of the ileum, and the long diameter of the ulcer corresponds with the longitudinal axis of the intestine. In the second case the ulcer is, at all events, smaller than the entire Peyerian gland, its shape irregular, the margin sinuous or round. Several ulcers are often grouped together. For the typhous infiltration of a solitary follicle a circular or slightly oval ulcer is substituted.

The deep submucous cellular layer, which invests the muscular coat, forms the base of the ulcer.

The mesenteric glands decrease in size, as soon as the detachment of the intestinal morbid growth has commenced, in proportion as the grayish-red medullary substance, with which they are infiltrated, is removed, though they still continue larger than they are in the healthy condition; in consequence of the enduring congestion and enlargement of the vessels they present a reddish-blue tinge.

The typhous ulcer consequently presents the following characters:

Firstly. Its form is elliptical when it corresponds to the infiltration and detachment of a larger patch of Peyer's glands; it is round when it corresponds to a solitary follicle or a rounded patch, or to the partial detachment of a glandular plexus; and, lastly, it may also be irregular or sinuous when corresponding to a partial detachment.

Secondly. The size or circumference of the ulcer varies, from that of a hemp-seed or a pea to that of half-a-crown.

Thirdly. The position is peculiar in reference to those of an elliptical shape; they are placed opposite to the insertion of the mesentery, and their long diameter is always parallel to the longitudinal axis of the intestine; the typhous ulcer never forms a zone; at least, we have only once seen this occur in many hundred cases.

Fourthly. The margin of the ulcer is invariably formed by a well-defined fringe of mucous membrane, which is a line or more wide, detached, freely movable, of a bluish-red, and subsequently of a slaty or blackish-blue color.

Fifthly. The base of the ulcer is formed by a delicate layer of submucous tissue which covers the muscular coat; like the marginal substance, it is quite void of morbid growth.

Sixthly. The small intestine is the seat of the ulcerative process, and the lower third is most liable to be involved—the number and size of the ulcers increase as they advance towards the cæcal valve.

The cure of the typhous ulcer to be complete, requires several local and general conditions, of which the chief are the termination of the local process, and the complete extinction of the typhous dyscrasia. When such favorable circumstances occur, the cure is effected in the following manner:

The fringe of mucous membrane which lies upon the base of the ulcer, gradually connects itself, from without inwards, with the cellular tissue that invests the base, and uniting with it becomes paler and thinner. At the same time the cellular layer becomes whiter and denser, and is finally converted into a serous lamina, the circumference of which is dovetailed between the muscular and mucous coats. The margin of mucous membrane is bevelled off in such a manner, that the union is imperceptible; the former does not advance uniformly on all sides towards the centre of the ulcer, hence the elliptical is converted into a sinuous, the round into an elliptical ulcer. At the same time the margin, as well as the neighboring mucous membrane, are thinned down in such a manner, that at last their villi appear to have been transferred to the serous lamina. The edges unite finally at one or more spots, and coalesce. We have sometimes observed, that long before the union of the edges, small villosities formed independently on the serous lamina, a fact which has been also remarked by Sebastian.

Instead of the ulcer we find, in proportion as the above process is effected, a slight depression on the internal surface of the intestine, dependent upon the thinning of the mucous membrane and its connection with a thin cellular layer of denser structure,—or we find a spot at which the mucous membrane is more firmly attached and less movable, in the middle of which, by oblique light, we may often discover a smooth remainder of the serous lamina of the size of a millet-seed; or, if even this is not the case, we discover a spot at which the mucous membrane is more tense, void of plicæ, smooth, less vascular than the surrounding portion, and particularly less villous.

Such cicatrices have occasionally been observed thirty years after the typhus had occurred.

It is singular, and characteristic of the typhous ulcer and its cicatrix, that it never in any way gives rise to a diminution of the calibre of the intestine.

The mesenteric glands in the meantime have returned to their normal size; they not unfrequently shrivel up, so as to become considerably smaller, and at the same time tough and pale.

Summary of the Alterations occurring in other Organs.

a. In the abdominal cavity.—We find that here only the spleen and the venous system of the fundus ventriculi offer important and constant changes, although these do not belong exclusively to typhus, and still less to ileo-typhus.

The spleen is enlarged to from twice to six times its natural size, it swells, and its sheath becomes tense and smooth ; the tissue of the organ is friable, and contains a dark purple or blackish-red, semi-coagulated, pultaceous, or perfectly fluid mass, which gives rise to a tumor of peculiar appearance, occasionally communicating the sense of fluctuation ; not unfrequently a spontaneous rupture of the organ ensues.

At the fundus ventriculi we find venous congestion, which may be traced back to the vessels of the spleen, and which is either limited to the larger trunks or affects the capillary vessels in the tissue of the mucous membrane ; in the latter case, the mucous membrane of the fundus is dark red, lax, and turgid, and, in consequence, similar to the condition of the spleen, rather more friable than in the normal state. Allied to this condition is the first stage of softening, which, however, does not appear in the ordinary course of typhus.

b. In the thorax.—The bronchial mucous membrane and the parenchyma of the lungs present certain constant changes, which, however, vary in degree.

The former is affected by a peculiar catarrh, accompanied by dark-red discoloration, and the secretion of a viscid gelatinous mucus, which increases in amount as we descend to the smaller subdivisions of the bronchi; the pulmonary parenchyma presents symptoms of hypostatic congestion, which is generally limited to the posterior and lower portions ; the tissue appears dark red, or purple, is filled with dark-colored glutinous blood, is denser, and resembles the spleen in consistency (splenification); this is sometimes increased to hepatization (pneumonia), though it is to be carefully distinguished from secondary, and still more, from primary pneumonic typhus.

The heart is commonly flaccid, its muscular portions are pale, or of a dirty-red color, but without any further anomaly, and more especially without that softening of its substance described by Stokes as occurring in the typhus fevers of Ireland. The endocardium and the lining membrane, or all the coats of the vascular trunks, frequently present a brown or purplish discoloration produced by imbibition.

c. Alterations in the nervous system.—The brain and the spinal cord and their membranes present the most various gradations with reference to the amount of blood they contain, from hyperæmia to anæmia; they sometimes are characterized by remarkable density and tenacity, sometimes by a humid and softened condition.

The double condition which is frequently and distinctly seen in the central ganglia of the vegetative system, is of still greater importance, and the results obtained at the Viennese Hospital, since the year 1824, with regard to this question, are in the main corroborated by the observations made at the Wurzburg school of medicine.

The ganglia of the solar and superior mesenteric plexus are, in the first stages of typhus, in a state of turgescence, with a blue or greenish-red discoloration; they are softened in the ulcerative stages, and subsequently we find them collapsed, pale, flaccid, shrivelled up as it were into coriaceous, tough, white or grayish masses.

We have never discovered in the nervous system the characters of genuine inflammation, a fact which is also established by the investigations of Rey, in opposition to those of Grossheim.

Summary of the most remarkable Anomalies of the Local Typhous Process.

An acquaintance with the many anomalies of this process is of such importance, that we would not trust a person ignorant of them to judge of a post-mortem examination in a case of acute fever. Their diagnosis is the result of researches which we have for many years devoted to the subject. We add a list of the anomalies, and subjoin the most essential explanations at once:

1. Anomalies in reference to the *amount* of the process occurring on the intestinal mucous membrane.

a. Arrest of its development.

a. Arrest in the congestive stage—diffused typhous process in the intestinal mucous membrane.

β. Imperfect development of the patches—low plasticity of the morbid product. This variety is allied to the diffused form.

γ. Retrogression (retrogradwerden) of the morbid growths by absorption;—to this head belong Chomel's *plaques à surface reticulée.*

δ. Slow metamorphosis of the morbid growth, tardy separation of the slough, and purification of the ulcer.

ε. Scanty formation of the morbid growth.

b. Excessive development of the local process.

a. Tumultuous[1] (tumultuarisch) metamorphosis of the morbid growth, violent congestion of the intestine, unusual turgescence of the morbid growths. The congestion not unfrequently gives rise to peritonitis, which proceeds from one of the patches; or an extravasation of blood occurs between the intestinal coats, and in their tissue; intestinal apoplexy, or a fungoid degeneration of the morbid growths takes place, and death ensues from excessive vegetation, by paralysis, or from exhaustion by hemorrhage (hæmorrhagia intestinalis).

β. Numerous formations of morbid growths—extension of the same to the solitary follicles.

γ. Extension of the process beyond the ileum to the jejunum and stomach, or to the colon.

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[1] [The German word "tumultuarisch" implies, violent symptoms taking place with suddenness and rapidity; Rokitansky has himself used the term in a new sense; the translator, to avoid frequent circumlocution, has therefore ventured to employ the word "tumultuous" as most adapted to convey the author's exact meaning.—ED.]

2. Anomalies in *quality*.

a. Impeded cicatrization of the ulcer—it assumes the torpid form.

b. Degeneration into a perforating typhous ulcer.

These two forms constitute genuine typhous intestinal phthisis. We have seen that the local condition for the cure of the typhous ulcer consists in a complete termination of the local morbid process in the intestinal mucous membrane, and a perfect purification of the ulcer of all morbid growth, and that, as a general condition, an extirpation of the typhous and of every secondary dyscrasia is required; it is therefore evident that the degenerations of the typhous ulcer which we are now considering may be complicated with a variety of anomalies; of these some have already been considered.

Perforation of the intestine by the typhous ulcer constitutes a very remarkable phenomenon. How is this effected? The typhous process invariably meets with an isolating tissue in the lower stratum of the submucous cellular layer and of the muscular coat; the destructive process which occurs beyond the mucous membrane, is therefore not the result of a previous typhous affection (infiltration), but of an essentially distinct process. It is this that affords a marked distinction between the perforating typhous and the perforating tubercular ulcer. The process by which perforation of the intestinal parietes at the base of the ulcer is effected, is softening or mortification of the tissue; the slough that results only affects the deepest parts of the ulcer to a small extent, and we rarely find the orifice larger than a pin-hole, or a millet or hemp-seed.

The varying period at which in the course of typhus the ulcer degenerates in this manner, is remarkable, as also the rapidity with which occasionally the perforation is effected. We have observed it occur rapidly in ulcers that had scarcely formed, whilst the remaining morbid growths were engaged in the metamorphosis, or even in the crude stage; and again we have seen it occur slowly or quickly at every subsequent stage; long after the termination of the local process, and even after the genuine typhous had subsided into the atonic ulcer.

The consequence of the intestinal perforation, and of the resulting effusion of the intestinal contents into the peritoneal cavity is peritonitis; it generally gives rise to tolerably copious, but uncoagulable and liquid exudation; it frequently takes place even before actual perforation has ensued, and is developed as soon as the process of perforation approaches the peritoneum.

The exudation commonly induces an adhesion between the perforated coil and another coil, or between its mesentery and the pelvic parietes; which may certainly be looked upon as an effort of the *vis medicatrix naturæ*, but which our investigations have proved, never to effect a radical cure of the typhous perforation of the intestine.

Our experience with regard to the perforating process, does not, except in rare cases, allow us to concur in the view adopted by several French observers of distinction, that it is to be considered as a rupture of the ulcerated part; nor can we sanction the doctrine of Judas, that the intestine, when on the point of being perforated, moves into the pelvic cavity, in order to find suitable spots for adhesion, inasmuch as the typhous intes-

tine sinks into the lowest region of the abdominal cavity long before the ulcerative degeneration takes place.

Appendix.—Anomalies in reference to the Degree and Character of the Typhous Process in the Mesenteric Glands.

1. Tumultuous metamorphosis of the typhous product in the mesenteric glands.

This sometimes consists in very violent congestion of the gland, which not unfrequently gives rise to inflammatory injection of the mesenteric laminæ above the gland; or great tumefaction takes place, and the gland is converted into a medullary, ichorous pulp; or, lastly, a fungous growth forms, which perforates one of the mesenteric laminæ, commonly the anterior one. General peritonitis is a frequent consequence of these occurrences; in the last instance we have, as in the case of intestinal hemorrhages, extravasations into the peritoneal sac. This anomaly generally occurs in a gland that is seated near the termination of the ileum, and is accompanied by a tumultuous metamorphosis in the intestinal mucous membrane.

2. Atrophy of the mesenteric glands.

The involution of the mesenteric glands after the termination of the typhous process is sometimes carried to excess, and an atrophy of these glands results. They then appear shrivelled, flaccid, coriaceous, perfectly bloodless, pale or gray, or even of a dark blue color. Occasionally the atrophy is less perceptible, as the gland appears of its normal, or even beyond its normal size; this, however, is only the consequence of passive congestion or stasis, from dilatation of the vessels, which are full of blood, and give it a bluish-red color, the glandular tissue itself being diminished.

The typhous ulcer of the intestine at the same time assumes a torpid condition, or it may have advanced, in some measure, to cicatrization.

We shall show at a future period how this condition forms an important anatomical basis for the constitutional debility consequent upon typhus.

3. Secondary typhous processes.

An acquaintance with these processes is the more important, as they throw much light upon the nature of typhus, as they have hitherto been but little known, and as their connection with the primary disease is commonly overlooked. They almost invariably present anomalies in reference to the seat of the typhous process; several of them are remarkable for the frequency of their occurrence. They are to be distinguished either as genuine or as degenerated typhous processes.

Genuine secondary typhous processes generally depend upon marked anomalies in the degree of the primary process. The most exquisite form in which they present themselves is seen in the mucous membranes, which we must consider as the true nidus of the typhus.

a. Secondary processes in mucous membranes.

a. Recurrent eruption of the mucous membrane of the small intestine. —We may find typhous patches of recent formation in the crude stage, intervening among typhous formations which are undergoing the metamorphosis or at the side of the typhous ulcer. These must be carefully distinguished from the patches which are less advanced in their development. This eruption is occasionally seen in a very undeveloped state, in the form of miliary swelling of the solitary follicles.

β. Secondary typhous process in the mucous membrane of the colon and stomach;—secondary colo-typhus, secondary gastric typhus.—The latter is a rare occurrence; it stops short at the congestive stage when it does take place, and very rarely presents the nodulated form of the typhous deposit.

γ. Secondary laryngeal typhus.—This is a secondary typhous process of considerable importance, both on account of its frequency and on account of its unfavorable prognosis. Its seat is the posterior surface of the larynx and the edges of the epiglottis; it not unfrequently gives rise to typhous laryngeal phthisis, accompanied by necrosis of the cartilages.

δ. Secondary pharyngeal typhus occurs much more rarely than the former, and never except in company with it.

ε. Secondary bronchial and pneumonic typhus.
This must be carefully distinguished from the hypostatic pneumonia frequently developed in the course of typhus, as well as from capillary phlebitis of the lungs.

ζ. Secondary typhous process in the vesical mucous membrane.

η. Secondary typhous process in the mucous membrane of the female sexual organs.

b. Secondary typhous processes in serous membranes.—Among these we reckon the typhous inflammations of the pleura, of the meninges, of the capsule of the aqueous humor, and of the internal coat of the vessels (Phlebitis typhosa).

c. Secondary typhous processes in parenchymatous organs.—These are the typhous inflammations of the liver, the spleen, the parotid, and the ganglionic substance of the brain and spinal cord.

The degenerated secondary typhous process occurs in various forms; in almost all of them a suspicion arises of the existence of a disease analogous to typhus, and this fact offers the more interest, as we have arrived at similar results in our special investigations of the morbid anatomy of ileo-typhus. Autenrieth describes them as neuroparalytic inflammations, Schönlein as neurophlogoses, Eisenmann as pyra, Buzorini under the head of typhus. They are based upon a corresponding degeneration of the typhous process in the blood, and may be classed as follows:

a. Degeneration into croupy inflammation.—This includes the entire exudative processes of the mucous membrane, of the respiratory organs, the œsophagus, the stomach, the intestinal canal, the female sexual organs; as well as all the secondary exudative processes occurring on serous membranes; and the exudations that ensue on the cellular and muscular base of the typhous ulcer, as a degeneration of the local affection of the intestinal mucous membrane.

β. Degeneration into acute softening.—To this class belong first of all

the remarkable and frequent cases of black ramollissement of the fundus ventriculi and the œsophagus in which the spleen sometimes participates, and which originates in the vascular system ; and in a second degree the softening of the pulmonary parenchyma, and of the mucous membrane of the bladder. When occurring as a degeneration of the local process, it is found at the base of the typhous ulcer, and may superinduce perforation of the intestine. (Vid. p. 69.)

γ. Degeneration into gangrenous inflammation and primary gangrene. —This includes the well-known phenomena occurring in the course of typhus in the shape of noma, gangræna pulmonum, sloughing of the nates (decubitus), of parts to which vesicants have been applied, and of the female sexual organs. It may occur as a degeneration of the local process, and by sloughing at the base of the typhous ulcer induce perforation of the intestine.

d. Degeneration into a process in which pus, or rather a fluid analogous to pus is formed.—This involves suppuration of the patches and of the typhous ulcer, in the mesenteric glands ; as well as suppuration in the lungs, the spleen, the liver, the parotid, in the subcutaneous cellular tissue, between the muscles, &c.

Besides these anomalies, there are other sequelæ of typhus, which are based upon a permanent depression of the entire vegetative system, such as tabes universalis ; or upon a diminution of nervous power, as obtuseness of the senses or paralysis ; or again upon continued irritation, as hydrocephalus ; or lastly, upon a secondary constitutional disease, as presented in œdema, anasarca, permanent suppurative processes, and Bright's renal disease.

The depression of the vegetative system remaining after typhus demands special investigation. It is presented either as a very slow progress of convalescence, or in the advanced degree as genuine tabes ; both forms are distinguished by their peculiar type. The following are the anatomical points which characterize them :

a. Genuine intestinal phthisis, or where a cure of the intestinal ulcers has begun, or is almost terminated, a loss of villi and follicles ;

β. A shrivelled condition or marasmus of a considerable number of mesenteric glands ; and

γ. The flaccid atrophic condition of the abdominal ganglia, and more especially of the solar and superior mesenteric plexus, which, as well as the former, we have already adverted to.

EPICRISIS.

Firstly. Typhus is characterized anatomically and in reference to the alterations in the solids, by the deposition of a peculiar product, which undergoes a peculiar metamorphosis.

Secondly. Its habitat varies and depends upon the specific relation existing between the general disease and certain organs. Indigenous as well as exotic typhus show the mucous membranes and the lymphatic glands to be the chief seat ; in Austria it is chiefly the mucous membrane of the small intestine, yet even here bronchial and pneumonic typhus occur as a primary affection, and ought probably to be considered as the

basis of the exanthematic form; we also, though very rarely, meet with colo-typhus.

Thirdly. The product of typhus presents in its first, but still more, in its later stages of metamorphosis, the greatest analogy with cancerous growths, and more particularly with medullary cancer.[1]

Fourthly. The local typhous process is a species of inflammation; but not one of those to which we attribute a phlogistic crasis of the blood, but one which, on account of the peculiar diseased condition of the blood, we term typhous.

Fifthly. The local affection of the mucous membrane of the small intestine, is a constant accompaniment of the typhus seen among ourselves; but as, according to our previous observations, it may occasionally be subject to an arrest of development, we find solitary exceptions in which there is no intestinal affection; in that case it is necessary to watch the other mucous membranes closely, or, indeed, the process, without being localized, may run its entire course in the blood.

It is well known that typhus occurs chiefly during the period of puberty and during the prime of life; before and after this epoch, it is very unfrequent; we must however guard against considering every typhoid appearance in Peyer's patches, during the early years of life, as genuine typhus. The predisposition seems to disappear with the involution of the sexual powers; still it does occur now and then, after the sixtieth and seventieth years of life.

Typhus presents a peculiarly interesting negative relation in reference to its capability of forming combinations. Pregnancy offers an almost entire immunity from typhus, lactation less so, and cases in which it is complicated with tubercular affections, with cyanosis, cancer and the cancerous cachexies are exceptional, whereas it is frequently complicated with syphilis and gonorrhœa.

d. The Dysenteric Process.[2]—We are acquainted with the dysenteric process as a substantive disease of the mucous membrane of the colon, and inasmuch as it is here presented in its most exquisite form, its habitat has been correctly fixed ever since the days of Hippocrates.

The dysenteric process is divisible into four natural degrees or forms.

In the lowest degree, the mucous membrane commonly presents a layer of a thin secretion, of a dirty gray and reddish color, underneath which, certain parts, commonly the projecting folds of the mucous membrane, are reddened and swollen. In this manner striæ are produced, which more or less encircle the intestine. The epithelium is either raised in the shape of small vesicles which contain clear serum, or it forms a grayish-white layer, resembling the mealy scurf of the epidermis, an appearance which probably induced Linnæus to term dysentery *Scabies intestinorum interna.* The subjacent mucous membrane seems excoriated, slight pressure induces hemorrhage, and it may be easily detached in the shape of a light red sanguineous pulp; its submucous cellular tissue appears infiltrated.

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[1] We must leave a further development of this doctrine to oral instruction. Dr. Mohr, in his Contributions to Pathological Anatomy (Stuttgart, 1838, p. 131), quotes, in connection with this subject, an authority which is quite foreign to the matter.

[2] Vide Oestr. Jahrb. xx. I.

The anatomical characters may be summed up as—swelling, injection and reddening, softening (red and bleeding), serous exudation in the shape of a delicate vesicular eruption and consequent branny desquamation of the epidermis.

In the second degree, the textural alterations are not limited in the manner described, but extend over a larger surface, still, however, presenting a greater development at one part than at another. The mucous membrane is invested to the same extent, by a dirty-gray layer, consisting of desquamated epithelium and a thick glutinous exudation; or this may already have been removed, and the subjacent mucous membrane, in either case, appears converted into a soft, sanguineous, pale-red and yellowish gelatinous substance, which may be easily detached. The internal surface of the intestine commonly presents more or less numerous protuberances, which closer examination proves to consist of a very copious infiltration of the submucous cellular tissue: these projections or tumors were first observed by Hewson and Pringle; other authors speak of them as warty tubercular swellings, or fungoid excrescences, and M. Gély has lately termed them *Hypertrophie mamelonnée du tissue sousmuqueux.*

They correspond to those points of the mucous membrane at which the morbid affection is most developed; with the exception of slight redness and intumescence, especially in the circumference of the follicles, an increase in the mucous secretion, and a slight desquamation of epithelium, the intervening parts of the mucous membrane do not generally offer any marked textural changes. The entire portion of intestine is generally in a state of passive dilatation; it is distended with gas and with a dirty brown fluid, which consists of the most different materials, such as intestinal secretions, epithelium, lymph, blood, and fæces; its coats are thickened, and the submucous tissue particularly is in a state of tumefaction.

At this stage we meet with the laminated and tubular coagula in the evacuations, described by ancient and modern authors, especially if the exudation be of a more plastic character.

Occasionally the affection of the follicles predominates and is accompanied by irritation, exhausting secretions, and softening: these probably constitute the characteristic signs of the so-called catarrhal or white dysentery, but which, in an anatomical point of view, is the same follicular affection of the colon as that which we have already described as accompanying chronic diarrhœa.

In the third stage, we find the protuberances more closely set, so as to produce an uneven, lobulated appearance. The mucous membrane that invests these protuberances partly retains the above-described conformation; in part it is converted into a slough, which is here and there blended with the desquamated epithelium and the exudation, and is firmly attached to them; it is of a dark-red or blackish-brown, sugillated, or grayish-green color; or the mucous membrane may have disappeared, so as to expose the infiltrated submucous cellular tissue, to which the remnants of the mucous membrane remain attached in the shape of solitary, dark-red, flaccid, and bleeding vascular tufts, or as dilated follicles, which are easily removed. The interstices of the mucous membrane are the seat of the affection in a lower degree.

The protuberances occasionally are found to have coalesced, and the

intestine then presents an uneven plicated surface, accompanied by an equable degree of infiltration and thickening of its parietes; the mucous membrane is uniformly affected over a large extent, and there are no free interstices.

The contents of the intestine are of a dirty-brown or reddish, ichorous, fetid, flocculent and grumous character.

In the fourth and highest degree, the mucous membrane degenerates into a black, friable, carbonified mass, which may often be subsequently voided in the shape of tubular laminæ (so-called mortification of the mucous membrane). The submucous cellular tissue appears to be previously infiltrated with carbonified blood, or a sero-sanguinolent fluid; or it is pallid, and the blood contained in its vessels is converted into a black, solid or pulverulent mass: subsequently it shows purulent infiltration, in consequence of the reactive inflammation which is induced in the lower healthy strata, for the purpose of eliminating the gangrenous portions.

The affected portion of intestine, which contains a putrid, brownish-black fluid, resembling coffee-grounds, may appear in a state of passive dilatation, as above described, but it is much more frequently collapsed; and if the two highest degrees continue for any length of time, the muscular coat will be reduced. The tissue of the latter is condensed, pale, ashy, peculiarly elastic and friable, and analogous to the yellow fibrous tissue.

The peritoneal coat presents, in the higher, and particularly in the highest degree of the affection, a dirty-gray discoloration, and a total absence of lustre; at intervals it presents a dilatation and injection of its capillary vessels, and is invested with a brownish, ichorous exudation; occasionally the meso-colon, and even the mesenteric laminæ, that have been in contact with them, participate in the affection. This affords a means of distinguishing dysenteric disease of the intestine on its outer surface.

The glands of the meso-colon present a corresponding tumefaction; they are of a dark-blue color, congested and tumefied; but we have not succeeded in detecting in them a peculiar (specific) solid morbid product, as we have in typhus.

The mucous membrane of the colon is, as we have already observed, the seat of the dysenteric process; and we may state it as a rule, that its intensity increases from the cæcal valve downwards, and consequently is met with, in the most fully-developed state, in the sigmoid flexure and in the rectum. It not unfrequently passes beyond the cæcal valve, towards the ileum, but is here only seen in its mildest form.

It commonly runs an acute course, though it is frequently chronic in the milder degrees; this, however, does not materially alter its character.

The manner in which it terminates varies.

1. The disease is fatal, in consequence of the more or less rapid, or more or less penetrating destruction of tissue, and the coincident exhaustion.

2. The disease may terminate in cure, if the mucous membrane has not become disorganized in the manner above-described, the normal cohesion returning, and a new layer being generated under the desquamated epithelium.

3. In the higher degrees of the disease, when disorganization has occurred in one of the above-described processes, and the mucous membrane has suffered more or less extensive destruction, one of two results ensues :

a. A real cure of the loss of substance, with consolidation of the abraded portions of the intestine follows ; or,

b. The entire process assumes a low chronic form, the specific nature of the disease is lost, and we have atonic inflammation and suppuration of the intestinal coats.

If a cure ensues, the portions of mucous membrane which were affected in a lower degree are first restored to their normal condition ; between them are small patches, or more extensive spaces, with a sinuous circumference, at which the mucous membrane is deficient, and the submucous, pale, infiltrated cellular tissue is exposed. Not unfrequently we perceive detached remnants of mucous membrane adhering to these parts. The exposed submucous cellular tissue is gradually converted, as proved by cadaveric examinations at the most various periods after the cessation of dysentery, into serous tissue ; this is further condensed into sero-fibrous tissue, and by it the sinuous portions of mucous membrane, at the edge of the impaired surface, are, like the isolated remnants of mucous membrane, compressed into warty, pediculated (polypous) prolongations, and thus the originally sinuous circumference obtains a fringed, dentated appearance. In cases in which the loss of substance is inconsiderable, the new tissue may contract so as to bring the edges of the mucous membrane into apposition with one another and with the polypous remnants of mucous membrane, and the cicatrix is then represented by a large number of agminated warty excrescences of the mucous membrane, between which the sero-fibrous basis from which they proceed, may be detected.

In cases of extensive destruction of substance, the approach of the edges is rendered impossible ; the deeper layers of the tissue, which takes the place of the mucous membrane, is frequently condensed into fibrous bands, which form corded projections into the intestinal cavity, interlace with one another, and not unfrequently encroach upon the calibre of the intestine in the shape of valvular or annular folds, thus giving rise to a stricture in the colon of a very peculiar form. This mode of regeneration is the more remarkable, as it closely resembles that following the destruction of the œsophageal mucous membrane by mineral acids.

In the second case the specific affection terminates after having previously given rise to more or less extensive disorganization, but without being followed by the healing process just described. The entire disease now assumes a chronic character, and appears on the residual portion of mucous membrane as chronic catarrhal inflammation, the follicles being more or less prominently affected, and suppuration occurring in the shape of sinuses and abscesses under the mucous membrane, and between the external coats of the intestine ; at the same time the intestinal canal contracts, its coats assume a rusty, dark-blue color ; there is occasional exacerbation of the peritoneal irritation, and the intestine becomes fixed in consequence of exudation and infiltration in its cellular sheath and its mesentery. In this case the mucous membrane is found of a dull, red color, tumefied, and invested by a copious secretion of a glairy or purulent

character; the follicles, particularly those at the end of the colon, are dilated, distended by a glassy pituita, or in a state of suppuration; there are small abscesses, of the size of a hemp-seed or pea, under the mucous membrane, and in the cellular tissue lying between the muscular fibres. These abscesses open upon the mucous membrane by the suppurating follicles or by other minute orifices, forming fistulous passages in various directions, and penetrating into deeper parts, so as to reach the peritoneum, and there induce inflammation; or they give rise, in the vicinity of the rectum, to the formation of large abscesses, as described by Morgagni.

The concurrent contraction of the intestinal tube probably causes in this case, also, a diminution of its calibre, but this form presents no peculiarity to distinguish it from the effect which may be produced in every case of catarrhal inflammation attended by repeated exacerbations. (Vide p. 60.)

The dysenteric process occurs in its exquisite and primary form in the colon only, with the exception of the mucous membrane of the female sexual organs, where it affects the uterine mucous membrane in the shape of the puerperal disease.

The dysenteric process offers the greatest analogy to the corrosion of the mucous membrane produced by a caustic acid. The consequent destruction of the tissues, as well as the phenomena of reaction, present throughout a close resemblance in both cases, and the stricture produced in the œsophagus has no analogue but that resulting in the colon from the dysenteric affection.

We have found a further analogy with the dysenteric process in the erodent effect produced upon the mucous membrane of the œsophagus by the gastric juice in scirrhous stenosis of the pylorus.

Appendix.—*The Non-typhous Intumescence of the Follicles and Villi of the Intestines.*

Although the intumescence of the intestinal follicles occurring in various morbid conditions is not the consequence of palpable inflammatory action, it may yet be fairly considered at this place, as it commonly appears to result from the relation of certain general morbid states to the follicular apparatus.

We find that the patches of Peyer in the small intestine, the solitary follicles of the small and large intestine, and the follicles of Lieberkühn, in the small intestine, may be affected in this manner. The affection is observed:

1. In substantive affections of the intestinal mucous membrane, as in diarrhœa, and particularly when occurring in children, in whom it is marked by more or less vascularity and congestion, but frequently also by an anæmic condition of the parts. In the diarrhœa of children and young persons we find, besides an enlargement of the solitary and of Peyer's glands, a dilatation of Lieberkühn's follicles; a grayish-white creamy matter accumulates in their interior, which produces a whitish punctiform appearance in the intestinal mucous membrane, or, in transmitted light, gives rise to so many opacities.

2. The affection occurs most frequently as a reflex of constitutional disease : under these circumstances, the swellings of the solitary and of Peyer's follicles are found principally in the colon in typhoid gastro-enteric fevers, as an imperfectly-developed secondary typhous eruption in almost all the exanthemata, but especially in scarlet fever, variola, and erysipelas ; in acute rheumatism and gout ; in croup ; in suppurative and gangrenous disease ; in febrile affections of the lymphatic glands in scrofulous individuals ; in hydrocephalic fever ; in a marked form in common Asiatic cholera ; and lastly, in acute convulsions, trismus, and tetanus. The villi are generally also much swollen, but we invariably find the mesenteric glands in a state of tumefaction.

Swelling of the follicles is the consequence of a deposition of a grayish-red, dull-white, or yellowish substance, of a lardaceous or creamy and glutinous consistence in the cavity of the follicle, accompanied by an analogous infiltration of its parietes ; thus the follicle and the deposit not unfrequently appear to constitute a homogeneous body, to which the term " granulations of the intestinal mucous membrane" has been applied. This follicular affection differs from that occurring in typhus in everything that characterizes the latter, and especially in reference to the metamorphosis of the typhous follicle.

According to the predisposing constitutional causes, the affection we are treating of is more or less acute and transitory ; the deposit, the follicular tissue, and the mucous membrane in very rare cases fuse into a small shallow ulcer ; induration and a further development occasionally take place, and the mucous membrane being pushed forwards, a species of polypoid pediculated growth is formed.

3. *Gangrene of the mucous membrane.*—We have had occasion to examine the ulcerative process consequent upon inflammation in a variety of forms, and any further investigation of the subject were superfluous. We pass at once to gangrene of the mucous membrane, although we must observe that it rarely is a direct consequence of inflammation.

Gangrene of the mucous membrane is brought on by compression and traction, and is generally accompanied by gangrene of the other intestinal coats, as in incarcerated hernia at the point of strangulation, or in consequence of excessive dilatation of a portion of intestine above a stricture, at various scattered points ; it may occur in large patches in consequence of mechanical hyperæmia brought on by incarceration, or of passive congestion induced by paralysis ; it may take place in the shape of a circumscribed slough of the mucous membrane consequent upon inflammatory action (gangrenous inflammation strictly so called), in which the peculiar anomalous state of the blood and the peculiar nature of the product, are the cause of mortification. To this head belong the sloughs of the intestinal mucous membrane, which occur with symptoms of general adynamia and putrescence, in acute dyscrasia of the blood, in purulent and ichorous infection of the blood, under the form of degenerated typhus, cholera typhus, &c.

After the slough has become detached there is a loss of substance in the mucous membrane which demands some attention, as it may be confounded with an intestinal ulcer ; the diagnosis is established by the

existence of an external or internal cause of gangrene, or by a correspondence, in seat and form, between the latter and the external influence (compression, traction); again, the slough of gangrenous inflammation is distinguished by its oblong, striated form, and very varying seat, by its defined contour, and by the absence of morbid growth at the edge, at the base, as well as in the circumference of the eroded part.

4. *Inflammation of the submucous cellular tissue.*—In several of the processes we have hitherto considered, we have had occasion to notice the various modes and degrees in which the submucous cellular tissue is involved in disease of the mucous membrane. An isolated inflammation of the submucous cellular tissue is very rare, and when it does occur it is commonly metastatic and terminates in suppuration. It takes place in the shape of distinct foci of varying extent, which either give rise to perforation of the mucous membrane, or advance towards the peritoneum, and here produce peritonitis; or in certain portions of the intestine, as in the cæcum, colon ascendens, and rectum, produce extensive suppuration of the cellular tissue.

5. *Softening of the intestinal canal.*—We may pass over the softening of the intestinal mucous membrane which we have described when treating of the Exudative Processes and Dysentery, as converting the tissue into a pulp, which, in proportion to the state of vascular action, and to the quality of the exuding matters, is either easily removable or is spontaneously detached. We have here to allude to the gelatinous ramollissement of the intestinal mucous membrane, which offers an analogue to the gelatinous softening of the gastric mucous membrane. It is of much rarer occurrence than the latter, though like this, it affects the small intestine as a complication of cerebral disease, of acidity in the primæ viæ, of extreme general collapse, atrophy of the muscular tissue, and anæmia of the intestine; it involves the external coats of the intestine, converts them into a homogeneous, grayish-red, transparent, and deliquescent gelatine, and leads to spontaneous perforation. We also advert to the analogue of black softening of the stomach, which occasionally, though much more rarely, attacks the intestine. It occurs under the same conditions, and mainly affects the mucous membrane of the cæcum, and in this case occurs, like gelatinous softening, on the cellular base of the typhous ulcer.

6. *Morbid growths in the intestinal canal.*—Under this head we consider lipoma, the formation of an anomalous, serous, and fibro-serous tissue, fibrous and fibro-cartilaginous tissue, calcareous concretions, erectile tissue, tubercle, and scirrhus.

a. Lipoma occurs of various size in the shape of lobulated accumulations of fat in the submucous cellular tissue. It forms rounded tumors which are invested by mucous membrane, project into the intestinal canal, and are sessile, or pediculated: in the latter case they push the mucous membrane before them in the course of their development, and become suspended by a pedicle of mucous membrane. Although pre-

senting a polypoid shape, they must be carefully distinguished from true polypus.

b. Anomalous serous and fibro-serous tissue occurs as a temporary or permanent substitute of loss of tissue in the mucous membrane, and in very rare cases in the shape of serous and fibro-serous cysts between the intestinal coats.

c. Fibrous and fibro-cartilaginous tissue is found in the submucous cellular tissue of the stomach, and less frequently, of the œsophagus; it assumes the shape of the rounded or oval, flattened concretions; of a bluish-white color, and elastic and firm consistence, which we have described above. They do not attain a greater size than that of a lentil or pea, and are freely movable under the mucous membrane.

d. Chalky concretions more or less resembling bone, though destitute of its peculiar organization (so-called ossifications), occur very rarely in the intestinal canal. If we sum up the results of the observations made in reference to this point, taken in connection with our incidental remarks when considering the diseases of the peritoneum, we arrive at the following deductions:

α. The concretions occur as lamellæ or delicate cords in the sero-fibrous tissue which is formed supplementary to a loss of mucous tissue;

β. As ossification of the fibroid tissue occurring in the submucous and subserous cellular layers;

γ. As a loose chalky concretion or indurated calcareous pus between the intestinal coats in sinuses accompanying catarrhal intestinal phthisis;

δ. As calcareous tubercle of the intestinal mucous membrane or the peritoneum;

ε. As ossification of peritoneal exudation on the intestine.

e. Erectile tissue occurs as a pediculated polypus (mucous or cellular polypus), or in the shape of large, broad, sessile tumors, chiefly as a consequence of catarrh in the colon and rectum. It may in this case also be the seat of medullary carcinomatous infiltration.

f. Tubercle.—The presence of tubercle in the tissue of the intestinal mucous membrane, and by extension, in the deeper-seated coats, constitutes a most important disease—tuberculosis of the intestine in the wide, tuberculosis of the intestinal mucous membrane in the narrower sense. It may proceed to ulcerative destruction, and this establishes genuine intestinal phthisis.

Amongst ourselves this affection rarely occurs in the idiopathic form, except during the first years of life. It is commonly the consequence of pulmonary tuberculosis, and in the majority of cases, takes place after the latter has attained the suppurative stage (pulmonary phthisis), and the general tubercular cachexia has become fully developed.

The course it runs is frequently chronic, but much oftener acute: the latter is more particularly the case when it follows the tumultuous fusion of numerous pulmonary tubercles. The tubercular deposit offers corresponding varieties in reference to its original form, its seat, and its metamorphosis.

In the chronic affection we find the mucous membrane, and the adjacent layer of submucous cellular tissue, to be the original seat of the tubercular deposit; there is no perceptible inflammatory action, and the

disease appears in the shape of the gray, transparent, tubercular granulation, which softens at its centre, and is gradually converted from within outwards, into the yellow cheesy tubercle. It seems blended with the mucous membrane, and projects into the intestinal cavity in the shape of a sessile, hard nodule.

When the local appearance of tubercle takes place in the acute form, there is considerable inflammatory action. The deposit is effected similarly to that occurring in the pulmonary cells; in the first instance it is deposited in the cavity of Peyer's glands, then into the solitary follicles, and lastly, in every other part of the intestinal mucous tissue; it appears in large masses, and in the shape of yellow, cheesy matter, which speedily undergoes a purulent transformation. The surrounding tissue is found extensively congested, reddened and turgid; and when the deposit is excessive, the mucous membrane of an entire coil may be in a state of congestion and irritation. In this case tubercular tumors, either scattered over the surface of the intestine or more or less accumulated, are found occupying Peyer's patches, offering considerable projections and distinguishable through the mucous membrane by their yellow tinge.

Tubercular deposit in the intestinal mucous membrane, being the result of a fully-developed tubercular cachexia, commonly advances rapidly to softening, and this process is effected with peculiar violence in the second variety. The investing mucous membrane gives way at its most elevated point, and as the orifice enlarges, the suppurating tubercular matter escapes.

A cup-shaped ulcer, of the size of a millet seed or a pea (the primary tubercular ulcer) results; its margin is firmly attached, rounded and indurated, and of a pale or red color in proportion to the reaction that occurs in the surrounding tissue; its base is either formed by the condensed submucous cellular layer, or by the granulated texture of the parietes of the dilated follicle. It is only in very rare cases that the tubercle fuses under the mucous membrane without giving rise to perforation; it then forms at the expense of an inclosed abscess, which enlarges the submucous cellular tissue (vomica submucosa).

The increase of the ulcer takes place with more or less rapidity, it loses its original form, but only to exchange it for a more characteristic secondary one.

The increase is effected by fusion of the tubercular infiltration of the margin of the ulcer, and by concurrent suppuration of the tissue. In the first instance, the small adjoining ulcers coalesce into one of larger size; the common base presents sinuous projections of the common margin of mucous tissue, ridges of mucous membrane may be seen traversing it in various directions, or even solitary insular remnants of this tissue are found upon it.

If this process has occurred, as it does in acute intestinal tuberculosis, in one of Peyer's patches, the ulcer may, on account of the elliptic form prescribed by the shape of the glandular apparatus, be mistaken for a typhous ulcer, but we shall immediately point out that the peculiar relations of the margin and the base afford a satisfactory clue to the diagnosis.

The ulcer, which is formed by a coalition of other smaller ulcers, enlarges in the same manner as the original solitary ulcer, in the direction of the intestinal circumference, and at last presents a zone of varying width and uniformity. Its margin is sinuous or dentated, inverted and tumid, and is formed by mucous membrane of a light red color; from the latter being infiltrated with a transparent gelatinous substance, an analogy is offered with the gelatinous infiltration occurring in the vicinity of tubercular pulmonary abscesses. The base is formed by callous cellular tissue of a dirty white color, underneath which the remaining intestinal layers are found similarly condensed and tumefied.

Both in the marginal tissue and at the base we find a deposition of gray, or more commonly of soft, yellow, tubercular matter. The ulcer presents a very peculiar appearance, on account of the remnants of mucous membrane seen on its base. These adopt the characters of the margin, and become infiltrated with gelatinous matter, so as to form crispated, transparent, condylomatous excrescences of a light-red color.

In the same manner as the tubercular ulcer extends laterally, it may advance in the opposite direction, and thus giving rise to perforation, cause sudden death. Secondary deposition of tubercular matter may equally take place in the callous cellular tissue of the base, and as it fuses at this point, in the muscular and subserous layers also. The peritoneum may become perforated in consequence of tubercular suppuration being established in it, or in consequence of mortification induced by the approach of an abscess. It follows that the tubercular ulcer perforates the intestinal parietes without losing its original character, inasmuch as the progress of the tubercular affection is not arrested by an isolating tissue; in this it differs from the typhous ulcer, which does not perforate the intestine in its original form, but affects the parts beyond the submucous cellular tissue in its degenerated character.

At an earlier or later period we find moderate inflammation attacking points of the peritoneum which correspond in position to the intestinal ulcer; a fibrinous exudation results, which is entirely, or in part, converted into tubercle; in the latter case it is partly converted into cellular tissue. By the intervention of this new product an adhesion is often effected at the point of ulceration, between the intestine and a neighboring organ, e. g. the bladder, the omentum, and thus a more or less substantial impediment is offered to the free discharge of the intestinal contents into the peritoneal cavity on the occurrence of perforation.

The mesenteric glands, lying in the vicinity of the affected portion of the intestine, are variously enlarged: in the primary intestinal tuberculosis of children they frequently attain the size of a walnut or hen's egg; they appear tuberculated and pale, and present a deposition of grayish, medullary, and hard, or of yellow, grumous, and deliquescent, tubercular matter.

The small intestine is the common seat of intestinal tuberculosis, and in most cases the disease is limited to this part; still it often passes on to the colon and descends to the rectum, or it ascends into the jejunum, and in very rare cases mounts to the duodenum and the stomach. Sometimes it is much advanced in the colon and then appears to have

been first developed at this point and subsequently to have extended to the small intestine.

We may gather from the circumstances accompanying intestinal tuberculosis, that the further it has advanced the less a cure is to be hoped for. Still in the same manner as in the tubercular abscesses of the lungs, we sometimes obcerve a healing process established in a few among a large number of ulcers. It takes place in the following manner.

The first indispensable condition is the cessation of all secondary tubercular infiltration at the margin or base of the ulcer; the callous base is then condensed into a fibro-medullary cord, and the edges of the ulcer approach one another. This process sometimes advances so far, that the dentated edges almost touch, and between them a whitish, callous cord may be observed. Occasionally, the edges are soldered together over the callosity, yet so as to leave a fissure at one end of the ulcer. In very rare cases an entire consolidation is effected.

In consequence of the contraction of the ulcer, a cicatrix forms on the surface of the intestine, which presents a more or less elevated tumid ridge on the internal surface of the intestine. If the ulcer was of considerable size, or if it encircled the entire intestine, a callous annular ridge remains, which diminishes the calibre of the intestine, and when viewed from without, occasionally gives rise to an appearance of invagination.

Thus the cure of a tubercular intestinal ulcer is always accompanied by a diminution of the intestinal calibre.

g. Scirrhus, carcinoma of the intestine.—The carcinomatous affections of the intestine, occur in the three forms of fibrous, areolar, and medullary cancer, with and without the formation of pigment : two of these or all three, may be combined with one another, from their first origin or consecutively. The areolar form, however is, at least with us, of very rare occurrence.

The colon is almost exclusively the seat of cancerous degeneration, but there is a gradation in the proclivity of its different sections to the affection. The rectum is most frequently attacked, in second order the sigmoid flexure, and the remaining portion of the colon but rarely. The small intestine is scarcely ever the primary seat of cancer; it is almost always involved secondarily after adhesions have been effected with a cancerous portion of the colon by means of peritoneal exudation. Medullary carcinomatous cachexia, which is frequently acute and very extensive, forms an exception, inasmuch as it gives rise to a medullary, white or colored infiltration of the mucous membrane of the small intestine and its submucous cellular tissue in the patches of Peyer. If we except this case, carcinoma occurs as a primary affection of the intestine in three forms :

Firstly, In the mucous membrane, as carcinomatous infiltration of the erectile tissue, into which the former has been previously converted —fungus;

Secondly, More frequently in the submucous cellular tissue, as round nodulated accumulations;

Thirdly, Most commonly as an annular deposit of the cancerous tissue in the submucous cellular layer.

When the intestine is secondarily involved, it is attacked laterally, and the disease commonly proceeds from the lymphatic glands of the mesentery, or from those of the lumbar plexus.

A distinction of the two latter forms is of importance, in reference to the observations that we are about to make.

Here also, carcinoma presents the well-known stages of crudity and metamorphosis; and we merely direct attention to this again, because a consideration of the fact is absolutely necessary for a complete exposition of cancerous intestinal stricture, which, next to cancer itself is of extreme interest.

Cancerous stricture of the intestine[1] (Enterostenosis scirrhosa, cancerosa) is the most common variety of stricture that results from alterations in the intestinal coats, and at the same time the one that advances to the highest degree; it also offers the first elements for a rational theory of ileus.

We have already alluded to the two main forms in which cancer affects the intestine: it is either a narrow annular tumor surrounding the intestine, the primary form, which gives rise to annular stricture; or the intestine is secondarily affected by a propagation of the disease from neighboring organs; in this case one side only may be involved to a considerable extent. In the latter case, however, the cancerous degeneration may gradually extend over the entire circumference of the intestine, as in the former the original annular stricture may extend upwards or downwards over a larger portion of intestine.

The annular stricture is commonly the most important; if the morbid growth continues in the crude stage, the calibre of the intestine may be reduced to the size of the little finger, a goose's or crow's quill. The passage of the intestine is frequently much interfered with in the lateral degeneration by protrusion of the morbid growth, but there is generally a corresponding dilatation of the normal portion of the parietes, and the width of the tube is thus not unfrequently found increased, even after the morbid growth has enveloped the entire circumference of the intestine. Although the former is by far the most dangerous, and soon proves fatal by ileus, this also follows sooner or later in the second case, notwithstanding the existing dilatation.

The metamorphosis of intestinal cancer is of importance in reference to the stricture, both in its first development and in its further progress; it may render the stricture much more dangerous, or may lead to a certain improvement in the symptoms. The turgescence that takes place in the morbid growth at the commencement of the change, and the fungous excrescences that arise on the surface of the intestine during its progress, may render the stricture narrower, and even induce perfect occlusion of the intestine. On the other hand, the contraction may be relieved by sloughing of the softened morbid growth, and imminent ileus thus be postponed. The intestinal disease may, unless death ensue, as it often does from exhaustion, be subsequently ameliorated in various ways. After destruction of the morbid growth, an ichorous cavity is left, into which the descending contents of the intestine pass and stagnate; this

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[1] Oestr. Jahrb. xviii. 1.

condition is sometimes borne for a considerable period, provided there is a sufficient discharge downwards. In other cases ulcerative perforations may establish one or more communications between the portions of intestine lying above and below the stricture, or ulcerative destruction may take place in a different direction, and give rise to artificial (vicarious) anus ; thus affording a hint as to the mode of cure to be adopted by the medical man.

The degenerated and strictured portion of the intestine may remain unattached, or become fixed. The primary degeneration of the intestine, exhibited in annular stricture, is commonly unattached, and it then, in proportion as the diseased mass increases, sinks to a lower region of the abdominal cavity. This may, in the same manner as the scirrhous pylorus, when it has descended to the umbilical or hypogastric regions, give rise to an error of diagnosis. The dislocation is particularly liable to present an obstacle to the passage of the intestinal contents, if the contracted portion is bent at an acute angle, as occurs in the descent of strictured portions of the transverse colon, or of the flexures of the colon.

The diseased portion of intestine may be fixed, as is the case in the secondary lateral degeneration of the intestine from its commencement ; the annular stricture may become attached in the same, or in a different manner. In the former case the intestine is either directly connected with the large lobulated morbid growths that extend to the glands of the lumbar plexus, or even to the ligamentous appendages and the periosteum of the vertebræ (Lobstein's retroperitoneal growths), or it is attached to them by the intervention of a cord or peduncle which passes through the mesentery. In consequence of the partial contraction of the tissues, and especially of the intestinal coats, and of the unequal distribution of the morbid growth, the degenerated portion of the tube is more or less inflected.

The annular stricture, which in the first instance is unattached, may, as the cancer advances, become fixed in a similar manner at the point of its original development, or at different parts at a distance from this point, either by cellular tissue, or by a fusion of the carcinomatous tissues. The propulsion of the intestinal contents will, in that case, be impeded to a greater degree than in simple dislocation, and the more so, the greater the dislocation itself, the more acute the angle of inflection, and the more firm the adhesions are.

The intestine lying above the diseased portion is found affected to a various extent, and commonly in proportion to the amount of contraction, by active dilatation, i. e., dilatation accompanied by hypertrophy of the muscular coat. The parietes of this section of the intestine are occasionally found very much thickened and indurated ; the muscular coat presents a yellow discoloration and is friable, the cellular layers are infiltrated with a gelatinous medullary substance, the mucous membrane is thinned and resembles a serous membrane, and the contents of the intestine accumulate to a considerable extent above the affected point. The portion of intestine which lies below the cancerous mass is more or less permanently contracted and empty.

In considering the metamorphosis of intestinal scirrhus, we have ad-

verted to its terminations; it commonly ends fatally with symptoms of intestinal inflammation and ileus.

. Cancerous ulceration, more frequently than any other variety of ulceration, gives rise to communications between the affected portion of intestine and neighboring cavities and passages, and more especially with the rectum.

Intestinal carcinoma often occurs in the isolated form, but it is not unfrequently complicated with cancer of the stomach, the liver, the lymphatic glands, and the bones, with osteomalacia, and universal cancerous cachexia.

There are certain ulcers which occur only in the large intestine, and especially in the sigmoid flexure and the rectum, and are nearly allied to cancer, and particularly to cutaneous cancer. They are generally solitary, but there may be two, three, or four at a time. They invariably give rise to intense pain, and appear etiologically connected with an abuse of ardent spirits. Although in many respects analogous to the ulcers hitherto considered, they offer distinctive characters. They are invariably zonular and have a callous base, upon which occasionally a discolored, brownish, grumous discharge is visible, and they are surrounded, by a thick, tumid, spongy, carneous, and irregularly-sinuous margin of mucous membrane. They generally cause a diminution of the capacity of the intestine, though not to any considerable degree. A further investigation into their nature still remains a desideratum.

7. *Theory of the ileus produced by cancerous degeneration of the intestine.*—Independent of the degree of stricture, the degenerated portion of the intestine, owing to the adventitious growth deposited in the submucous tissue, and still more from the consequent disorganization of the muscular coat, is in a completely passive condition. The propulsion of the fæces through this portion is therefore effected by the muscular activity of the higher part of the intestine, even when the lateral position of the disease allows of dilatation. The more considerable the stricture, or the more extensive the growth, and the more copious the feculent accumulation, the more will this activity be called into play.

The contents of the intestine necessarily stagnate and accumulate in that portion which lies immediately above the diseased point, and dilate it. If the dilatation is effected suddenly, paralysis at once ensues; otherwise the accumulated masses, a certain portion of which are only propelled through the degenerated section of the intestine, give rise to reaction, hypertrophy of the membranes follows, and as these influences increase, gradual exhaustion and paralysis result. This paralyzed portion of intestine is the proximate cause of the supervening ileus. As soon as the fæces have accumulated within it to such an extent as to reach the adjacent sound portion of intestine, the latter undertakes their discharge. Its capability of effecting this will diminish in proportion to the amount of accumulation, and to the contraction of the stricture. The consequence is, that the peristaltic action is reversed, and that the antiperistaltic movement conveys the intestinal contents to the stomach, from which they are ejected by vomiting.

The coexistent intestinal inflammation, which commonly occurs as general peritonitis, also has a share in the process. It commences at

that point immediately above the stricture, which has become most dilated by the accumulated contents, and it is there most intense. This portion of intestine presents a dark-blue or blackish-red discoloration, with a tinge of brown or green; its coats are infiltrated with blood; the peritoneal investment, which is covered with a dirty-green or brownish exudation, is easily detached; the muscular coat is discolored and friable; the mucous membrane, owing to its distension, is devoid of plicæ, villi, or follicles; dark-red, distended at some parts with coagula, and sloughy. Sometimes all the intestinal coats are perforated at these points, and there is consequently an extravasation of the intestinal contents into the abdominal cavity.

The inflammation extends from this portion of the intestine upwards, and is followed, *pari passu*, by paralysis. It passes from the intestine to the mesenteries, to the omentum, and to the parietal laminæ of the peritoneum.

In some cases the inflammation is the result of irritation existing in the morbid product, which is transferred to the peritoneum, and causes paralysis of the muscular coat above the stricture, dilatation of the intestine and ileus.

It follows that, to appreciate the causes of ileus arising from scirrhous strictures of the intestine correctly, we must take into consideration:

Firstly; the absolute degree of stricture.

Secondly; the degree of attachment of the affected portion of intestine, with or without dislocation and inflection.

Thirdly; the degree of the consecutive affection of the part above the stricture.

Fourthly; the degree of the inflammation present.

Appendix.—Diseases of Separate Sections of the Intestinal Canal.

Separate sections of the intestine demand special attention, inasmuch as not only many diseases occur more frequently at one part than at another, and are subject to numerous modifications in reference to their issue and result, but as many diseases exclusively affect one portion of the intestine. We shall consider the diseases of the duodenum, of the cæcum and vermicular process, and of the rectum, separately, on account of their peculiar importance.

a. Diseases of the duodenum.—We frequently meet with cellular adhesions between the upper transverse portion of the duodenum and the concave surface of the liver and the gall-bladder.

The mucous membrane of the duodenum not unfrequently bulges out through the muscular coat in the shape of a *hernial diverticulum*, an occurrence which is undoubtedly favored by the absence of the peritoneal investment.

Catarrhal irritation, and even inflammation, undoubtedly often affect the duodenal mucous membrane, and are frequently induced by an anomalous condition of the bile. It appears that they may extend to the biliary ducts, and induce icteric symptoms by a retention of the bile (Stokes). We often find evidence of chronic catarrh or blennorrhœa of

the mucous membrane in the dead subject, accompanied by brownish-red or slate-gray discoloration, by hypertrophy of the mucous membrane and Brunner's glands, and by the formation of polypi.

As regards ulcerative processes, we find, besides tubercular ulcer, which is very rare, the perforating ulcer occurring at the upper transverse portion (vide perforating gastric ulcer), and perforation resulting from an extension of the process from the gall-bladder to the duodenum.

Carcinoma very seldom occurs in any shape as a primary affection of the duodenum; it is sometimes secondarily attacked posteriorly by an extension of the disease from the cancerous lymphatic glands surrounding the head of the pancreas and the gall-ducts.

b. Diseases of the cæcum and the vermicular process.—The cæcum and the vermicular process are occasionally absent, or are only imperfectly developed; in some cases the former has been found fissured (Fleischmann).

Anomalies in the position of the cæcum are confined to its position on the left side in lateral dislocation of the abdominal viscera, and to its position in large inguinal or umbilical hernia. Its attachments are sometimes very loose, and this appears to result from repeated dilatation.

Catarrhal inflammation of the cæcal mucous membrane is remarkable on account of the frequency of its occurrence, and that form which is occasioned by habitual constipation, so-called typhlitis stercoralis, is peculiarly characteristic. It chiefly originates in sedentary habits, indigestible food, and rheumatism of the muscular coat. The symptoms are those of catarrhal inflammation generally; it runs an acute course, is subject to frequent relapses and degenerates into the chronic form. Removal of the accumulated pus, and avoidance of fresh accumulations, generally suffice to establish a cure. If this is not effected, ulcerative destruction of the mucous membrane, and continued sinuous suppuration of the muscular coat, result. In this manner rapid perforation of the intestinal parietes, and especially of the posterior side, may follow, either inducing extensive inflammation, ichorous destruction of the cellular tissue in the iliac and lumbar regions and death; or giving rise to general peritonitis, in consequence of the destructive process passing from the right iliac region in a different direction.

In the chronic form the cellular tissue at the posterior surface of the intestine condenses, and the adjoining muscular coat and the entire cæcum shrivel up; on cessation of the ulcerative process, the cæcum is found converted into a slate-colored capsule, with dense parietes, of the size of a walnut or a pigeon's egg; in the place of the mucous membrane there is a sero-fibrous, retiform and trabecular tissue.

In reference to the cæcum we observe, that the inflammation of the loose, stringy, cellular tissue external to the iliac fascia (perityphlitis), is of considerable importance. It is occasionally idiopathic, but more frequently metastatic; it is very dangerous, both on account of the facility with which the pus spreads, and on account of the perforation of the cæcal parietes which may ensue, and the consequent extravasation of intestinal contents into the seat of inflammation.

The vermicular process is sometimes reduced to a mere cellular sinus

of the cæcum; it varies in size from that of an insignificant nodule to five or six inches.

There are considerable variations in the position of the cæcum.

Adhesions of its free extremity may become a matter of importance forming rings or fissures in which the intestine is strangulated.

Catarrhal inflammation of the vermicular process is a disease of common occurrence, and very dangerous on account of its consequences. It much resembles typhlitis stercoralis, and is invariably the result of fæcal matters and foreign bodies, especially small fruit-stones, having become lodged and hardened in it.

The affection has a torpid character, may exist for a long period as blennorrhœa, and is accompanied by thickening of the coat of the vermicular process. After frequent exacerbations it passes into ulceration, which may, if the foreign body remains loose, attack the entire process, or if the former becomes fixed, affect only the point of attachment, or the vermicular process. In the second case, the constant irritation at one spot, or the accumulation of ulcerative secretion and the consequent distension, induce a rapid development of the morbid process.

Under favorable circumstances, especially if the foreign body is discharged, the ulceration terminates, and the vermicular process partially or entirely shrivels up and forms a lead- or slate-colored ligamentous appendix.

In the opposite case the ulceration, especially when gangrene is superinduced, more or less speedily brings on perforation of the vermicular process; this may occur at various points, sometimes at or near the termination, sometimes at the circumference, in such a manner as to cause a division into two parts. This perforation and the consequent discharge of the purulent contents into the peritoneal cavity, are not immediately followed by general peritonitis, inasmuch as the previous irritation has induced adhesions with the neighboring peritoneal folds, which render the ultimate perforation innocucous for a time, as far as regards the remainder of the peritoneum. In the interior of the circumscribed cavity the ulcerative process in the mean while continues, the adhesions gradually give way, and general peritonitis ensues.

We further occasionally observe a metamorphosis of the vermicular process produced by obturation, which is analogous to dropsy of the efferent ducts of glands, and which is most apparent in the gall-bladder (hydrops cystidis felleæ). The foreign body sometimes attaches itself to a certain point and closes the canal without inducing ulceration; in consequence of an accumulation of the mucous secretion the vermicular process dilates, the mucous membrane thins and is gradually converted into a serous membrane which secretes an albuminous fluid. The vermicular process is thus metamorphosed into an hydropic capsule, which in the course of time certainly may become the seat of inflammation, resulting in ulceration and perforation.

Typhous and tuberculosis affections frequently extend to the vermicular process, and both may be followed by perforation.

c. *Diseases of the rectum.*—The main defect of development to which the rectum is liable, is that represented by atresia ani, or congenital occlusion of the anus. In this case the rectum either has a blind termina-

tion, is absent, or opens into the urinary and genital passages (cloaca). In the first case the rectum may reach down to the point where the orifice should be, but the orifice is closed by an expansion of the common integuments over it; these are distended by an effort at defecation, and the meconium may even be seen through them. There may however be a greater deficiency of the rectum, the latter terminating at a higher point, or it may be totally absent, and its place be occupied by dense cellular tissue. In these cases the pelvis appears in an undeveloped state, especially in its antero-posterior diameter; it is very much inclined, and the external genital organs are placed very far back. This affords a valuable aid in the diagnosis, as it allows us to infer a considerable deficiency in the rectum.

Anomalies in the calibre of the rectum are both frequent and important, and appear in the form of dilatations or contractions. The former attain a very considerable extent, presenting sacculated sinuses, and an accumulation of indurated fæces; they are accompanied by considerable thickening of the coats and blennorrhœa. The latter vary much in form and distribution, but more still in respect of their causation. In the first place, the rectum is more liable than any other portion of intestine to be compressed by neighboring viscera, by the pregnant uterus, by tumors developed in the uterine or vaginal parietes, by diseased ovaries, the retroverted uterus, the hypertrophied prostate, vesical calculi, pessaries, &c. The contractions dependent upon disease of the coats of the rectum are of still greater importance. To these belong contractions from hypertrophy of the coats, accompanied by an accumulation of fat, and induration of the surrounding cellular tissue; contraction consequent upon catarrhal inflammation and suppuration, or gonorrhœal ulcer; contraction resulting from a dysenteric cicatrix, polypous tumors, and various forms of cancer. Of these the strictures consequent upon dysentery and cancer are the most important.

Hypertrophy of the sphincter is a remarkable affection; it may in rare cases, especially in children, give rise to obstinate constipation and even ileus, but it frequently induces excoriation of the mucous membrane, the so-called fissure of the rectum.

We have already (p. 56) discussed prolapsus ani.

Catarrh and blennorrhœa, accompanied by hypertrophy of the coats, which frequently gives rise to plicated and polypous excrescences of the mucous membrane, are very frequent affections of the rectum. Gonorrhœal catarrh of the rectum represents a peculiar variety: it affects the same uniformly, or in a circumscribed spot: in the former case it is followed by a shrivelling of the rectum, and the mucous membrane gradually disappears; in the latter by a callous induration of the coats of the rectum, and not unfrequently by the formation of an ulcer, which as well as the stricture is placed in the vicinity of the sphincters, and is distinguished by its zonular form, its sinuous circumference, and its callous puckered base.

The hemorrhoidal ulcer is peculiar to the rectum. It results from the irritation of the mucous membrane, produced by lasting congestion in inversion and prolapsus, strangulation by the sphincters, compression of the hemorrhoidal swellings, and undue medicinal interference. It is

distinguished by its seat in the vicinity of the sphincters, its irregular shape, its indented and sinuous flabby margin of mucous membrane, and the similar ridges of mucous membrane that surround or pass over it. On account of the absence of reaction in the parts, corrosion of the vessels not unfrequently brings on violent hemorrhage.

An inflammation of cellular tissue resembling perityphlitis, occurs in the rectum, as periproctitis. The remarks made in reference to the former apply to the latter also (vide p. 88). It occasionally becomes chronic, and thus induces hypertrophy and callosity of the cellular and adipose tissues surrounding the rectum, which however differ from the analogous result of cancer. Like the hemorrhoidal ulcer, it may cause fistula recti.

Of intestinal cancerous affections, those occurring in the rectum are the most frequent, especially if we include the scirrhous degenerations which involve it by extension from the female sexual organs, but which we do not allude to at present.

Cancerous disease attacks the rectum in the various forms above described as affecting the intestine at large. The following however are particularly remarkable :

a. Erectile tumors developed in the tissue of the mucous membrane, and infiltrated with medullary carcinoma; they assume the shape of broad, sessile, or pediculated fungi. They are commonly placed at the commencement and posterior surface of the rectum, at about three or four inches from the orifice; we find these excrescences only in exceptional cases, at or close to the sphincters.

β. Annular carcinoma and stricture of the rectum. It occurs almost exclusively at the upper portion of the rectum, and especially at the point at which the sigmoid flexure terminates in the rectum, and which, in its normal condition, presents a distinct contraction. The strictured part is either unattached, or, as is more commonly the case, firmly agglutinated laterally to the promontory; notwithstanding its elevated position, it is, as Cruveilhier correctly remarks, pushed down by the feculent accumulations above, which generally precede the occurrence of ileus, it is therefore easily reached in exploring with the finger.

γ. Scirrhous degeneration of the rectum over a large surface, or throughout its entire extent.—This primarily affects the submucous cellular tissue, from which it extends through the entire muscular coat to the cellular sheath of the intestine, the cellular and adipose tissue of the pelvic cavity, to the posterior surface of the vagina, and even to the uterus; or it originally attacks one of the last-named tissues, and involves the rectum secondarily. The rectum is firmly attached, from being agglutinated in its entire extent to the sacrum, or adherent to the vagina, or it appears wedged into the pelvis by the surrounding morbid growth; its calibre may be variously diminished, though it sometimes is unaltered; its internal surface is uneven, nodulated, and hard, or it is filled with soft, fungous, bleeding growths; the anus, especially if the morbid product extends to the sphincters, is patent, everted, and varicose; even the perineum appears more or less swollen, protruded, and hardened; and this induration extends to a considerable extent over the nates in consequence of the condensation of the subcutaneous adipose tissue.

The foreign bodies found in the rectum may either have reached it from above, but not proving injurious until they reach this point, or they may have been introduced, per anum, in consequence of morbid sensations or perverted sexual desire. In the latter case they are commonly very singular objects and of alarming size.

§ 6. *Anomalies of the Intestinal Contents.*—1. Excessive accumulation of gas is very frequently caused by an increase in the secretion on the internal surface of the intestine, accompanied by an impediment to its escape. This occurs over a large extent of intestine in morbid affections of the mucous membrane, and especially in the exudative processes, such as typhus, in the shape of tympanitis; the escape of the gas is impeded by the paralyzed state of the muscular coat. This condition also accompanies anomalous states of other intestinal secretions, especially of the intestinal mucus, or morbid affections of the nervous, especially the ganglionic, system; in the latter instance, however, there is frequently no increase in the amount of gas secreted, but in consequence of the atony of one portion of intestine, and spasmodic contraction of the remainder, or of atony of the entire tube, it accumulates, and is retained in the shape of tympanitis throughout the canal.

Occasionally, an excessive accumulation of gas is brought on by the consumption of certain flatulent articles of diet in a debilitated state of digestion, or where there is an absence of the due amount of bile.

2. The intestinal mucus is very often found in excess, and occasionally the amount secreted is insufficient: in the former case, it also undergoes considerable modifications as to quality. The increase of secretion either exists throughout the intestinal canal, or affects certain sections in the shape of chronic catarrh or blennorrhœa; the mucus is either milky, white, yellowish and purulent, or glutinous, transparent, vitreous, spawny. In the congestive state of typhous and typhoid diseases, we find a peculiar gelatinous mucus on the intestinal mucous membrane, and more especially on that of the small intestine and cæcum.

A diminution in the quantity of mucus accompanies excessive formation of bile and of feculent matter (copropoësis).

3. There can be no doubt that a peculiar gelatinous constitution of the mucus is the nidus of intestinal entozoa, and the cause of helminthiasis. There are two orders of worms, the nematoidea and the cestoidea: to the former belong the ascaris lumbricoides, the trichocephalus dispar, and the oxyuris vermicularis; to the latter, the tænia solium and the botryocephalus dispar.

The lumbricus occurs in the small intestine of children and young persons, and is sometimes found in large numbers, forming knotted accumulations. It often ascends to the stomach, into the œsophagus and pharynx, it may even pass from here into the larynx, and thus, as has been distinctly observed, produce suffocation. Occasionally, several lumbrici may be found undertaking such and similar preposterous peregrinations at the same time.

The trichocephalus dispar inhabits the cæcum and the adjoining portion of the small intestine. Its occurrence in the gelatinous, feculent contents of these parts in typhus, is very important.

The oxyuris (ascaris) vermicularis inhabits the rectum.

The tænia is found, one or more in number, in the small intestine.

We may still be permitted to doubt the fact that the entozoa ever perforate the intestine, at all events, it is a very rare occurrence. It is well established, however, nor is it of very unusual occurrence, and this applies especially to the lumbricus, that they pass through orifices in the intestinal parietes into the abdominal cavity, into abscesses, into the bladder or the vagina.

4. The fæcal matters offer various important points for consideration.

They sometimes accumulate in the intestine to an extraordinary degree, in consequence of repletion, torpor of the intestine, diminution of the intestinal secretion, increase of the absorbent powers of the intestine, and induration of the fæces. These accumulations occasionally affect single portions of the intestine, and may, if persistent, induce disease of the coats.

The occurrence of an excessive elimination of fæces (copropoësis excedens) from the intestinal secretions, is an established fact. It takes place as a critical discharge in various diseases, and especially in those that are accompanied by increased secretion in the intestinal canal; but recent observations have demonstrated its occurrence as an idiopathic disease, which may, by the excessive drain it causes, give rise to atrophy of the intestinal coats and to general emaciation. The color of the fæces mainly depends upon the color and degree of saturation of the bile. They may be dark-brown, dark-green, black, pitchy, or, in the absence of bile, grayish or clayey. Occasionally the fæcal discharge is brown internally, and invested by a white clayey covering, of varying thickness.

The consistency of the fæces varies considerably: they are liquid when the serous exhalation of the mucous membrane is excessive; semifluid when the secretion is muco-gelatinous; or they are mixed, with the secretion in the shape of flocculent grumous particles. The feculent matter found above the various intestinal strictures presents a peculiar frothy appearance.

The fæces may have hardened, so as to present lumps or scybala of various sizes. This scybalous induration generally takes place in the sigmoid flexure and the rectum, though it occasionally reaches up to the cæcal valve. If accompanied by flatulency, small portions of feculent matter are found to adhere to the intestine, and after the mucus by which they were made to adhere has dried up, they appear agglutinated to, and even imbedded in, the internal surface of the intestine.

Figured fæces either form cylinders, which may be variously affected by pressure of the intestine or by stricture, or they form tubers of various size. This leads us to a consideration of fæcal concretions and intestinal calculus.

5. Intestinal concretions are either formed in the intestine, or after being formed external to it, reach it by the natural or by abnormal passages.

To the former belong indurated scybala, which may be produced under all the circumstances that give rise to a retention of fæces; and especially the tuberculated fæcal concretions that form in and adhere to the cavity of colonic diverticula. They may be various foreign bodies, such

as fruit-stones, indigestible portions of vegetables or pieces of bone, which have been introduced into the intestine, and become incrusted with fæcal matter. Or such bodies, especially when occupying a blennorrhoic portion of intestine, as the vermicular process or cæcum, give rise to deposits of grayish fatty matters, chalky and saline substances.

To the latter belong biliary calculi, which have reached the intestine by the natural passages, or by ulcerative communication; and the fatty and chalky concretions which have formed in abscesses adjoining the intestine and have passed into the latter.

Intestinal concretions prove injurious to the intestine, in proportion to their size and form, as we shall have occasion to explain further on.

With regard to serous, muco-serous, albuminous, puriform, and purulent discharges, to fibrinous coagula, and pseudo-membranous formations in the intestine, we refer to the remarks given under these heads.

6. Blood is found in large or small quantities, coagulated and fluid, red or variously discolored, in the vicinity of the point at which it was discharged, or extended over a large surface. Hemorrhage occurs in consequence—

Firstly; Of active, passive, and especially of mechanical hyperæmia; the latter being a frequent result of obstacles in the portal system. The mucous membrane presents no essential textural alterations, but is either congested and suffused, or in consequence of the excessive hemorrhage, pale and anæmic. The source of hemorrhage is scarcely discoverable. We have lately seen two remarkable cases of this description, in which exhausting hemorrhage resulted from intense and extensive burns of the abdomen.

Secondly; In consequence of the various exudative processes accompanied by solution of the mucous tissue and its vessels, e. g. in dysentery.

Thirdly; The hemorrhage may be caused by other morbid degenerations of the mucous tissue, e. g. erectile fungoid excrescences, the typhous deposit at the period of metamorphosis, or torpid ulcers.

Fourthly; In rare cases the hemorrhage results from the rupture of a varicose vein in the submucous tissue of the intestine, the investing mucous membrane giving way at the same time. It is more frequently caused by corrosion of an artery or vein at the base of a hemorrhoidal ulcer of the rectum.

Every variety of hemorrhage, but especially the one first cited, is favored by diminished density of the blood.

When the blood is found extravasated over a large surface, it may have come from above, but it frequently happens that the source of the hemorrhage is below the extravasation; this is particularly the case in hemorrhage of the rectum.

Moreover, the blood may have reached the intestine from the stomach, the œsophagus, the hepatic viscera, and even from the respiratory organs.

The longer the blood remains in the intestine, the longer it has been exposed to the operation of the intestinal secretions, the more it becomes discolored, assuming a chocolate or black tinge; and when it has experienced the influence of the gastric juice, it is frequently converted into a pitchy or tarry mass. Bile in a very concentrated form often presents a similar appearance.

The intestine sometimes offers a passage by which acephalo-cysts of the liver (the so-called hydatids) are discharged.

7. We must lastly investigate the foreign bodies found in the intestine.

To this class belong concretions formed within the body, and especially in the biliary ducts, and substances that have been introduced by mouth or per anum. They prove injurious by producing lesions of the intestinal parietes, as in the case of rough or pointed bodies, bones or fragments of bone, portions of stone, glass, needles, &c. After attaching themselves to the mucous membrane, suppuration is established, and they may thus escape through the intestinal and abdominal parietes; or the perforation may communicate with another portion of intestine, or with a neighboring hollow organ, and the escape be effected through the urinary and genital organs. The foreign bodies may also block up the intestine and induce ileus; these cases are of extreme importance, and they admit of the following subdivision:

Firstly, The foreign body is arrested at a certain point of the intestine, in consequence of its rough and angular form.

Secondly, the foreign body is retained simply from a disproportion between the calibre of the intestine and the size of the substance, and occlusion is the result.

Thirdly, The foreign bodies accumulate to a considerable number at one point, and the consequent extreme dilatation and paralysis of the intestine induce obstruction.

Rough, angular bodies, if not very large, frequently pass through the intestine without difficulty, in an envelope of mucous and feculent matter; but they often become attached to the intestine, by inserting their edges and processes into it, and may, by the consequent inflammatory swelling, give rise to an obliteration of the passage.

Large round or oval bodies, with a smooth surface, may be retained at various points of the small intestine, but especially at the terminal portion of the ileum, which presents a distinct diminution in calibre.

We class among these foreign bodies large biliary calculi, which have escaped from the bile-ducts into the intestine.

Indigestible substances that have been taken in large quantities, especially the peel of fruit, cherry, and plum stones, often accumulate at particular points of the colon, as the cæcum or the sigmoid flexure. They give rise to uniform or lateral dilatation of the intestine, accompanied by atony and paralysis of the latter. This condition may, sooner or later, in a ratio with the size of the accumulated mass, give rise to ileus; or if the accumulation is inconsiderable, and the action of the superior portion of the intestine capable of effecting a partial discharge, it may last a considerable time, and end in a cure; or it results in chronic inflammation, the formation of sinuses, and the ultimate contraction of the intestine, which again may give rise to occlusion.

Appendix.—On spontaneous Ileus.

We distinguish between the so-called organic ileus, into the nature of which we have inquired in preceding paragraphs, and dynamic or spontaneous ileus. The latter deserves a careful investigation of its cada-

veric relations, the more so as a sound theory of its nature, based upon practical experience, is very much wanted.

Ileus is a rare occurrence, and undoubtedly often dependent upon atony of an intestinal segment, which must be viewed as the proximate cause, in contradistinction to the case just examined, in which the accumulation of foreign matter is the primary affection. It takes its origin in a sedentary mode of life, in depressing physical conditions, repletion, superstimulation by purgatives and injections, rheumatic affection of the intestine, diseases of the spinal cord, and even of the brain. The colon is the part almost invariably affected. Stagnation and accumulation of the fæces in the affected portion of intestine follow, dilatation is induced, and the atony ends in paralysis; when this happens, ileus is at hand. Its actual occurrence, however, as well as the improvement and cure of the affection, depend upon the state of innervation in the upper healthy portions of intestine. If the action of these portions suffices to propel the fæces through the dilated segment, and thus from to time to empty it, the latter may resume its functions, and thus return to a healthy condition. If, however, the upper portion of the intestine does not possess sufficient power, which will be the case if the accumulation be excessive, or the paralyzed segment has sunk to a lower region of the abdomen, the accumulation will proceed, and at last reach up to the healthy intestine. Here the peristaltic action is reversed, the fæces are thrown back into the stomach, and are expelled from there by vomiting.

When the paralysis has reached a certain point, inflammation and sloughing set in, and enteritis peritonealis results. As this induces paralysis of the muscular coat and passive dilatation in the upper portion of the intestine, a change occurs in the ileus, inasmuch as the point at which it commences advances with the advance of the inflammation.

All pathologists of distinction deny the possibility of spasmodic contraction or spasmodic stricture in a portion of the intestine, being the cause of obstinate constipation or of ileus. The *modus operandi* of the various remedial agents employed fully confirms the theory given with regard to this simple form of ileus. The benefit derived from purgatives is to be explained by the force with which the healthy intestine propels the fæces downwards, and the rapidity with which they pass through the distended portion; the advantage of opiates consists in diminishing the activity of the healthy portion, and the consequent accumulation in the dilated part, and in allowing the latter time to recover its activity.

It is highly probable that the use of narcotic enemata of tobacco or belladonna, effects an evacuation of the dilated portion, by inducing a complete relaxation in the inferior portion of intestine, which is thus enabled to admit and convey onwards the accumulated fæces. If the injected fluid can be propelled as far as the diseased part, the discharge of the fæces is aided by the mechanical distension of the intestine, and is undoubtedly further promoted by the change of position which the injection effects in the healthy intestine. It follows that injections of fluids that exert no remedial influence, such as air, may effect an evacuation, and thus establish the first condition of a cure.

CHAPTER II.

ABNORMITIES OF THE ACCESSORY ORGANS OF THE ALIMENTARY
CANAL.

SECT. I.—ABNORMITIES OF THE LIVER.

THE diseases of the liver have continued to remain to the present day
a subject of extreme difficulty, in spite of the progress made in the ana-
tomy of this viscus. As one of the chief organs concerned in sanguifica-
tion, it affects, as might indeed have been inferred *à priori*, the somatic
and psychical character of the individual in the most varied and exten-
sive manner within the range of physiological bounds ; and on the other
hand, many of its morbid affections, which are beyond the reach of the
scalpel, become intelligible only by attending to the anomalies presented
in other organs. It is to be hoped that future inquiries may elucidate
them more fully by showing the influence these anomalies have upon the
constitution of the blood, and by explaining the various spontaneous de-
rangements of the vital fluid.

§ 1. *Arrest and Excess of Development.*—The liver is absent in very
imperfect monstrosities, especially in acephalous monsters, in which the
heart, the lungs, and the greater part of the intestinal canal are also de-
ficient ; in biventral monsters the liver presents more or less marked
traces of duplication.

§ 2. *On the Irregularities of Volume generally, and on Hypertrophy
and Atrophy in particular.*—We find the liver either abnormally
enlarged or abnormally diminished in size. The former defect, in which
the left lobe remains permanently enlarged, so as to extend to the left
hypochondrium and beyond the spleen, is occasionally congenital. Both
conditions, when acquired, become extremely interesting in a diagnostic
point of view.
Increase in the volume and weight of the liver depends upon—
Firstly, Hyperæmia, congestive turgor ;
Secondly, Inflammation, inflammatory swelling ;
Thirdly, Congestion and stasis in the capillary gall-vessels ;
Fourthly, True uniform hypertrophy ;
Fifthly, Excessive, but morbid, nutrition, i. e. the deposition or in-
filtration of a substance foreign to the hepatic tissue in quantity or
quality—conditions which have hitherto been considered as hypertrophy
of one of the component parts of the organ ;
Sixthly, Adventitious products, which directly increase the weight and
volume of the liver in proportion to their own number and size, and in-
directly contribute to that effect by the congestion they give rise to in
the surrounding tissue.

Diminution in the volume of the liver is the result of atrophy and alteration in the tissue.

a. Hypertrophy.—Under this head we consider not only the abnormal condition dependent upon exalted nutrition and increased deposition of the peculiar normal constituents of the organ, but those anomalies also in which the increase of size is the result of excessive deposition of separate elements of those constituents, and of the deposition of heterogeneous matter. The former is genuine hypertrophy; the latter, which are often mistermed hypertrophy, includes the nutmeg liver, the fatty liver, and its variety the waxy liver; and lastly, the infiltration of an albuminous, lardaceous, and gelatinous substance.

Although the last-named abnormal conditions are closely connected with deep-seated constitutional and acquired derangement in the vegetative sphere, it is of practical utility to consider them in this section until we shall have arrived at an accurate knowledge of the infiltrated abnormal matter, and of the corresponding anomalies in the vegetative system at large. We are the more justified in adopting this course as the enlargement of the viscus, and especially the peculiar features in its growth which are perceptible to external examination, afford a valuable aid in the recognition of these internal conditions.

a. Pure hypertrophy, i. e. a simple increase of the normal specific tissue, can scarcely occur without uniform hypertrophy of all the constituents of the liver. It is not unfrequent; it is a result of hyperæmia, and presents the following anatomical signs: the liver is increased in volume, but retains its usual shape; it is hard, lacerable and full of blood; the acini appear enlarged, and of the normal reddish-brown color. This coarse-grained texture must be carefully distinguished from so-called granular liver.

β. The nutmeg liver.—That condition of the liver in which a separation of the yellow and reddish-brown substances takes place, especially if the former predominates, and which presents a close resemblance to the section of a nutmeg, has been termed the nutmeg-liver; it is commonly considered as a hypertrophy of the so-called white or secreting portion, the red portion either remaining unaltered or being more or less condensed by the former.

According to our own researches the nutmeg liver occurs under two different conditions, and there are consequently two varieties.

aa. In one case it appears as an enlargement of the capillaries of the biliary canaliculi, accompanied probably by hypertrophy of the latter (the secreting substance), and resulting from excessive secretion of bile and stasis of the secretion. The two substances are the more defined, the darker the color of the bile and of the red substance.

ββ. In the other case it is due to an increased deposit of the fat normally due to the liver.

In either case we trace several degrees:

Firstly, In the lowest degree the normal distinction between the two substances is simply more marked, the white substance appearing more developed;

Secondly, In the second degree the predominance of the white sub-

stance becomes more apparent, and forms circumvolutions that envelope the red substance;

Thirdly, In the highest degree the organ approaches, in the first variety, to the granular; in the second, to the fatty liver.

The liver appears, in the second variety, to be slightly enlarged, at least it is never diminished in size; in the advanced stages it has a tendency to become flattened, and to expand whilst its edges are thickened.

Mechanical hyperæmia of the portal system from disease of the heart is peculiarly liable to encourage the development of the nutmeg liver. The affection occurs very frequently; it may present no symptoms whatever, or be accompanied by distinct signs of hepatic disease, though not such as to indicate the specific derangement. In the form in which it presents the early stage of the fatty liver, it most probably gives rise to the numerous complaints which are relieved by neutral salts, alkalies, mineral waters containing these substances, saponaceous compounds, and the so-called resolvent vegetable extracts.

γ. Fatty liver, the adipose metamorphosis, morbid accumulation of fat in the liver.—A well-marked case is distinguished by the following anatomical characters: the liver is enlarged, the increase of size taking place chiefly in a lateral direction; its edges are flattened and swollen, the peritoneal covering is smooth, shining, transparent and tense; the organ is soft and pits on pressure; its color, internally and externally, is uniformly yellowish-red or light yellow, resembling that of autumnal foliage; it is pale and exsanguine, and contains a large amount of fat, as evidenced by the greasy deposit when cut with a dry warm blade, or as proved by submitting the liver to high temperatures.

The disease consists in a deposition of free adipose tissue to such an extent as not only to replace the true glandular structure, but to penetrate the entire parenchyma to the exclusion of the vascular tissue.

In the earlier stages of the affection the various signs alluded to are less marked.

Two conditions chiefly favor its production:

In the first instance it very commonly accompanies tubercular phthisis; and, according to the researches of Louis, is found in two-thirds of all cases of phthisis. Andral has explained this occurrence on the ground of impeded secretion of hydrogen by the lungs; but extended investigation allows us to conclude that this impediment, which is not even demonstrable, is not the cause of the deposit; but that it is an essential constituent or pathognomonic combination of the tubercular dyscrasia, inasmuch as it allies itself with tubercular affections of every kind, with tubercle of the intestinal mucous membrane, of the bronchial glands, the serous membrane, the bones, &c.

Secondly; The fatty liver is also developed—independently of tubercle—in consequence of a luxurious and indolent regimen, in children that have been gorged with food, and especially as a result of dram-drinking. In this case it is accompanied by accumulations of fat in the omentum, the mesenteries, the pericardium, the heart, and the subcutaneous cellular tissue, by fatty degeneration of the muscular fibres of the gall-bladder, and even of the muscular tissue of the heart; the common integument has a leaden hue, and the perspiration has a greasy appear-

ance and a peculiar odor. The fat bears throughout a resemblance to tallow.

The waxy liver is a variety of the fatty liver; it is distinguished from the latter by a color resembling that of beeswax, by its greater consistence, dryness, and brittleness; and these qualities depend upon a peculiar modification of the infiltrated fat, which, although accumulated to a considerable amount, leaves but few traces on the scalpel.

Occasionally the tallow is seen deposited at a few points only, or it accumulates at particular spots. They are commonly superficial, though they are also seen in the deeper parts in the shape of irregularly-circumscribed maculæ, which are the more conspicuous by their change of color the less the other portions of the liver are involved in the disease, and the darker they are.

δ. Lardaceous (speckig, baconny) liver.—Next in order to the fatty liver are the infiltrations of the hepatic parenchyma by a coarser, gray, sometimes transparent, albuminous, lardaceous, or lardaceo-gelatinous, substance. This affection is found concurrent with constitutional disease of the vegetative system, especially with scrofulous and rickety disease, with syphilitic and mercurial cachexia, and it may consequently be congenital. It appears that it is occasionally developed as a sequela of intermittent fever in cachectic subjects.

The following are its anatomical characters: considerable increase of size and weight, with remarkable lateral development and flattening of the organ; smoothness and tenseness of the peritoneal investment, a certain degree of doughy consistency combined with hardness and elasticity, anæmia, pale, watery, portal blood; gray, grayish-white, or grayish-red color, tinged with yellow or brown; the surface of a section being smooth, and homogeneous, resembling bacon, and leaving but a slight fatty stain on the scalpel. Sometimes, however, there is an adipose deposit in the entire liver, or in certain parts of the organ, and the blade of the scalpel then shows the fatty appearance when a section is made.

In many cases the foreign substance is also deposited in the shape of white lardaceous spots, the edges of which are not distinctly circumscribed.

The spleen is very commonly affected in a corresponding manner; it is found much enlarged, and infiltrated by a similar substance (vide Spleen). Bright's disease of the kidneys and analogous renal affections are also very often complicated with the lardaceous and fatty liver.

b. *Atrophy.*—Atrophy of the liver, independent of the *marasmus senilis* of the organ, appears in various forms. We first draw attention to two distinct forms which have not been remarked hitherto, and which, similarly to the hypertrophic affections, are the expressions of a constitutional malady, and have their immediate origin in anomalies of the blood. Owing to their distinctive coloring, they may be appropriately termed yellow and red atrophy.

a. Yellow atrophy.—This affection is characterized by the saturated yellow color, owing to a diffusion of bile throughout the tissue, by extreme flabbiness and pulpiness, loss of the granular texture, extreme rapidity in the reduction of size, which chiefly affects the vertical diameter, and consequently induces a flattening of the liver. It occurs chiefly in the

early years of life, during puberty, and in the prime; it is remarkable for the rapid course it runs, for extreme tenderness of the liver, nervous attacks, and jaundice; it terminates fatally with febrile symptoms of a disorganized state of the blood, irritation of the brain and its membranes, and hydrocephalic softening of the former, and with symptoms of exudation and suppuration generally, and especially of the mucous membrane, pneumonia, &c.

The blood contained in the large vessels of the liver, and even that contained in the trunk of the vena portæ, is reduced in consistence, and of a dirty reddish-brown color; and the coats of the latter vessel are tinged with bile. This points to the fact that the portal blood itself contains such an excess of biliary constituents, that they are separated here, and still more in the capillaries, and thus fill the entire vascular and biliary system; the coats of the vessels and their cellular strata thus absorb bile by exosmosis, the true glandular tissue fuses, is lost in the biliary colliquation, and disappears. The immediate consequences of this condition are that the blood in the vena cava is infected and overcharged with bile, causing intense jaundice; when this has reached a certain point, the above symptoms terminate in a rapid consumption of the blood and in exhaustion. We commonly find biliary matter of a deep yellow color, or if the disorganized blood has exuded through the mucous membrane, a black tarry substance in the intestine.

β. Red atrophy.—This is distinguished from the former by its dark-brown or bluish-red color; the liver is gorged with blood, and presents a spongy elastic consistency; there is an absence of granulation, and a section offers an appearance of perfectly homogeneous texture; the organ is reduced in size, though its thickness preponderates over the other dimensions.

The disease is chronic, and is always accompanied by torpor of the abdominal ganglia, venous plethora of the abdominal viscera, and by the formation of brownish-black, or greenish-black, tarry bile, and fæces of a similar constitution. By itself it rarely proves fatal, though death may ensue from the marasmus brought on by the enduring congestion of the portal system. In addition to these two forms, we consider—

γ. Laennec's cirrhosis in its advanced stage, a chronic affection which resembles acute yellow atrophy, but besides being chronic, is distinguished from the latter by the liver being firm, or, if flabby, very tough.

Granular liver is a variety of this species; it appears essentially as secondary textural degeneration, and although commonly treated of as atrophy, and from ignorance of the above described forms as the only variety of atrophy, we refer for a minute examination to a subsequent portion of this work. Finally, we have—

δ. Atrophy of the liver from obliteration of the ramifications of the vena porta (vide, the acquired Lobular Form of the Liver, p. 103).

§ 3. *Abnormities of Form.*[1]—These abnormities are either congenital, and are then in part fœtal conditions of the liver, in part acquired. To the former belong the round, the unlobulated, or but slightly lobulated (embryonic) liver, the semiglobular, the broad, the flattened, the triangular and quadrangular, and multilobular liver.

[1] Oestr. Jahrb. xx. 4.

The acquired irregularity of form is either the result of external influences, or it depends upon an affection of the tissue of the liver. The former consists in a flattening of the liver anteriorly, in indentations or furrows, produced by contractions or deformities of the thorax, by stays, exudations, enlarged viscera, or morbid growths. The latter are of peculiar interest, as the nature of the hepatic malformation, taken in connection with the increase or diminution of size, is characteristic of the internal affection of the viscus. We shall devote some further consideration to this class.

Malformations of the liver must be considered in reference :

Firstly, To the relation of the vertical to the longitudinal and transverse diameters, or the circumference of the edges ;

Secondly, To the condition of the edges, which may be bevelled off, thinned, acuminated, or thickened, enlarged, and rounded ;

Thirdly, To the state of the surface, which may be variously smooth and level, or as variously uneven.

With reference to the first variety, we are able to affirm that the development of the vascular tissue generally, is connected with swelling and enlargement of the liver and with a preponderance of the vertical diameter (thickness); that the so-called development of the yellow tissue (infiltration) is complicated with lateral enlargement, or increase of size with flattening, and corresponding diminution of the vertical diameter.

In reference to the edges, we have to remark that in the last-named states, at least in their advanced degrees, they are absolutely thickened and rounded.

We find the following irregularities of form to occur more particularly in connection with the above-mentioned varieties of enlargement.

1st. When the increase of size is the result of congestion, or of temporary hyperæmic turgor, the liver retains the general outline of its normal condition : but if this affection becomes permanent, the vertical diameter soon predominates considerably. This is still more the case in genuine hypertrophy.

2d. The nutmeg liver, the fatty and waxy and lardaceous liver, induce a lateral enlargement of the organ : the vertical diameter diminishes, and the liver is flattened : this becomes more apparent when, as in the higher degrees, there is at the same time, an increase in the substance of the edges, i. e. when the latter become thicker and globose.

An evident exception occurs when this condition takes place in early life, or when it is congenital. The above-mentioned irregularity of form is in that case less marked, as the preponderance of the vertical diameter of the liver is normal in the fœtal state and during the first years of life.

Even in the varieties of atrophy of the liver, the remarks made as to the alterations of form, are confirmed in the main ; in the yellow variety the liver is generally reduced in its vertical diameter, whereas in the red variety, the decrease is chiefly perceptible at the edges, and the vertical diameter consequently predominates ; in the former case the organ presents a disk-like shape, in the latter that of a hemisphere or ball.

The irregularity of form consequent upon that textural disease which is called the granular liver, is very remarkable. It is almost always

accompanied by a considerable diminution of size; the granulations and the atrophy generally commence at the edges, and the latter attains its extreme development at this point; the edges consequently appear very much thinned, and at last form a mere seam, consisting of cellulo-fibrous tissue, which is contained between two condensed laminæ of peritoneum, and reflected over the convexity, or inverted into the concavity of the liver. The left lobe of the liver is frequently shrunk into a very small, flattened, cellulo-fibrous appendix, and the thick hemispherical or globular mass of the right lobe represents the entire organ.

Occasional exceptions arise from the granular disease being developed in a liver that was previously affected by some other disease, as by the fatty degeneration; in this case the reduction in size only takes place very slowly, and the edges instead of being thinned down, are often thickened and rounded.

The more violent inflammations of the hepatic peritoneal lamina, affect the surface-layer of the liver, and thus induce changes in form, that vary in proportion to the intensity of the inflammation. Thus the liver is not unfrequently converted into a thick cake with rounded edges, if the inflammation has been uniform, or it may be converted into a globular mass, compressed into a small space by peritoneal investment, which, in consequence of repeated attacks of inflammation, is transformed into a fibro-cartilaginous tissue. A malformation which we shall have occasion to revert to subsequently (superficial lobulation) results from an intense development of this process in detached spots.

The surface of the liver offers several points for consideration.

Hyperæmic turgor, and still more all the varieties of hepatic infiltration, are distinguished by their producing a smooth surface.

Unevenness of the surface is produced in various forms and degrees; the chief forms are the racemose and the lobulated.

The racemose form appertains to the granular liver; it depends upon the granulation of the peripheral layer, and appears delicately or coarsely moulded, of partial or uniform occurrence, in proportion to the development of the acini.

The lobulated liver is either a congenital abnormity or an acquired malformation.

The congenital form of this affection is owing to an arrest of development; the liver is divided into several lobes, and this division may proceed so far as to present several small livers which are only connected with the main organ by peritoneal folds and the vessels enclosed in them. This condition is not accompanied by any perceptible shrivelling or condensation of the peritoneum in the fissures or sulci, and still less by a condensation of the parenchymatous cellular tissue, or an obliterated state of the vessels. We may assume *à priori*, and experience confirms the view, that the lobulation commences and is chiefly, if not exclusively, developed on the concave surface of the liver, as the natural point of departure for the fissures.

Acquired lobulation of the liver presents itself in various degrees, and depends upon various causes. We base our division upon the latter, and thus arrive at their chief varieties, which at the same time, represent as many degrees.

Very superficial lobulation, one of which there is a mere indication, is occasionally the result of superficial inflammations affecting the hepatic sheath. These induce fibrous condensation of the parenchymatous cellular tissue, and cicatriform contraction of the investing peritoneum, beyond which the neighboring parenchyma projects in the shape of shallow, convex, and smooth protuberances, circumscribed by slight furrows.

A second form, in which the lobulation is more marked, is developed in the granular liver. In the same manner as the granulations may produce a racemose appearance of the hepatic surface, they may, when several of them are grouped together, produce larger protuberances, or lobes; if the interstitial cellular tissue is much condensed, the peripheral groups may become pediculated, so as to resemble mere appendices.

The third form and highest degree, which bears most resemblance to congenital lobulation, results from the obliteration of one or more branches of the vena portæ, from inflammation and the consequent shrivelling and atrophy of the hepatic sections supplied by their ramifications. These sections shrink in the direction of the obliterated trunk, the peritoneum generally follows, the surface is affected, and fissures result, which run in various directions, and above which the healthy tissue projects in the shape of large rounded protuberances. The enlargement of these protuberances appears to be encouraged by the additional labor thrown upon them, and still more so if these portions have become the seat of fatty and other infiltrations.

Irregularities of the hepatic surface of a different kind are induced by the development of adventitious products, such as cancer in the liver; but these will be discussed hereafter.

§ 4. *Abnormities of Position.*—Abnormities of position are either congenital or acquired. To the former belong the abnormal position of the liver, external and internal to the abdominal cavity; as in cases of fissure of the abdominal parietes and eventration, of deficient diaphragm, of congenital umbilical hernia, of lateral transposition of the viscera. In the latter case, the entire relations of the organs have undergone a corresponding change, the large right lobe now being on the left side, and *vice versa*, and the vesical fossa to the left of the umbilical fissure.

Some of the acquired malpositions of the liver resemble the former, as in the case of extensive wounds of the abdominal parietes, and of the diaphragm, and of certain rare anomalies, resulting from acquired umbilical hernia. A more common occurrence is the abnormal position of the liver within the abdominal cavity, in consequence either of pressure exerted by other viscera, or of a change in the size and weight of the organ. We find the liver and the neighboring organs pushed out of their proper place by distortions of the spine; by hypertrophied neighboring viscera, e. g., the right kidney, by expansions of adjoining cavities, as of the pericardium, but more especially of the right pleura. In the latter case it is forced down into the mesogastric region by the diaphragm which is depressed by the accumulation of gases or fluids in the pleura; and as the pressure especially affects the right lobe, this portion occupies the lowest position, and comes to be placed under the left lobe.

The liver may be pushed upwards into the concavity of the diaphragm and into the thorax, by gaseous accumulations in the abdominal cavity, by ascites, by peritoneal effusion, and by tympanitic distension of the intestines. It is as variously affected by partial exudations and by morbid growths, and the change of position corresponds to their seat and magnitude.

The spontaneous change of position which the liver undergoes in consequence of increase in size and weight, is invariably a descent to a lower region of the abdomen, and it follows from the anatomical relations of the parts that it must be the right lobe which is peculiarly involved.

§ 5. *Changes of Consistency.*—As these changes are always allied to other anomalies of more importance, and have therefore been already alluded to, or will be subsequently considered, we here only advert to the diminution in the consistency of the organ which takes place without any change in the hepatic tissue, in all dyscrasic processes accompanied by decomposition or subsequent to excessive elimination of the fibrine of the blood, as occurring in typhus and typhoid states, in purulent infection of the blood, and acute tuberculoses, or subsequent to extensive exudation on serous membranes, and especially in puerperal fever. The liver appears flabby, collapsed, and pultaceous; its parenchyma is softened and infiltrated with serum, generally very pale and exsanguine, or containing only pale, thin, and watery blood.

§ 6. *Diseases of the Tissues. a. Hyperæmia, apoplexy, anæmia of the liver.*—Hyperæmia of the liver appears in three forms : as active hyperæmia, resulting from idiopathic or consensual irritation ; as passive hyperæmia, dependent upon torpor in the portal vascular system ; and lastly, as mechanical hyperæmia, chiefly induced by obstacles in the circulation through the heart and lungs; the last form is one of very frequent occurrence, and is marked by the intensity and extent to which it affects the entire viscera. In rare cases an anomalous anastomosis of the epigastric cutaneous veins with the umbilical veins which have remained permanently open, gives rise to persistent hyperæmia of the liver. (Vide Veins.)

The anatomical signs are congestive turgor of the viscus, increase of size, especially in the vertical diameter, but without any further change of form, dark-red color, and obliteration of the yellow substance, softening of the parenchyma, and a large supply of blood. In habitual, and particularly in permanent mechanical hyperæmia, the vessels in the liver, as well as the trunk of the vena portæ, and the branches from which it arises, are found dilated and varicose.

Habitual hyperæmia of the liver is apt to be followed by hypertrophy; and as a consequence of an increased production of portal blood, and an exaggeration of its peculiar qualities, the nutmeg-liver may result, which again, may give rise to granular degeneration of the organ.

Apoplexy of the liver is a very rare occurrence ; it results from congestion which has rapidly attained a very high degree, and undoubtedly commences as capillary hemorrhage ; an apoplectic spot is thus caused,

which may enlarge and induce a rupture of larger vessels. According to the seat of the hemorrhage we find two varieties, viz., peripheral or deep-seated hemorrhage; both may however occur simultaneously. In the former, the hepatic peritoneum, especially that investing the convex surface of the right lobe, is detached in a varying extent, and underneath it is found fluid or coagulated blood to a larger or smaller amount. These hemorrhages occur chiefly in infants, as a consequence of impeded respiration and pulmonary circulation, from suffocative catarrh. The hepatic peritoneum may become ruptured, and thus cause an effusion of blood into the abdominal cavity. The liver is in a state of permanent congestive tumefaction, and being overcharged with blood, presents a dark-red color, and looseness of texture. We are reminded by these effusions of the analogous bleedings at the cranium, accompanied by a detachment of either the pericranium or the dura mater, which constitute the so-called thrombus or cephalhæmatoma.

In the second variety, apoplectic spots of various forms and sizes are found in the parenchyma; there are generally several of them dispersed through the organ. This variety is found more frequently in adults than the former, but the two may take place at the same time. If a cure follows, a cellulo-fibrous callous cicatrix remains.

Anæmia of the liver is the result of hemorrhages, exhaustion, or a reduction of the mass of blood by extensive exudative processes, and is accompanied by a diminution of the consistency of the liver. It is also constantly associated with many hepatic diseases, such as the fatty, the lardaceous, and waxy liver, to which we have already adverted.

b. Inflammation of the Liver (Hepatitis).—Although inflammation of the liver may not be a very rare affection, it is certain that the intense degrees, which terminate in suppuration and abscess, do not occur very frequently with us. We may remark that the most various diseases of the hepatic tissues are at the bedside taken for hepatitis.

If we sum up the observations of solitary instances of well-marked hepatitis, taken in connection with the condition of the hepatic tissue surrounding wounds and recent abscesses of the liver, we find the following to be the anatomical signs of hepatitis previous to its termination in suppuration:

Inflammation never attacks the entire organ, but occurs in one or more patches. Commonly there is but one spot, but it may vary in extent, and the process is here found developed in various degrees. The viscus is swollen in proportion to the number and size of the inflammatory patches, and this tumefaction is particularly perceptible when a section is made, the turgid tissue rising above the edges of the incision and the peritoneal sheath. The parenchyma is loosened and lacerable, and the structure becomes more apparent from the enlargement of the acini, which gives the broken surface a granular appearance; the acini become altered in shape, and assume an oval form; their circumference becomes transparent, so that each acinus seems imbedded in a gray or grayish-red layer of gelatinous matter, with which it is however intimately blended. In the advanced stage of inflammation, the granulated structure disappears, the tissue seems perfectly uniform, and the broken surface has a laminated appearance. The organ has a paler color, and

it is almost uniformly brown, or grayish-red in some parts, or yellowish-red or pale-yellow in others. The capillary vessels are filled with albuminous and fibrinous coagula.

If the process extend to the circumference, the peritoneal investment becomes opaque, thickened, and is easily detached; in many cases it is inflamed, and covered by an exudation of varying thickness.

Acute inflammation frequently leads to suppuration of the parenchyma and to hepatic phthisis. We then find small spots of pus occurring here and there in the infiltrated tissue, which gradually increase, coalesce, and form an hepatic abscess. The large abscesses found in the dead subject may almost always be proved to have resulted from a union of several smaller spots, by the remains of the fistulous passages that connected them, by the sinuous shape of their circumference, or by the débris of the former partitions.

The size of hepatic abscesses varies. They are often of the size of a fist, or a child's head, and may even occupy an entire lobe.

The seat of the abscess corresponds with the seat of the previous inflammation; it therefore most commonly occupies the right lobe, is generally found in the deeper parenchyma, and is often accompanied by an abscess in the left lobe, or extends into the latter.

The recent abscess represents an irregular cavity with uneven parietes, which are infiltrated with pus and consequently very friable; prolongations of the same tissue project into the cavity.

The abscess increases by fusion of the adjoining tissue, and thus assumes a round form, which becomes sinuous if a communication is established with other abscesses.

When the suppurative process has reached the boundary of the original inflammation, it meets, if no further inflammatory reaction is established in the vicinity, with infiltrated, tumid, and discolored parenchyma. In this manner the abscess may remain passive for a considerable period, retaining the shape and other characters above described. It is commonly lined by a suppurating and loosely-attached membrane. In reference to its contents, the hepatic abscess presents considerable differences at different periods, depending in part upon the communication established with the biliary vessels. The pus contained in the recent abscess is mixed with little or no bile, as the acini and the capillary gall-ducts have become obliterated by the inflammation; the bile contained in them at the commencement of the inflammatory attack, is at most found in combination with the pus. A large abscess of long standing, invariably contains pus mixed with a considerable amount of bile, which arises from the communication established between the cavity and larger gall-ducts. These are, like the bronchi, affected by a continuation of the suppurative process, and are generally eaten across in a transverse or slanting direction; and in exceptional cases only, and in very large abscesses, are they attacked and opened laterally. The pus contained in old abscesses is always discolored, generally greenish, and possessing a strong ammoniacal odor: we must undoubtedly attribute to it the extensive discoloration of the surrounding parenchyma. The bloodvessels opening into the abscess are blocked up, so that hemorrhage very rarely occurs.

Before a fatal issue takes place, the hepatic abscess may discharge its

contents in different directions, and with various results. The discharge is very rarely effected into the peritoneal sac, as from the peritoneal investment having been either primarily or secondarily involved in the inflammatory process, adhesions will have been formed, which prevent this occurrence. We have to notice the following modes of discharge:

α. The hepatic abscess induces suppuration in and between the thoracic and abdominal parietes, and after a communication has been established between the former and the superficial abscess, it discharges externally by straight or sinuous, narrow or wide passages; and by this means a cure is sometimes brought about.

β. The diaphragm may be perforated, and a discharge be effected into the right pleura, where, sooner or later, fatal inflammation is set up; or if the lung had previously been agglutinated to the diaphragm, suppuration of the pulmonary lamina of the pleura follows, and an opening being effected into the bronchi, pneumonia and pulmonary abscess supervene.

γ. The hepatic pus may be eliminated by the bronchi.

δ. The contents of the abscess may be discharged into the stomach, the duodenum, and the colon; and in these cases the hepatic abscess is reported to have healed.

ε. A discharge may take place into the gall-bladder, or more frequently into one of the larger branches of the hepatic duct, the hepatic pus is conveyed to the intestine by a longer passage, and thus escapes.

ζ. Cases in which the central aponeurosis of the diaphragm is perforated, and the pus discharged by longer or shorter sinuses into the pericardium, inducing pericarditis, are very rare. They have been observed by Smith and Graves, and once by ourselves.

η. Finally, very rare cases have occurred in which the hepatic abscess has discharged itself into large vessels, such as the vena cava; we have observed a case in which a communication was established between an hepatic abscess and the vena portæ and duodenum.

A cure of the hepatic abscess is effected after the pus has been discharged by one of the above-described methods, or it may result without this occurrence from more or less complete absorption of the pus by the cellulo-vascular membrane investing the sides of the abscess; for, as soon as that portion of the parenchyma which has undergone purulent infiltration is entirely broken down, the abscess comes in contact with a surface of tissue which is in a less inflamed state, or which does not put on any reaction till now. This, however, gives rise to an exudation, which invests the smoothed surfaces of the abscess, and after being repeatedly redissolved, at last forms a permanent coating. The subjacent layer in the interim has been converted into fibro-cellular tissue, and the cellulo-vascular investment becoming incorporated with the former, induces a gradual absorption of the enclosed pus, the walls of the abscess gradually approach one another, and at last unite to form a callous cicatrix. Not unfrequently a remnant of pus, which is converted into a cheesy concretion, and gradually becomes cretified, may still be found locked up in the tissue of the cicatrix; the parenchyma, lying above the situation of the original abscess, is found collapsed; and if the abscess extended to the

circumference, the hepatic peritoneal lamina forms a cicatrized, dense, shrivelled covering.

The true glandular tissue of the acini, and the interlobular tissue, are undoubtedly to be considered as the seat of the inflammation we have just examined; it must be carefully distinguished from inflammation of the capillary gall-ducts, as well as from abscess resulting from suppuration in the latter, which is characterized by its large admixture of bile. We shall advert to this form in connection with diseases of the gall-ducts.

In the same manner we have to distinguish between the hepatic abscess above described, and secondary or metastatic purulent deposits.

Induration and obliteration of the hepatic parenchyma are the more frequent result of slight and chronic inflammatory attacks. The product of inflammation solidifies, and the hepatic parenchyma becoming oblite-rated, is converted into a cellulo-fibrous callosity, which gradually contracts, and induces a collapse at the surface of the liver proportionate to its vicinity to the surface. If this occurs simultaneously at several points, the surface of the organ obtains an uneven, undulated, and slightly lobulated appearance. These accumulations of cartilaginous tissue are to be distinguished from the obliterations and atrophy which affect the hepatic tissue, as a result of obliteration of the portal ramifications consequent upon phlebitis.

The investigation of true chronic inflammation of the liver offers still greater difficulty, inasmuch as, in the dead subject, we generally have to deal with its products only, in various degrees of development; many cases of the so-called granular liver are probably referable to this head. At the bedside, the most heterogeneous conditions when accompanied by tedious and oppressive morbid sensations and by painful symptoms, especially by enlargement, are diagnosed as chronic inflammation of the liver.

c. *Inflammation of the vena portæ.*—This is, under all circumstances, a very important affection. It occurs both in a primary and in a secondary form, and may in either lead to obliteration or suppuration, and may attack the trunk and the ramifications of the vessel, or the latter only.

Inflammation ending in obliteration of the branches of the vena portæ within the liver demands a special notice, as it occurs very frequently, although we rarely have opportunities of investigating it in the dead subject otherwise than in its termination and its consequences. It would appear to be owing to an anomalous condition of the portal blood, and to belong to the adhesive form. Several cases that we have observed, in which irregular anastomoses were discovered between the portal and the general venous system, by means of the patulous umbilical vein, seem to authorize this view.

Under certain indented and contracted parts of the surface of the liver, we discover an accumulation of cellulo-fibrous callous tissue, which, on more minute examination, is found to conduct to a larger or smaller portal branch, with which it is connected. The vessel itself is converted into a ligamentous cord, or it is plugged up with a fibrinous, cheesy, or calcareous deposit.

The consequences of the obliteration are, atrophy of that part of the

liver which is supplied by the ramifications of the vessel, lobulation of the liver, as described at page 103, and in extreme cases, ascites.

d. Deposits, metastases in the liver.—Metastases occur in the liver under the same conditions under which they take place in the lungs. They are, however, much less frequent in the former than in the latter and in the spleen; and the so-called hepatic abscess, more especially consequent upon important surgical operations, wounds and injuries of the cranium, is found much more rarely than has been hitherto supposed. Besides, we always simultaneously discover deposits in other organs, particularly in the lungs and the spleen. We are unacquainted with the special conditions which give rise to a predominant deposit in the liver, with the exception of those cases in which the source of the poisoning of the blood is within the compass of the portal system.

The deposit in the liver is also caused by the deposition or exudation of fibrin through the coats of the capillaries into the tissue, or by the coagulation of the blood in the capillary rete of vessels. In both cases metamorphoses may ensue which vary according to the nature of the morbid essence absorbed into the blood; occasional induration and shrivelling are induced, with consequent obliteration of the parenchyma and the capillaries; more frequently purulent or ichorous fusion result, and then either suppurative inflammation of the surrounding parenchyma is established, or a solution of the coats of the capillary vessels is effected.

The deposit presents, as in the lungs, the appearance of a circumscribed nodulated accumulation, of a dark-red or brownish-red color, which, as it approaches the state of fusion, is converted into a dirty yellow or greenish color.

The deposit has a rounded form, varying in size from that of a pea to that of a walnut; it is found in considerable numbers, and is commonly seated in the peripheral layer, where it gives rise to inflammation of the hepatic peritoneal lamina. This is a guide to distinguish it from the abscess which originates in idiopathic inflammation of the liver; the diagnosis is also aided by the acute course of the affection, by its originating in another morbid affection, by the typhoid symptoms, by the occurrence of similar processes in other organs, more especially in the lungs and the spleen, by the disorganization of the blood, and the resulting jaundice.

e. Gangrene of the liver.—Gangrene of the liver is very rare, in fact Ferrers and Bérard deny its occurrence, but we have seen it in one well-marked case, associated with pulmonary gangrene. It is developed in parts affected with inflammation and suppuration, not so much as a result of intense inflammation as of certain peculiar conditions, which cause a tendency to gangrenous degeneration. It occurs in more or less circumscribed spots, in which the parenchyma is dissolved into a brown or greenish-black pulp, which diffuses the characteristic odor of sphacelus. We find suppuration in the vicinity, which is the product of reactive inflammation, and which defines the boundaries of the mortified part.

f. Granular liver.—Granular liver is one of the most important, though in many respects, and especially in reference to its pathogeny, one of the most enigmatical affections of the liver; it is termed by Laennec cirrhosis: older authors have considered it identical with or related to

scirrhus; and if viewed in reference to its termination only, it may be called induration of the liver.

It undoubtedly presents many degrees, which merge into one another; from the very unsatisfactory state of our knowledge, however, in reference to the elementary process and fundamental nature of the disease, we consider it necessary to sketch the affection as seen in a marked case, without any further complication, and subsequently to state what is known of the earlier stages of the disease, and of the later metamorphoses of the organ.

In a case of the kind alluded to, the viscus appears considerably diminished in size, and this decrease is accompanied by a characteristic change of form. The margins are thinned down to such a degree, as to represent a cellulo-fibrous seam, which is folded upon the remainder of the organ; the vertical diameter of the liver has increased, and is found to consist chiefly of the hemispherical or globular right lobe. (Vide p. 103.)

The external surface presents a granular, warty, racemose appearance, which results from the projection of the peripheral so-called granulations, of the liver. These granulations may all have the same size, e. g. that of a hemp-seed, and the surface then is uniformly racemose: or they vary in size, and the surface is then unevenly racemose.

The hepatic surface intervening between the granulations is of a dull white color, tendinous, shrivelled, and contracted; the granulations are thus circumscribed, separated from one another, and even occasionally pediculated.

The viscus, when it has this appearance, is to a certain extent elastic and tough, and even indurated, so as to offer a cartilaginous resiliency; it cannot be broken, as it possesses the tenacity of leather.

The scalpel itself confirms the fact of induration, as the instrument meets with a scirrhoid substance, which may even cause a crunching sound.

A section shows the above-mentioned granulations to be either isolated or grouped together; an accumulation of dirty white, dense, resilient cellular tissue, which is almost destitute of bloodvessels, and which forms a nidus for the former, is seen between them.

The color of the organ is variously modified; being dependent upon the color, either of the granulations, which we shall have still further to examine, or of the intervening fibro-cellular tissue.

The liver is frequently attached to adjacent parts, especially to the diaphragm, by means of cords or laminæ of new matter; the adjoining peritoneum, and especially the peritoneal covering of the gall-bladder, and the folds which leave the liver, are opaque, shrivelled, and tendinous.

The granulations have given rise to the name of granular liver; and from the coexisting atrophy and diminution of size, the affection is also termed granular atrophy of the liver.

The granulations are the most prominent sign in the sketch we have given, and the question arises as to their nature.

Laennec viewed the granulations as an adventitious product, and as his specimens offered a yellow color, he termed it cirrhosis (κιρρός, fulvus).

One may easily be convinced of the incorrectness of this view, as a

careful examination at once proves that the granulations consist of nothing but hepatic parenchyma, which, however, as we shall subsequently have occasion to show, is variously modified.

It follows from our demonstration that in granular liver the hepatic parenchyma has become reduced to the granulations, and that the portion which has disappeared, has been replaced by fibro-cellular tissue.

The desire to obtain more accurate views as to the nature of the granulations and their mode of origin, has caused the promulgation of various doctrines which are untenable or incomprehensive in proportion as their authors attached too much importance to the ideas of hypertrophy and atrophy and their combination, or attempted to construct a theory from isolated observations, or because they did not sufficiently distinguish between the diseases of the hepatic parenchyma preceding the formation of granulations, and those affecting the granulations themselves, and other morbid conditions not essentially connected with them.

According to Bouillaud, with whom Andral coincides in the main, the granulations are the result of hypertrophic development of the so-called white or secreting substance, accompanied by obliteration and gradual atrophy of the red or vascular tissue.

Cruveilhier advocates a different opinion. He thinks that cirrhosis consists in the atrophy of a considerable number of the hepatic acini, accompanied by hypertrophy of the remainder, which, as it were, take the place of the former.

We pass over the unsatisfactory and erroneous doctrines of other writers, which are based upon investigations of solitary cases, or of anomalies in the elementary tissue, and merely remark, that we do not adopt any one of the above views exclusively, as they do not appear to us to embrace the entire characters of granular liver.

Granular liver presents considerable varieties. The granulations themselves offer numerous variations in reference to texture, number, size and form.

With regard to their texture, we sometimes find that they consist of normal, or at least tolerably normal, hepatic parenchyma. Commonly, however, this is not the case; the parenchyma of the granulations is itself abnormal, and variously diseased; such cases render the analysis of the hepatic granulations difficult, and cause errors in the conclusions arrived at, as not sufficient attention is paid to the distinction between the essential and non-essential characters of the abnormity. The alterations of tissue in the granulations are either such as constitute the *causa proxima* of the entire metamorphosis, i. e. they are essential, or they are mere accidental complications, which may either precede or accompany the formation of granulations. As we shall subsequently have to show the development of the granulations from the former, and as we are also compelled to examine into the complications of granular liver, we here give a summary of the abnormal conditions, without reference to the above distinctions.

Firstly. The parenchyma of the hepatic granulations occasionally presents a coarse-grained hypertrophy of the acini, the granulations pro-

jecting on a sectional surface in the shape of dark reddish-brown and elastic points.

Secondly. It frequently appears in the various degrees of the nutmeg liver (Laennec's cirrhosis of a low degree).

Thirdly. The granulations appear in the shape of rounded or lobular convolutions of dilated, turgid, yellow, gall-ducts, the red vascular substance in the vicinity having disappeared. This yields one of the commonest and most exquisite forms of the granular liver; it is genuine cirrhosis, which originates in the first variety of the nutmeg-liver, dependent upon stasis and dilatation of the biliary ducts. The majority of authors have evidently taken their description of granular liver from specimens of this kind.

Fourthly. The parenchyma of the granulations is frequently infiltrated with fatty matter or similar products, and the granulation then presents on a small scale all the signs discussed at page 98. Gluge has evidently employed a specimen of this description for his investigations.

Fifthly. We occasionally find the granulations in the condition of what we have termed yellow acute atrophy; they are then yellow throughout, and appear at the surface and on section as pulpy, collapsed, friable, yellow masses.

Sixthly. The parenchyma of the hepatic granulations frequently presents symptoms of an inflammatory condition; it then appears pale, of a homogeneous structure, with obstruction of the small biliary canaliculi, commencing induration and obliteration.

The granulations vary much as to number, and are either uniformly distributed through the surrounding cellulo-fibrous tissue, or they coalesce into groups of various extent. The more numerous they are, the less the hepatic parenchyma is destroyed; the number of the granulations therefore indicates the degree of atrophy that has taken place, and, if we take the quality and quantity of the textural changes into consideration, the stage of the disease generally.

The size of the granulations varies from that of a pin's head to that of a horse-bean, according as a single acinus, or an entire lobule, or a large portion of the organ is affected; they are generally of a rounded form, though they are very frequently of an irregular and especially of a lobulated shape. In the majority of instances we find one size and form to prevail.

The cellulo-fibrous tissue intervening between the granulations, is either diminished or increased in amount. There is generally an inverse ratio between this tissue and the number of granulations; but we find exceptional cases in which the granulations are very numerous, and the interstitial cellular tissue is also much increased. The latter varies much as to density, resiliency, vascularity, succulence, and color. Sometimes it is loose, friable, vascular, more or less reddened, and succulent; at other times it is tough, less succulent, of a dirty gray or greenish color, at others again, dense, indurated, dirty white, of fibro-cartilaginous, scirrhoid, resiliency and elasticity, crepitating when cut, &c.

Having discussed the two constituent parts of granular liver, we must now examine into the origin of the metamorphosis.

We have seen that in granular liver the granulations represent the

persistent hepatic tissue, and that the parenchyma which has been re-moved is replaced by cellulo-fibrous tissue. The question arises whether this reduction is primary or secondary, and supposing the latter case, which is the primary anomaly? It is commonly set down as mere atrophy, in consonance with the view of the French observers above quoted.

We are not of opinion that granular liver always takes its origin in the same fundamental affection; we are inclined to adopt two morbid states as the essential and original anomalies, which give rise to granu-lations in the hepatic parenchyma as a secondary affection.

a. In one case there is a morbid development of the capillary gall-ducts (the so-called secreting tissue); an accumulation of the secretion, and probably also a hypertrophy of the parietes of those vessels giving rise to the nutmeg liver, and to an obliteration of the capillary blood-vessels, the so-called vascular substance. We then have to do with the gradual reduction of the organ, already described under the head of Atrophy, as an advanced stage of cirrhosis; in this condition granular liver takes its origin, for the granulations are formed by the biliary ducts coalescing into rounded fasciculi or coils of the size of a pin's head or hemp-seed. They are more or less of a yellow color, containing fat, and either solitary or collected into lobular groups; they are surrounded by a spongy, cellular, soft, succulent, red, and vascular tissue, from which they can only be separated by rupture of the latter and of its vessels. This anomaly is commonly met with in various degrees of development at different parts of the viscus; it is generally more advanced in the peripheral portions, the deeper portions presenting at the same time the appearance of the nutmeg degeneration; the liver is frequently enlarged, but certainly not diminished in size, and preserves the thick, massive edges peculiar to the nutmeg liver.

A secondary metamorphosis now gradually supervenes, the stage of obliteration and atrophy. The interstitial tissue gradually loses its vascularity, its red color, succulence, and spongy texture; it becomes more and more pale, of a grayish-red, and dirty white color; it shrivels up, and becomes denser and drier, coriaceous, and even of scirrhoid hardness; and it presents a cellulo-fibrous, fibro-cartilaginous structure. The granulations at the same time undergo important modifications. The obliteration of the interstitial tissue not only destroys the vascular connection between the latter and the granulations, but, as their nutri-tion becomes impaired, their secreting power also ceases. We now find the granulation enclosed in a cellulo-fibrous case, from which it may be easily removed, as it is only connected with its investment by a few de-licate cellular threads, or is even quite detached, with the exception of a single vascular pedicle; it is found collapsed, pulpy, of a dirty yellow color; it gradually diminishes in size, the surrounding tissue also becom-ing atrophied; it soon appears only as a minute yellow or greenish spot, and at last vanishes entirely. In exceptional cases, in which the liver has become so much indurated as to be incapable of further condensa-tion, the tissue surrounding the individual granulations is converted into a cyst with a serous lining, in which the granule floats, attached only by a vascular footstalk, and surrounded by a yellowish or pale green, watery,

or gelatinous fluid. In consequence of the vascular obliteration, it is gradually so much reduced as at last to present nothing but a minute nodule attached to the internal surface of the cyst, which is now entirely filled with the fluid.

In this variety, therefore, the original anomaly consists in the hepatic parenchyma being gradually reduced to the capillary gall-ducts which have assumed the shape of the granulations; and in so far as this is genuine cirrhosis of the liver, it certainly bears some resemblance to the pulmonary cirrhosis described by Corrigan. The secondary metamorphosis causes a gradual atrophy of the granulations, accompanied by a predominance of the interstitial cellulo-fibrous tissue, and a uniform diminution of the entire organ.

The degree attained by the metamorphosis is proportionate to the number of obsolete granulations, or to the amount of parenchyma remaining capable of performing its functions; the organ decreases in proportion to the shrivelling and condensation of the interstitial, cellulo-fibrous tissue; and it often appears reduced to one-quarter, or even one-sixth, of its ordinary size. The condensation of the cellulo-fibrous tissue, as it gives rise to a decrease of the organ, also induces a corrugation and shrivelling of the peritoneal investment. The latter will be more or less opaque, and thickened; and, being retracted between the projecting granulations, these not unfrequently appear to have a neck-like contraction. These changes in the hepatic peritoneal covering take place without any symptoms of inflammatory action.

The secondary metamorphosis chiefly affects the margin of the liver, and more particularly the left lobe. The organ very commonly appears to have been almost or entirely deprived of parenchyma, and to consist exclusively of fibro-cellular tissue, the edges more particularly being thinned off and turned back upon the body of the organ, the left lobe of which is converted into a mere appendix of fibro-cellular structure, of the size of a hen's egg or a walnut.

Not unfrequently the granulations assume, in the advanced stages, and after a long duration of the disease, a bluish or dark-green color, which particularly affects those seated at the concave surface of the liver.

This form of cirrhosis of the liver undoubtedly originates in hyperæmic states, a view that is confirmed by their frequent connection with organic disease of the heart: its frequent occurrence in drunkards also points to a peculiar anomaly in the constitution of the portal blood.

β. In the second case, the original affection of the hepatic parenchyma in granular liver is proved, by the post-mortem appearance of the granulations, to consist in a slow chronic inflammation. This induces a gradual obliteration of the parts attacked, and their conversion into fibro-cellular tissue, the amount of which varies in proportion as the processes of absorption or of organization predominate in the inflammatory product. This secondary metamorphosis, from not occurring uniformly, results in a subdivision of the organ into larger or smaller scattered compartments, which present the characteristic rounded form of the granulations in the same ratio as they correspond to single hepatic lobules. Their parenchyma is frequently found in the original state of chronic inflammation,

but it may be unchanged, or it may offer one of the other accidental anomalies alluded to.

It is intelligible that the diminution of size in this variety is often inconsiderable, that the organ may even be enlarged, and that the fibrocellular tissue is accumulated in such a manner as to preponderate over the parenchymatous cellular tissue. A marked decrease of size occurs when the obliteration is extensive and the cellulo-fibrous tissue has shrunk; and as this decrease advances, the pressure exerted by the shrivelled tissue upon the parts not originally affected by the anomaly, induces an atrophy in them; they fade, and put on a rusty or dark yellow color.

Granular liver frequently presents an abnormity which appears peculiar to this variety. We allude to the presence on the condensed peritoneal investment of pseudo-membranous formations, of a cellular or cellulo-fibrous texture, which generally extend to the diaphragm in the shape of corded adhesions. They are the result of inflammatory processes, which have become extinct long before the occurrence of the secondary metamorphosis, and which appear to afford evidence of the inflammatory nature of the hepatic disease itself.

Besides these two modes of development of granular liver, the affection may also be viewed as a retrograde process, manifested in depositions or infiltrations of the hepatic parenchyma, arising from an anomalous state of the blood.

In reference to the external conformation of granular liver, we have still to advert to a variety which is characterized by the hepatic parenchyma not being reduced to granulations, but continuing in large masses, the more superficial of which are pushed out by the shrinking interstitial tissue, and being more or less contracted at their base, cause the entire organ to appear lobulated.

Granular disease of the liver is found complicated with all the essential or accidental anomalies which we have described as occurring in the paranchyma of the granulations, and these anomalies may either precede the granular disease or supervene after its development. The complications may be hypertrophy, nutmeg liver, cirrhosis, adipose and other infiltrations, acute yellow atrophy, inflammatory and other hepatic diseases. The granular disease arising from one of the essential anomalies, e. g. from inflammatory causes, is more particularly liable to combine with another essential anomaly, as, for instance, with true cirrhosis.

The complication with adipose deposit is peculiarly interesting. The latter may,—

Firstly, be the primary affection upon which the granular disease is grafted in the shape of cirrhosis. As the cirrhosis advances, the reduction of the organ generally, but more particularly of the marginal portions which have been infiltrated with fat, is impeded, and the atrophy that does take place is characterized by its affecting the margin much less than in the uncomplicated form.

Secondly; the adipose deposit may supervene upon a granular state of the liver; and if it does so before the secondary metamorphosis has advanced very far, and whilst the granulations are still very numerous, it may prevent the liver from assuming the form peculiar to the granular

condition. If it occurs at a later period, it need not modify the characteristic form of the organ.

Thirdly; the cirrhotic and shrinking granulation which is cut off by dense cartilaginous interstitial tissue may degenerate into a flabby, dirty yellowish-brown fat-lobule, the degeneration apparently proceeding from the confined biliary matter.

A similar relation exists in regard to the modifications of form between the granular condition and other infiltrations of the hepatic parenchyma.

Granular liver is also very frequently coincident with the most various morbid affections of the heart, which give rise to congestion in the vena cava and in the portal system; of these, hypertrophy, dilatation, and valvular disease are the most common. Disease of the heart must be considered as an important momentum in the origin of the hepatic disease.

The symptoms resulting from the granular state of the liver bear a ratio with the degree of its development; the impermeability and obliteration of its secreting tissue induce, on the one hand, congestion in the portal system, hyperæmic states of the intestine and of the peritoneum, a blennorrhoic condition of the former, tumefaction of its membranes, and ascites; on the other, dyscrasic conditions of the blood allied to scurvy and frequently accompanied by icterus, an inclination to exudative processes, with an especial proclivity to hemorrhage, anasarca, and anæmia.

We cannot admit that the relation existing between Bright's disease of the kidneys and granular liver, though the two often coexist, has been accounted for. In one set of cases both affections would seem to have originated in common causes; in another, Bright's disease is evidently of more recent date, and has supervened upon the existing granular state of the liver; but whether in this case it is due to a separate cause, or is owing to the dyscrasia accompanying the hepatic disease, we are unable to determine.

Granular liver is invariably a chronic affection, which may often be arrested in its development for a short time, but never permanently. It terminates fatally by inducing anæmia and tabes complicated with dropsy; by disorganization of the blood, by exhausting and paralyzing exudations on the serous membranes, and especially on the peritoneum. It rarely occurs before the prime of life, but we have seen one case of it at the age of seventeen.

g. Adventitious growths. α. Anomalous production of fat.—This occurs in two distinct forms. We have already become acquainted with one in the shape of adipose deposition, or infiltration of the hepatic tissue with free fatty matter; the second is very unusual, and appears as a lipomatous morbid growth of a rounded or lobulated form, and rarely larger than a pea.

β. Cavernous tissue.—This is remarkable from its frequent occurrence in the liver. It resembles the tissue of the corpora cavernosa, and is commonly found in the peripheral substance of the liver only; from its dark-blue color it shines through the peritoneum, and the affection is therefore recognized on the external examination of the organ. It varies

in size, from that of a hemp-seed or pea to that of a hen's egg, and more; is generally irregular in form, and its cells contain a large quantity of dark blood; a connection may be always traced between the latter and some larger portal vessel. According to the amount of blood contained in the compartments, these are found in the dead subject projecting beyond the surface of the liver, or collapsed and sunk. Sometimes they are single, sometimes numerous.

γ. Cysts.—The liver is more liable to the formation of encysted tumors than any other parenchymatous organ; and we repeat that the rarity of tubercular deposit in the liver enhances the importance of the hydatid theory. We find in the liver—

aa. The simple serous cyst, a serous sac containing a clear watery fluid; this is not met with as often as

ββ. The acephalocyst of Laennec; which in the first instance is merely a serous, but from acquiring a fibrous investment, is converted into a fibro-serous sac, containing, besides serum, the so-called acephalocysts; these are small bladders (hydatids), formed of coagulated albumen and filled with an albuminous fluid; they vary in size and number, and are either attached to the parietes of the former or float in the serum.

The acephalocyst generally attains a considerable size in the liver. We have several extraordinary specimens in the Viennese museum, and there is one of a foot in diameter. In proportion as the heterologous growth increases, the hepatic parenchyma gives way, and the nearer the former originally was to the surface, the sooner will it reach the peritoneal investment; it then projects above the liver, with a larger or smaller segment of its circumference. Under these circumstances the peritoneum invariably inflames, and the consequence is a thickening of the latter upon and in the vicinity of the acephalocyst; an investment of pseudo-membranous cellular tissue is formed, by which the viscus becomes attached and agglutinated to adjoining organs.

Sometimes there is but one, sometimes there are several of these cysts; in rare cases, the entire liver appears converted into an aggregation of larger or smaller sacs. In the latter instance, two or more are often found to communicate with one another; either in consequence of atrophy of their parietes from pressure, of rupture from inflammation, or from a sudden increase in their contents.

The right lobe of the liver is the ordinary seat of the acephalocysts; the largest are always found at this part.

Acephalocysts are liable to inflammatory attacks, which entirely resemble those of normal serous and fibro-serous membranes, both in regard to the exudations they give rise to, as to their terminations and consecutive results. They may, by causing suppuration and obliteration, destroy the vitality of the acephalocysts, and thus bring about a cure.

The hepatic acephalocyst may discharge its contents in various directions; the portion that projects above the surface of the organ, and has lost the support it previously received from the surrounding parenchyma, may become atrophied and thinned, or its tissue be weakened or destroyed by inflammation and suppuration, and thus communicate directly with the abdominal cavity; or having first become agglutinated to neighboring

viscera, it may perforate the latter and discharge externally, or into other cavities and canals. The contents may thus make their way

Into the right pleura, or into a pulmonary abscess, and be removed by the bronchi :

Into the intestinal cavity, and especially into the duodenum and transverse colon, so as to pass off by vomiting or defecation :

Into the gall-ducts, i. e. into a large branch of the ductus hepaticus, by which passage they may ultimately be conveyed into the intestine ; though the protrusion of the acephalocyst more frequently induces dangerous obstruction of the biliary passages :

In rare cases, into a neighboring bloodvessel, and lastly :

Into a neighboring circumscribed abscess, resulting from peritoneal inflammation.

Occasionally the acephalocyst opens in various directions at once. After the discharge of its contents, obliteration of the sac and cure, sometimes follow.

The contents of the sac are discharged unaltered or changed, according to the process accompanying its perforation ; the products of inflammation in the matrix, or of the parietes of other cavities (e. g. the pleura), the bile, the intestinal secretions, &c., are particularly prone to induce a maceration and complete solution of the acephalocyst.

On the other hand, not only the parietes of the investing sac are often found saturated with bile, but the bile extravasated from large gall-ducts is frequently mixed with its contents, and its parietes are incrusted with inspissated bile. In the same manner we may now and then discover blood in the cyst, which has been discharged from neighboring vessels.

The hepatic parenchyma is forced out of its position in proportion to the size and number of the cysts ; if otherwise affected, it presents the nutmeg degeneration.

Acephalocysts in the liver are frequently complicated with affections of the same kind in other organs, as the lungs, spleen, and kidneys ; the disease is also complicated with cancerous affections in other organs. Large acephalocysts of the liver give rise to ascites or peritonitis, and may thus prove fatal.

In reference to the etiology of these growths, it appears, according to some observations, that mechanical injury of the liver and intermittent fevers may influence their development. They seem not to occur before puberty.

δ. Tuberculosis of the liver.—Contrary to the received opinion, we assert that the liver is rarely the seat of tubercular disease. It scarcely ever occurs in this organ as a primary affection, but is not unfrequently found as a secondary complication of advanced primary tuberculosis in another organ, or of universal tubercular disease. It must, therefore, almost always be considered as the expression of advanced tubercular cachexia.

Hepatic tubercle occurs in the shape of semi-transparent, grayish, crude, miliary granulations ; in which case it is more especially the product of acute tuberculosis ; or as yellow, cheesy, adipose deposits, of the size of a hemp-seed, or pea, or more. It is consequently often larger

than pulmonary tubercle; but, on the other hand, with the exception of very rare cases, is much less extensively disseminated than the latter.

Hepatic tubercle is not limited in its seat to a particular section of the viscus, but attacks all portions indiscriminately, and the more so, the acuter its course.

The tubercular matter is deposited in the parenchymatous cellular tissue of the organ, and especially in that pertaining to the biliary capillaries. It very frequently surrounds a minute gall-duct, and thus presents a central canal, which gives rise to a biliary discoloration of the nucleus.

When the liver is attacked by acute tuberculosis, its appearance resembles the parenchyma of other organs similarly affected; it is in a peculiar state of turgescence, the tissue is relaxed, friable and pale, and gorged with a serous or sero-sanguineous fluid. All this will be the more evident, the more rapidly the tubercular deposit is effected, and the more the universal cachexia is developed.

The conditions under which hepatic tubercle occurs, render it apparent that it rarely passes into the stage of softening, and scarcely ever into that of cretification; the constitutional affection generally proves fatal from its violence and diffusion, before the tubercles of the liver have undergone these metamorphoses. Still we do occasionally find that, from the very violence of the constitutional affection, a solution of hepatic tubercle is effected; and then it is probably the yellow variety which is converted into a primary hepatic vomica, and which offers no peculiar characters beyond the biliary discoloration of its contents.

We do not, however, meet with a condition accompanying tubercular suppuration in the liver which may be considered analogous to pulmonary phthisis.

This vomica requires to be the more carefully distinguished from morbid dilatation of the gall-ducts, as the latter not only occurs frequently or almost invariably, in combination with hepatic tubercle, but is not unfrequently coexistent with tubercular disease of other organs. In this case small cavities, of the size of a millet-seed or a pea, filled with viscid, muco-bilious, dirty green matter, with flaccid parietes, are found scattered through the liver, which on close examination are found not to be tubercular, but to be dilatations of capillary gall-ducts. The hepatic tubercles exist at the same time, and at various distances; a tubercle may occasionally be found near one of these cavities, but it is not characterized by the symptoms of secondary deposit accompanying the fusion of tubercular matter.

The conditions of their origin, and their connection with the constitutional disease, have not been as yet ascertained; but we are warranted by numerous observations in stating, that they invariably indicate a high degree of the constitutional affection; and a tendency to universal tubercular deposition, and especially in the abdominal viscera.

Hepatic tubercle may be complicated with tubercular affections of almost all organs, as might be assumed from its originating in an advanced stage of tubercular dyscrasia; however, the abdominal organs are found chiefly implicated, viz. the abdominal lymphatic glands, the spleen, the peritoneum, and the intestinal canal.

ε. Carcinoma of the liver.—Carcinoma of the liver is a disease of much greater importance than tubercular deposition, as it occurs very frequently and is often a primary affection.

Although we do not coincide with Cruveilhier, as to the frequency of its occurrence, it still must be considered as a common affection, and we would give its numerical relation to carcinoma of other organs as one to five. The greater frequency of its occurrence, as compared with tubercle of the liver, and considered in reference to the frequency of both affections in other organs, and especially in the lungs, and to the facts connected with the formation of cysts in the lungs and the liver, is a matter of particular interest.

These remarks apply to carcinoma of the liver generally, but not to its different varieties; of these, some are frequent, some occur less frequently, some very rarely.

Four varieties of carcinoma are found in the liver, which we will examine in succession.

αα. Areolar cancer.—This form occurs so rarely, that it is never described among hepatic affections. One case of very extensive areolar cancer has come under my notice.

ββ. Carcinoma fasciculatum sive hyalinum (Müller). Although not as frequent as the following, it undoubtedly occurs often. It is generally taken for medullary carcinoma, and the mistake is accounted for by the fact that the two often coexist. It forms masses of the size of a filbert to that of a man's fist, which are surrounded by an investment of delicate cellular tissue; though the surface is uneven and lobulated, the general outline is round; its consistency varies, being sometimes but slight, at others almost cartilaginous; its color a pale yellowish-red, and generally of almost vitreous transparency. The carcinomatous masses are commonly found in considerable numbers, and like medullary cancer, they cause rounded protuberances of the viscus, and produce an increase in its weight and size.

γγ. Medullary carcinoma.—This is the most common form of hepatic cancer, and almost all investigations that have hitherto been made in reference to this subject, treat of this variety only. It occurs either in the shape of detached masses, or as an infiltration of the hepatic parenchyma.

ααα. The detached masses occur as tumors, which offer many peculiar features.

Their general form is spherical, though their surface not unfrequently is slightly racemose or lobulated. Those which have been developed in the peripheral portion of the organ, and are therefore in contact with the peritoneum, present a flattened, or even an indented surface, and the indentation may extend to the very nucleus of the morbid growth. The peritoneal lamina in the indentation is opaque and thickened, owing, not as is commonly thought, to cartilaginous induration, but to an homologous cancerous degeneration of the serous and subserous tissue. This condition of the peritoneum is analogous to the relation the common integument bears to subjacent cancerous growths.

In size the medullary cancer varies from that of a millet- or hemp-seed to that of a man's fist, a child's head, and more. In most instances mor-

bid growths of various sizes are found in the same individual. The larger those are which occupy the peripheral portion of the organ, the more prominent will be the protuberances on the surface.

The number of these adventitious products varies equally; sometimes there are but few, or even only a solitary one is found; at others they are very numerous. The greater the number of those occupying the peripheral portion of the organ, the more numerous will be the protuberances on the surface. When the morbid growths are numerous and large, two or more often coalesce.

We are unable to discover any peculiarity in reference to their position; they commence equally in the peripheral and in the deeper-seated portions of the intestine. They commonly make their first appearance in the right lobe.

As regards consistence, we find two varieties which have also been considered as differing in texture. They do not, however, constitute essential distinctions, but are merely different degrees of development of the same morbid growth.

One is of the consistency of bacon, and presents on section a smooth homogeneous, shining surface, of a dull white color, and without a trace of bloodvessels. On pressure, a small quantity of a thick creamy fluid exudes from the meshes of a dense fibrous structure. These growths are not detached from the adjoining hepatic tissue without considerable difficulty; and a distinct cellular investment can scarcely be demonstrated. The growths belonging to this variety, when coexisting with the second, are always the smaller of the two.

The second presents the physical characters of true encephaloid disease; its general color is milk-white, it is more or less vascular, and consequently in part gray, yellow, brownish, red, or even dark red; it is very spongy, and on pressure yields a large quantity of a thin milky fluid, which is contained in the meshes of a friable, fibrous tissue. The tumors are invested by a delicate cellulo-vascular sheath, and are easily detached from the hepatic parenchyma. When occurring simultaneously with the first variety, they generally form the large morbid growths.

The latter evidently represent an advanced stage of the morbid growth, as appears not only from the foregoing remarks, but also from the relations of the primary cell. (Vide vol. i.)

$\beta\beta\beta$. Infiltrated medullary cancer is analogous to the other infiltrations of the hepatic tissue, which we have already discussed. It always contains obliterated and obsolete bloodvessels and gall-ducts, which are gradually absorbed. The infiltration attacks larger or smaller segments of the viscus; it does not present distinct boundaries, but insensibly passes into the normal parenchyma. It rarely occurs without nodulated cancer. The carcinomatous mass presents the same two varieties in reference to consistence and to its elementary constitution. We find a transition from the diffused to the circumscribed form in the fact, that the nucleus of the latter is sometimes infiltrated hepatic tissue, which becomes endowed with independent growth, and merely forces the parenchyma out of its place.

The larger and the more numerous the carcinomatous masses are, the

more extensive the cancerous infiltration, the more does the viscus increase in size and weight. The extracancerous tissue presents the nutmeg and adipose degeneration.

Medullary cancer is here, as elsewhere, the seat of hemorrhages, which are proportioned to the rapidity of its growth and the looseness of its texture. In rare cases it penetrates through the peritoneal investment of the liver, its development then proceeds with extreme energy, and it induces exhausting hemorrhages. In other cases it perforates the coats of large gall-ducts within, or of the biliary passages external to the liver, and grows into their cavities. In the infiltrated form we not unfrequently find extravasations of bile to a greater or less amount.

Medullary cancer rarely passes into suppuration, as it generally terminates fatally by inducing universal cachexia and exhaustion. Its fusion is still more rarely found to take place within a fibrous sheath, as is comparatively oftener the case in the spleen. Occasionally nature seems to attempt an arrest of the morbid growth, by a conversion into fat or adipocire.

Hepatic cancer undoubtedly very often occurs as the first of a successive series of cancerous deposits; yet, in the dead subject, it is commonly found combined with carcinoma of the lymphatic glands, that are seated near the biliary passages and in the lumbar region, with cancer of the stomach, of the intestine (especially of the rectum), of the peritoneum, of the kidneys, and with universal cancerous infection. It is often developed with remarkable rapidity after the extirpation of cancerous growths, and is then generally accompanied by cancer in the lungs.

777. Medullary carcinoma not unfrequently occurs in the liver in the shape of *cancer melanodes* (melanosis), and equally as an infiltration, or in circumscribed masses. We find the most varied combinations of its elementary molecules with those of pure medullary cancer.

A common result of hepatic cancer making its way outwards, is inflammation of the peritoneum; the carcinomatous liver is consequently often found agglutinated to neighboring parts by means of cellular or cellulo-fibrous tissue, which may in its turn be subjected to cancerous degeneration.

SECT. II.—ABNORMITIES OF THE BILIARY PASSAGES.

We now come to the consideration of the diseases of the gall-bladder and its efferent duct, those of the *ductus communis choledochus*, of the *ductus hepaticus*, and of the branches and ultimate distribution of the latter. We include the entire apparatus under one head, though we shall devote a special consideration to the peculiar characters exhibited by separate sections.

§ 1. *Excess and Defect of Formation.*—In rare cases a congenital absence of the gall-bladder has been noticed, an anomaly which must not, however, be confounded with obliteration of the gall-bladder which is frequently consequent upon inflammation. When there are two livers, the gall-bladder and the entire apparatus correspond; but we also find, without any further anomaly, a twofold instead of a single common

duct; the two ducts then either both open into the duodenum, or one communicates with the duodenum, and the other with the stomach.

§ 2. *Irregularities of the Biliary Passages with reference to Calibre.* —Independently of congenital enlargement or diminution of these parts, we find important acquired anomalies in the shape of dilatation or contraction.

Dilatation either affects the entire apparatus from the duodenal orifice to the capillary gall-ducts equally or almost equally, or it only affects larger or smaller portions, whilst the remainder retains its ordinary size. The gall-ducts are capable of extreme distension.

We find that dilatation of the passages is caused by habitual accumulation of inspissated bile, and by everything that impedes the progress and the discharge óf the secretion. We allude to compression of the biliary passage within and external to the liver by morbid products or enlarged lymphatic glands, to diminution of their calibre by tumefaction of the coats, by cicatrices or unusually large folds or valves of the mucous membrane; to obturation by biliary calculi, by morbid growths projecting into the cavity of the biliary passages, by catarrhal or croupy secretions. Some of these obstacles occur mainly in one, others in another portion of the apparatus. If the impediment occupies the ductus choledochus, the dilatation gradually extends over the entire apparatus; but it must be observed that the dilatation of the gall-bladder does not in general correspond with the dilatation of the other portions, as its efferent duct (ductus cysticus), from opening into the common duct at an acute angle, is compressed by the enlarged ductus choledochus. The more completely the calibre is obstructed, the more complete is the capillary distension; the more rapidly it ensues, so as not unfrequently to induce rupture.

The ductus choledochus is either found compressed by disorganized, and especially by cancerous, lymphatic glands, or by the pancreas, or the passage is narrowed by the tumefied mucous membrane or by the tumefied valve, or it is closed up by a biliary calculus or a carcinomatous tumor from without. Occasionally it is so enormously dilated as to exceed the diameter of the small intestine; the slower this effect is produced, the more marked will be its active character; and the distension extends upwards, passing by the gall-bladder, as above observed, to the hepatic duct and its ramifications.

The channel of the ductus cysticus is found impaired by unusual flexures, or large and numerous mucous folds, consequent upon previous elongation and distension, by cicatrices and cancerous degeneration; it may become perfectly obliterated by the same means, or by biliary calculi, which are impacted in the neck, and more particularly in a lateral dilatation of the gall-bladder. Enormous dilatations of the latter result, which in the course of time induce an entire change in the tissue and the functions of the mucous membrane of the gall-bladder.

After this occlusion has been rendered complete, the residuary bile in the gall-bladder is absorbed; the mucous membrane secretes mucus more copiously, in proportion to the irritation exerted upon it by the stagnating mucus left after the removal of the specific contents of the bladder. The secretion gradually accumulating, the gall-bladder extends, and

its mucous membrane becomes converted into a serous membrane, which secretes a serous, albuminous fluid, resembling synovia; this is at first opaque, and subsequently becomes clear, and we detect in it, with the assistance of the microscope, nothing but solitary flocculi of pigmentary matter, and a few crystals of biliary fat. This affection of the gall-bladder is termed hydrops cystidis felleæ, and the bladder resembles the sound of fishes, being converted into a tense capsule,—a condition similar to that developed under analogous circumstances in the Fallopian tubes, the ureters, the pelves and calices of the kidneys, and even in the vermiform process.

The new lining membrane of the gall-bladder is subject to all the diseases to which serous membranes and their cavities are liable ; inflammations occur very frequently, giving rise to the most various exudations, and terminations as various. Among the latter, we allude especially to shrivelling of the gall-bladder, accompanied by diminution of its contents. These become inspissated, so as to form an adipose chalky pulp, or chalky concretion, with a subsequent ossification of the parietes.

The dilatation of the biliary ducts in the interior of the liver is either uniform, and affects the entire organ or certain portions only, or it occurs as a partial saccular dilatation of one or more of those ducts. In the former case the cause is generally to be found in an obturation of the biliary channels within or external to the liver, by means of concretions, cancerous growths, or croupy exudation ; and the dilatation very frequently extends from the ductus choledochus to the biliary passages within the liver. In well-marked cases the entire capillary network belonging to this apparatus is dilated and gorged with bile ; the parenchyma of the liver may be saturated with bile, and present a dark yellow or green color ; the viscus is turgid, though pulpy and friable, resembling the condition of yellow atrophy ; the larger ducts contain bile in a disorganized state, and not unfrequently blood in a similar condition.

This affection invariably proves fatal with symptoms of biliary infection of the blood, and consequent cerebral disease, which is often combined with exudation on the arachnoid, with intense icterus and extreme pain in the liver. The capillary ducts are occasionally ruptured, and this gives rise to larger or smaller accumulations of bile in the deep-seated portions of the organ ; or the rupture may occur in the peripheral layers, at spots where patches of dilated gall-ducts form rounded, fluctuating projections on the surface of the organ ; in this case the hepatic peritoneum frequently becomes involved, and extravasation may take place into the abdominal cavity. Finally, the bile that transudes through the coats of the gall-ducts may, if it reaches the peritoneum, induce peritonitis, which in its turn predisposes to rupture of the serous covering investing the approaching biliary abscess.

The second or saccular form of dilatation of the biliary ducts is generally the result of a catarrhal or blennorrhoic condition. Capsules varying in size from a pin's head to a hen's egg, with a loose mucous lining that forms valvular folds, are found scattered through the liver, and they contain a liquid consisting of blennorrhoic or purulent mucus and bile, which deposits a sediment or incrustations. The character of the investing membranes affords a sufficient distinction from other cavi-

ties containing a similar fluid ; but the afferent and efferent canal is not easily discoverable, even with the assistance of injections. These dilatations undoubtedly originate in an accumulation of catarrhal secretion, and are generally accompanied by a dull pain in the liver.

Contraction of the biliary passages is induced by the above-mentioned circumstances, and may advance to adhesion and obliteration, as is especially the case in the gall-bladder.

§ 3. *Anomalies in the Form and Disposition of the Biliary Passages.*—Among these we reckon the various congenital malformations of the gall-bladder, in which it presents an intestinal, cylindrical, extended, twisted, pyriform, or phial-shaped appearance, or in which it seems divided longitudinally or transversely, owing to a rigid condition of the internal folds. To this class also belongs the anomalous insertion of the ductus choledochus into the duodenum or stomach. The acquired malformations consist in contraction, obliteration, or dilatation of the gall-bladder ; in change of position of the biliary passages, owing to pressure exerted upon them by enlarged lymphatics, morbid growths, &c.

§ 4. *Solutions of Continuity.*—We regard as peculiarly interesting the spontaneous ruptures occurring in the biliary passages external and internal to the liver as a consequence of excessive dilatation, which is generally preceded or accompanied by inflammatory action. We have also to cite the perforations of the biliary passages external to the liver, resulting from suppuration of their coats, and the abnormal passages subsequently established between the biliary ducts and the stomach and intestinal canal ; as well as certain abscesses produced by suppuration of the capillary gall-ducts within the liver, of which we shall have occasion to speak more fully in the sequel. (See Textural Diseases of the Biliary Passages.)

§ 5. *Textural Diseases. a. Inflammation.*—We often observe catarrhal inflammation occurring in the biliary passages, with various terminations and results. Like catarrhs of other mucous membranes, it not unfrequently is a primary affection, and becomes chronic, or it as often is propagated from the intestine to the gall-ducts ; but it often evidently has its origin in the irritation caused by an accumulation or an alteration in the composition of the bile, and especially by biliary calculi. At the bedside the affection is undoubtedly often mistaken for irritation and inflammation of the hepatic parenchyma.

Owing to the paralytic state induced in the contractile and irritable layer of their coats, and to the accumulation of bile, the gall-ducts become distended, their mucous membrane relaxed and tumid, and the muscular coat hypertrophied ; within the liver saccular dilatations are formed ; the catarrhal disease induces a stagnation of bile, which gives rise to calculous concretions, and occasionally suppuration and perforation of the gall-ducts follow. In the range of the biliary capillaries it most probably causes, in the manner just described, the formation of peculiar accumulations (abscesses), which are remarkable for the blennorrhoic pus and the bile they contain, and are thus distinguished from the products of parenchymatous inflammation of the liver.

Inflammation originating in irritation, caused by biliary calculi, deserves a special consideration, on account of its terminations and its consequences; it occurs chiefly in the gall-bladder. Occasionally and particularly when brought on by an accumulation of bile from obturation of the neck of the bladder or of the ductus cysticus, it runs a very rapid course, attacking the submucous tissue of the gall-bladder, and terminating in rupture and effusion of its contents into the peritoneal cavity. At other times it proceeds more slowly, and after repeated relapses, induces suppuration and ulcerative perforation of the gall-bladder. The latter is most liable to occur at the dependent portion, which is chiefly exposed to irritation, viz. the fundus of the bladder; and as previous peritoneal exudation will have agglutinated it to adjoining viscera, the suppuration extends to them, giving rise to abscesses in the liver itself above the gall-bladder, or in the lesser omentum; or establishing fistulous passages through the abdominal parietes, or communications between the gall-bladder and the pylorus, the duodenum and the transverse colon. Lastly, in favorable cases, the coats of the gall-bladder may be converted into a fibrous, callous tissue; its contents are discharged by the normal or by the above-described anomalous passages, and the organ represents a thick-coated hollow capsule, with or without cicatrices on its inner surface, and containing, according to the condition of the mucous membrane, a mucous or serous fluid, and not unfrequently one or more calculi. This is the so-called obliteration or atrophy of the gall-bladder. The calculous inflammations of the biliary passages are followed, though less frequently, by similar results, viz. rupture, suppuration, gangrenous perforation, callous induration, and obliteration.

b. Croupy inflammation is of very rare occurrence. We have observed it in the mucous membrane of the gall-ducts in the liver, accompanying cholera-typhus and ileo-typhus. It gives rise to tubular exudations, in which the bile forms branched concretions which block up the passages, and thus cause dilatation of the capillary gall-ducts. We have already noticed the occurrence of the secondary and gangrenous typhous process on the mucous membrane of the gall-bladder.

c. Œdema of the coats of the gall-bladder.—Serous infiltration of the coats of the gall-bladder occurs in general dropsy, and especially in ascites, and also in the shape of subserous infiltration in inflammation of the peritoneum.

d. Adipose deposits in the coats of the gall-bladder.—An excessive deposit of fat under the peritoneal investment of the gall-bladder only occurs as an accompaniment of general adipose accumulation, or at least of accumulation of fat in the abdomen. Its occurrence is of some interest, inasmuch as, like the fatty deposit in the heart, it is likely to induce fatty degeneration of the muscular layer.

§ 6. *Adventitious Products. a. Fibroid tissue.*—Under this head we class the textural alteration occurring in atrophy of the gall-bladder after inflammation.

b. Anomalous osseous deposit—ossification, as elsewhere in mucous canals, takes place only as a consequence of previous textural alteration of another kind. Thus we find subserous osseous lamellæ formed in the parietes of the gall-bladder, after it has been converted into a sero-

fibrous capsule, in hydrops cystidis; or the fibroid tissue which is developed in the parietes of the gall-bladder, as a consequence of inflammation and partial suppuration, may ossify.

c. Tubercular deposit in the biliary passages is of very rare occurrence.

d. Carcinoma of the biliary passages is chiefly met with as a complication of cancer of the liver, but also of the lumbar lymphatic glands, and of the stomach. It occurs either as an idiopathic nodulated deposit in the submucous tissue, in rare cases giving rise to annular stricture and degeneration of the entire bladder into a cancerous capsule, or as cancerous infiltration of the mucous membrane; or, as is more commonly the case, the biliary passages are attacked from without, cancerous growths in the vicinity perforate the parietes, and push their way into the cavity. The gall-bladder is most frequently attacked by hepatic cancer; the ductus choledochus by carcinoma of the lymphatic glands. Obturation of the passages and hemorrhage are common consequences of the affection.

§ 7. *Anomalous Contents of the Biliary Passages.*—The most remarkable are those entirely abnormal contents of the biliary passages, which are either the product of textural changes and morbid processes in their coats, or which after being generated externally, are conveyed into the cavity by various passages. We allude to the sero-albuminous fluid of dropsy of the bladder, to mucus, to pus that has been formed in the biliary passages, or in hepatic abscesses, to blood derived from cancerous growths, to acephalocysts from the liver, lumbrici from the intestine, &c.

The bile itself presents great varieties as to quantity, but more still as to quality; in the majority of instances the anomaly has its origin not so much in disease of the liver, as in morbid conditions of other organs, especially of the intestine and of the portal blood.

As regards quantity, the bile is found accumulated to a large amount in the biliary passages and intestine, or it is remarkably scanty. It is to be observed that in the latter case the deficiency is sometimes compensated by the saturated condition of the fluid.

The qualitative anomalies of the bile are more numerous and important, and affect both its physical and its chemical constitution.

The color of the bile varies extremely: it may be pale yellow, ochrey, orange-colored, yellowish-brown, blackish-brown, black, or of all the different shades and tints of green. The consistency of the bile generally increases in a ratio with the increased depth of color, varying from the fluidity of water to the density of tar and of calculous concretions. In taste it varies as to the amount of bitterness, but it may also be more or less, or entirely, saccharine, saline, sour, alkaline, acrid, or insipid.

In reference to its chemical constitution, the bile presents, as might be inferred from its physical qualities, numerous deviations from the correct standard; the chief constituents vary in their relative proportions, or they are replaced by new anomalous substances.

The biliary calculi are of considerable importance. They originate in

a morbid constitution of the bile, which may be abnormal when secreted, or subsequently become so from stagnation and retention. They occur in the biliary passages external to and within the liver, but more especially in the gall-bladder.

Here too we find numerous variations with regard both to physical qualities and to chemical composition.

They vary in size from a millet-seed to a hen's egg, and more. We generally find the largest to be formed by several materials disposed in layers, with a preponderance of fatty matter. The larger they are, the less numerous will they be; sometimes several hundreds of small calculi are discovered in the gall-bladder.

Their form and surface vary much. Single calculi are commonly round, oval, or cylindrical; when very large, so as to occupy the entire cavity of the gall-bladder, they are frequently slightly curved; if several are present at the same time, they mutually prevent their enlargement, and in consequence of the friction and pressure they exert upon one another, they assume cubical, tetrahedric, prismatic, or irregularly polyhedric shapes, with convex or concave surfaces.

The calculi found in the ducts are generally cylindrical, occasionally branched, or entirely amorphous. Their surface may be smooth and unctuous to the touch, or rough, racemose, uneven, of a mulberry appearance, crystalline, or branched.

The texture of the calculi may be uniform or varied, in proportion as they consist of one substance, or of several layers. Many show no distinct arrangement; some have an earthy pulverulent fracture, or a fibrous, striated, laminated, micaceous texture, presenting a glassy, silky, or asbest-like gloss on fracture, as is particularly observed in calculi consisting of cholesterine.

Generally speaking, they are not very hard, and may, when first removed from the body, be easily compressed between the fingers. On drying, they crack and fall to pieces, and, at last become pulverulent, which is particularly the case with those concretions which consist of inspissated bile or biliary resin.

In color they vary considerably; they may be milk-white, bluish, chalky, light or dark-yellow, brown, black, or colorless, or transparent, with a slight yellow or green tinge. Those of an ochrey, red, green, and blue (bronzed) color are unusual. Sometimes we find them spotted, and either of a uniform color throughout, or varying in layers, or at least containing a differently colored nucleus.

Chemical analysis shows the biliary calculi to consist mainly of inspissated bile, biliary resin, coloring and fatty matter, and the calculus may be either formed of one of these substances or of a mixture of several. In the latter instance they either interpenetrate one another, or are disposed in distinct layers, which are distinguishable by their color or texture.

Large biliary calculi generally contain but a small portion of inspissated bile; the latter often forms small irregular concretions in the gall-bladder, or larger cylindrical and branched concretions in the gall-ducts, or it serves as a nucleus to the various calculi of the gall-bladder. The resin and pigmentary matter of the bile enter into the composition

of the majority of gall-stones, and that frequently to a considerable extent.

Cholesterine almost always preponderates ; it frequently occurs in a pure state as a white, mother-of-pearl like, shining, or opaque fatty investment, or in distinct layers of a striated texture, which are separated by colored resinous layers ; it may also exist in an isolated form, deposited round a colored nucleus, and give rise to translucent calculi of a striated and distinctly crystalline texture. In the latter case we generally find that small solitary calculi, in the former very large calculi result.

Picromel commonly occurs but in minute quantities, in biliary calculi ; the various salts they contain form but a small proportion compared to the amount of the above-named constituents. Those concretions in the gall-ducts which are found to consist of carbonate of lime, are not products of the bile, but of the blennorrhoic mucus and pus of the gall-bladder.

The calculi found in the same gall-bladder generally resemble one another in composition, shape, and size ; although we meet with occasional exceptions from this rule. Thus in dropsy of the gall-bladder, we often find, beside the calculus which closes up the cystic duct, and which is of an old date, and of complicated structure, a second crystalline calculus, of more recent formation, which consists of pure cholesterine.

The calculi are either unattached or sessile. In the latter case they may be grasped and retained by a portion of the bladder, or be agglutinated to its internal surface by exudation, or they may be included in compartments, formed by an inspissated albuminous product of the gall-bladder, or by organized lymph which has been converted into fibrous tissue. Small calculi are also occasionally formed within small saccular dilatations of the biliary mucous membrane, and may appear to lie external to the cavity of the bladder.

Biliary calculi frequently cause irritation, inflammation, and subsequent suppuration of the coats of the gall-bladder, which may terminate in various ways. Cicatrices are often left, which more or less diminish the cavity. They may induce complete occlusion of the biliary passages, followed by dilatation and retention of bile. We must, however, observe that sometimes, owing to the extreme distension which the biliary passages are capable of, calculi of the size of a hen's egg are enabled to pass.

Biliary calculi are of common occurrence. We have observed that their formation is peculiarly coincident with excessive deposit of adipose tissue and with carcinoma.

The entozoa occurring in the human gall-bladder are the endogenous acephalocyst of the hepatic parenchyma and the distoma hepaticum.

SECTION III.—ABNORMAL CONDITIONS OF THE SPLEEN.

§ 1. *Defect and Excess of Formation.*—The spleen is generally absent in acephalous monsters, together with other organs of the abdomen and thorax. Occasionally it is found wanting, together with the stomach or the fundus of the stomach, in subjects that are otherwise well developed,

or it exists in a rudimentary state, whilst the stomach is in a normal condition. The explanation of these phenomena is to be sought in the history of the development of the embryo.

The spleen is found double in biventral monstrosities. The multiplication of the spleen, in the shape of lienes succenturiati, is not to be viewed as an increase, but as a subdivision of the organ, which does not affect its individuality. We not unfrequently find, besides the main organ, small accessory spleens (lienes succenturiati) seated in the omentum and ligamentum gastrolienale. They vary in size from that of a millet-seed to that of a walnut, and in number from one to twenty. They are round, present the same structure as the spleen, and are morbidly affected at the same time, and in a similar manner as the latter. The marginal indentations of the spleen, or the complete separation of a portion of the organ by a horizontal fissure, form transitions to this abnormal condition.

§ 2. *Deviations of Size.*—Deviations of size consist either in an abnormal increase or diminution of the organ. The former is of particular importance, and those tumors afford a special interest, which depend upon congestion caused not by mechanical impediments, but by the peculiar relation of a morbid state of the blood to the spleen. With the rare exceptions of those cases in which, like analogous states of the liver, they are congenital, these conditions are acquired. They are either acute or chronic: in the former case they accompany other acute diseases, either during their entire course, or only during single stages; in the latter, the tumefaction results from dyscrasiæ or cachectic conditions, which induce congestion, induration, and hypertrophy of the spleen. These terms, however, from referring mainly to external appearances, are apt to cause the real nature of the disease to be overlooked.

It is unnecessary to enter more fully into the consideration of these changes affecting the splenic parenchyma, which are evidenced by tumefaction, as it will be more appropriate to treat the subject under the head of Textural Diseases. We merely add the following remarks:

a. Acute tumefaction is generally accompanied by considerable softening of the splenic parenchyma; chronic tumefaction by increase in the consistency of the organ. It is questionable whether the hypertrophy affects the elementary tissue and constitution of the spleen: this is a point which requires to be elucidated by further research; but there is no doubt of the fibrous trabeculæ of the spleen and its fibrous capsule becoming hypertrophied in old chronic tumors. When we have succeeded in reducing an acute or chronic tumor, or even a mere hyperæmic state of the spleen, we often find the sheath of the spleen thickened, opaque, corrugated, and relaxed after death—a fact which may serve as a useful indication.

b. The size attained by chronic tumors of the spleen is often very considerable. The spleen not unfrequently measures sixteen inches in its long, seven inches in its short diameter, and four inches in thickness; its weight may amount to thirteen pounds and a quarter, and, according to the observations of others, even to twenty and more pounds.

Diminution of the spleen is characterized by shrivelling of the fibrous tissue, which prevents the vessels from being injected ; and is peculiar to genuine cholera (cholera algida), or it occurs as atrophy, in consequence of a special change in the fluids at large. Under this head we must class numerous obscure cases of permanent diminution of the spleen in individuals who in no way resemble each other, of the reduction of the spleen observed by some pathologists as resulting from the use of steel, and of the senile involution of the spleen.

Atrophy varies in degree ; it occasionally advances to such an extent during the involution of the organ, as to reduce it to the size of a hen's egg or walnut.

The spleen in these cases is paler than usual, its consistency is increased or diminished, the organ may assume the toughness of leather, or become soft, friable, and pultaceous. Senile atrophy may be characterized in the following manner : the spleen is considerably reduced in size, and flabby ; its sheath is opaque, corrugated and thickened, but at the same time softened and easily ruptured ; the parenchyma consists of a pulp which is of the color of rust or the lees of wine, and which is enclosed in dense and equally friable, fibrous tissue. We not unfrequently find the sheath of the spleen indurated and cartilaginous, or ossified, and at the same time, ossification of the arterial ramifications and free calcareous concretions (phlebolithes) in the veins of the organ.

§ 3. *Deviations of Form.*—We not unfrequently meet with a tongue- or platter-shaped, almost cylindrical, globular, or angular spleen ; its edges may be more or less notched, which is particularly the case with the anterior margin ; and the indentation may extend so far as to cause a transverse division of the organ. These furrows are not to be confounded with the contractions that are occasionally produced by inflammation and metastasis, and which very much resemble the former.

§ 4. *Deviations of Position.*—The congenital anomalies that come under this head consist in the spleen occupying a place external to the abdominal cavity, when the latter is fissured, in its being placed in large umbilical herniæ, and in the left thoracic cavity when the diaphragm is absent, and in a varying position, consequent upon an anomalous congenital elongation of the peritoneal attachments.

Acquired deviations of position consist in a descent of the spleen, when forced down by enlargement of the left side of the thorax, or in its being pushed up by dropsical and ascitic accumulations, or by a tympanitic state of the intestine ; in its dislocation by various tumors, or in its descent from increase in size and weight. Enlarged spleens sink vertically into the left mesogastric region, or raise the diaphragm, or they descend to the ileum, and in the case of a still further increase of size, slide off from the latter, so as to occupy a diagonal position in the hypogastrium, and extend over the right ileum. There is no doubt that the spleen occasionally presents very loose attachments, and remains freely movable, even after it has been reduced from a hypertrophied state to its normal size, in consequence of the previous traction exerted upon its ligaments.

§ 5. *Solutions of Continuity.*—Under this head we class injuries of the spleen inflicted by cutting instruments, rupture consequent upon blows or knocks received in the region of the spleen, contusions, as in being run over, concussions, as in a fall, and spontaneous ruptures. The latter are of peculiar interest, as they are the result of acute and violent tumefaction of the organ, proceeding to a most intense degree. We are able to confirm the fact observed by other authors, of the occurrence of spontaneous rupture in typhus, in typhoid cholera, and in the hot stage of ague, and the consequent fatal termination from hemorrhage.

§ 6. *Diseases of Texture.*—The chief diseases that appertain to this class, the hyperæmiæ, the so-called infarction and hypertrophy, and inflammation of the spleen, require, in order to be duly appreciated, not only anatomical proof of the existence of the disease, based upon a clear notion of the structure of the organ, but more especially an advance in our knowledge of the pathology of the blood and the serum. Numerous diseases, and more particularly the simplest derangements, as many cases of hyperæmia, can only be elucidated by attending to these points. These diseases of the spleen are probably but rarely idiopathic; they almost always arise from certain anomalies of the blood and the serum, or from certain dyscrasiæ, which, though little known, and as little understood, bear a remarkable and positive relation to the spleen. The spleen may in fact be considered as the most sensitive test for a variety of dyscrasic states of the fluids. An acquaintance with this connection may serve to lift the veil which still conceals the true function of this organ. We shall now resume the consideration of tumefaction of the spleen, upon the basis of the above remarks, and enter into a more minute investigation of the subject than we could adopt in the previous general outline. The main points relating to deviations of consistency will at the same time be adverted to.

1. *Hyperæmia, anæmia.*—Hyperæmia of the spleen arises either from a mechanical impediment in the circulation of the blood, or from the peculiar relation alluded to as existing between the spleen and certain anomalous conditions in the fluids.

The first variety occurs in organic diseases of the heart and in hepatic obstructions, though not, especially in the former, to the extent, nor as frequently, as might be expected from obstacles or stasis affecting the entire system of the vena cava and venæ portæ. The deranged circulating fluid appears to have no affinity for the tissue of the spleen, and to be thus in part carried off, and in part mechanically retained. This latter portion, in the first instance, induces a hyperæmic turgor of the organ, and gives it a dark-red color, and subsequently, as is generally the case in these hyperæmiæ, induces hypertrophy of the fibrous tissue and of the pulpy parenchyma of the spleen. The organ is more consistent, indurated, and dense.

The second form accompanies various dyscrasic conditions of the fluids, and in proportion to their duration induces an acute or chronic tumor of the spleen, which differs in appearance, and in its primary and secondary constitution, according to the nature of the cause.

The hyperæmiæ affecting the peripheral portion of the organ not un-

frequently degenerates into inflammation of the peritoneal investment of the spleen; the resulting exudations are converted into the cellular, cellulo-fibrous, or cartilaginous tissues and adhesions, so often found upon spleens that have formerly been tumefied.

Anæmia of the spleen occurs in connection with the above-mentioned reductions in size.

2. *Tumors of the Spleen.*—We have already discussed the tumors of the spleen arising slowly or rapidly from hyperæmia, and from the congestion of dyscrasic blood, as far as regards the mere increase of volume. We have now to examine them more closely in other points of view.

These tumors are observed in typhus, and in many typhoid states, as in cholera typhus, in pyæmia, and in anomalous exanthematic processes, as occurring from disorganization of the blood after erysipelas, scarlatina, miliaria, or rheumatism, as found in drunkards, and in acute tubercular affections; they occur as a result of suppressed menstrual or hemorrhoidal discharge, of intermittent fever, of rickets, of lues and mercurial cachexia, and of many dyscrasic tubercular affections.

These tumors differ in character, and are owing partly to the hyperæmia, partly to the deposition of an anomalous fibrous product in the parenchyma of the spleen. We find the greatest difference in the consistence of the tumors; but the chronic indurated tumors, are undoubtedly soft at first, and subsequently attain greater hardness, according as the deposit is more or less coagulable. The same remark applies to the color of the tumor, which at first is undoubtedly red, but subsequently becomes paler in proportion as the coloring matter is absorbed, and the hyperæmia is forced to yield to the compression exerted upon it by the deposit. We find, as regards other qualities, that the morbid product offers very prominent varieties, which we will examine in the analysis of the chief tumors that follows; the finer, though not less different characters we leave to another department of science, which, though not yet cultivated, promises many and very important results.

a. Among the tumors which accompany acute diseases of the blood, those of typhus are distinguished by their rapid and extensive increase, by their lax tissue, both of which circumstances sometimes predispose to rupture, and by the dark-red color of the parenchyma. This variety originates in stasis affecting the vascular system of the fundus ventriculi, and in the deposition of a very lax, pultaceous, semi-fluid, blackish-red, dirty violet, or lighter-colored purplish mass, varying in amount, and resembling the pulpy medullary matter found in the typhous mesenteric gland. If this substance is deposited rapidly to a large amount, the fibrous trabeculæ of the spleen are rendered soft and friable by extension; and if the deposit is very soft, the viscus presents fluctuation.

The tumors occurring in the other acute dyscrasiæ above alluded to, are more or less allied to this one. When accompanying universal acute tuberculosis, the eliminated mass, partially at least, at once assumes the characters of tubercle. The spleen may increase from a slight enlargement to three, four, five, and six times its normal size.

Tumors occurring after suppression of the above-mentioned hemorrhages do not generally become a subject of anatomical research until they have attained a very considerable size. They are most probably

the result of repeated typical (typische) hyperæmiæ, and would be found at their commencement to be of slight consistency, and of a red color. A coagulable fibrinous deposit, however, takes place, and the tumor, therefore, in proportion to the amount of coagulation, becomes hard, elastic, and indurated, the parenchyma is reddish-brown, of the color of fresh muscle, and presents on section a fleshy (sarcomatous) appearance; by degrees the coloring matter is absorbed, the organ then presents a pale red, yellowish, or reddish-white appearance, and resembles fibrine that has been washed. During the hyperæmiæ the fibrous trabeculæ also increase in quantity and toughness, so that the tumor becomes more resistant; the fibro-serous capsule is also rendered more opaque, and is thickened; it is invested with a cellular pseudo-membrane, resulting from peritoneal inflammation, and is thus attached to the abdominal parietes. The deposit gradually increases to such an extent as to induce a compression of the vascular portion of the spleen, and to render it impermeable to injections; for the same reason, the tumor gradually becomes paler, and a vicarious development of the vessels at the fundus of the stomach ensues.

The third variety of splenic tumors bears a general affinity to those accompanying the above-named cachexiæ, but the deposit that occurs in them and is substituted for the parenchyma of the organ much resembles bacon in consistency and appearance; the organ on section offers a very smooth surface, a dull, lardaceous (speckig, baconny), waxy gloss, and its superficial layer appears partially transparent; the spleen is hard, but breaks with a peculiar fracture; it presents a color varying from dark purple to pale red, and the blood contained in the vessels is pale and serous; this variety of splenic tumor is often coincident with the analogous lardaceous infiltration of the liver (vide p. 100): it may, however, occur in an isolated form, or complicated with a similar affection of the kidneys (a variety of Bright's disease). Like the other varieties, this tumor may attain an extreme size, and dropsy, and especially ascites, are common results.

b. We have lastly to advert to the fact that many cases of swelled spleen depend upon the formation of certain corpuscles, in addition to the coexisting hyperæmia. These small bodies are quite distinct from the Malpighian corpuscles, found in the spleens of some graminivora; they are minute grayish-red, or grayish-white, opaque, soft, deliquescent, vesicular substances, of the size of a millet-seed, which occupy the parenchyma of the spleen. They accompany a morbid development of the abdominal lymphatics, and especially of the follicular apparatus of the intestinal mucous membrane, with turgescence of the mesenteric glands, occurring in those affections of children and young subjects, which we have spoken of at page 61; they are also found in typhous affections of these organs, and of the spleen, and indicate a predominance and qualitative derangement of the lymphatic system. They are consequently also found complicated with acute and chronic tumors of the spleen, and are not to be confounded with acute tuberculosis of that organ.

The consistency of the spleen, as may be gathered from the above remarks, depends almost entirely upon the state of aggregation of the parenchyma, or of the morbid product which has replaced the latter. The condition of the fibrous tissue also influences it to a certain extent,

but it varies much even within the limits of its physiological condition. The two extremes constitute softening and induration of the spleen, which we have already examined in their strict sense.

In very rare cases, the black softening of the tissues of the fundus ventriculi, is accompanied by a similar affection of the splenic tissue, which is converted into a black, carbonaceous, tarry, semi-fluid mass, originating in the vascular system.

3. *Inflammation of the Spleen.*—The very important conclusions to be derived from pathological anatomy in reference to inflammation of this organ, and with regard to its influence upon sanguification, will be self-evident.

We cannot doubt that the pulpy substance of the spleen may be the original seat of inflammatory action; the fact has not, however, been as yet anatomically demonstrated; in the same manner it is not improbable, though by no means proved, that many acute and chronic tumors of the spleen may be the product of inflammation.

The variety of inflammation for which pathological anatomy affords an explanation is, to name it from its seat, phlebitis, i. e., an inflammation of the numerous anastomosing and tortuous venous canals of the spleen. In fact, we have only to apply the doctrines promulgated with regard to inflammation of a vein to a venous ganglion, in order to obtain a correct picture of inflammation of the spleen; that which elsewhere takes place in a simple vascular tube is here found in a complicated venous apparatus.

This inflammation of the spleen occurs as a primary or as a secondary affection. Whilst the former is as rare as spontaneous primary inflammation of a vein, the latter is as frequent as secondary phlebitis.

Primary inflammation of the spleen, if not early combated, or unless ending in resolution, gives rise to an exudation of laudable pus or fibrine. In either case the circulating fluid may become infected, and coagulation be produced in the most various regions of the vascular, and especially in the capillary system. This is an explanation of so-called metastases. However, this is unusual in the case of fibrinous exudation, as the inflamed vessels are closed by the coagula, causing obliteration and subsequent conversion of the inflamed part of the spleen into a fibro-cellular callus, which may even ossify.

In the case of purulent exudation, inflammation of the spleen passes into suppuration, and abscesses form. In a favorable case, the abscess may be circumscribed by adhesive inflammation, and, being enclosed in a sac formed by obliterated parenchyma, which has been converted into fibrous tissue, may be borne for a long period; a partial absorption of the pus may take place, and the remainder becoming inspissated be reduced to a calcareous greasy pulp, or even to a hard concretion. The more common case is that the parietes of the abscess also put on inflammatory action, and suppurate, in consequence of which the abscess generally enlarges very rapidly, with symptoms of violent and universal reaction in the shape of hectic fever. We then have a case of florid (floride) splenic phthisis.

If the inflammation extends to the sheath of the spleen, inflammation of the splenic and neighboring peritoneal surface ensues; an occurrence which is analogous to the communication of disease from an inflamed

vessel to the tissues in its vicinity; the inflammation is not, however, apt to spread far.

The splenic abscess not unfrequently discharges,

Firstly, Into the abdominal cavity; the pus is then often enclosed by the product of circumscribed peritonitis, which causes the formation of a sac, bounded by the external wall of the abdomen and the diaphragm, the fundus ventriculi, the colon, and its mesentery; the entire spleen is thus occasionally destroyed by suppuration.

Secondly, Into the left thoracic cavity, after suppurative destruction of the diaphragm, or,

Thirdly, Into the cavity of the transverse colon, and into the stomach.

Secondary inflammation of the spleen is of frequent occurrence in all cases in which the blood is poisoned by the absorption of an inflammatory product, or has become affected in an analogous way spontaneously, a fact which indicates the delicate reaction of the spleen to a morbid condition of the blood. We then see the formation of inflammatory spots, which are in every way remarkable. They are well defined; they always occupy the peripheral portion of the organ, and generally present a cuneiform shape, the base being at the surface, the apex being directed towards the interior; there are often two, three, four, and more of these foci present at the same time; they vary in size from that of a pea, to that of a hen's egg, and in rare cases involve an entire third of the viscus.

The substance of the spleen appears considerably darker at these spots, from the commencement, and also denser and more resistant; it subsequently assumes a reddish-brown color, and its density also increases, so that the affection may be at once identified, even externally; its limits are now well defined, and reactive inflammation is set up in the adjoining tissue. The process may terminate in various ways: in favorable cases, especially when a benignant fibrinous exudation has been absorbed into the blood, as frequently occurs in inflammation of the internal membrane of the bloodvessels, and particularly of the endocardium, the diseased tissue is converted into a cellulo-fibrous callus, which contracts and causes a cicatrix at the surface, by drawing the sheath of the spleen inwards. The more common case is that pus or ichorous matter is absorbed, and that the inflamed portion is converted into a puriform, creamy mass, or into a sanious, greenish, greenish-brown, or chocolate-colored pulp; in the latter instance, the conversion is often effected with very violent symptoms, without previous induction of the paleness above described.

The entire process is a detailed repetition of that occurring in secondary phlebitis, and is nothing more than the metamorphosis of an infected coagulum within the channels of a vascular ganglion.

When the disease affects the peripheral portions of the organ, peritonitis frequently supervenes, and an eschar having formed in the sero-fibrous sheath, a discharge into the abdominal cavity is not rarely effected.

This secondary inflammation of the spleen is a very frequent complication of inflammation of the internal vascular coat, and particularly of endocarditis. Of all organs that are affected in a similar manner, by the absorption of a product of inflammation into the blood, the spleen is the

most liable to be attacked. When occurring as a result of spontaneous disorganization of the blood, it is particularly important in complication with croup, as also with exudative processes on mucous and serous membranes, particularly with pneumonia, and, lastly, with the analogous process of tubercular disease.

4. *Gangrene of the Spleen.*—Gangrene is as rare an occurrence in the spleen as in the liver; we have had an opportunity of observing it once in a chronic tumor of the spleen, affecting the organ to a considerable extent.

5. *Adventitious growths. a. Anomalous, fibrous, and fibro-cartilaginous tissue.*—This tissue occurs—

a. Very often upon the surface of the organ underneath its peritoneal sheath, in the shape of smooth and level, or tuberculated plates, of varying thickness and size. It occurs in this shape at advanced periods of life, as a result of the congestion to which the parenchyma and the investment of the organ have been exposed. It is not very unusual to find these laminæ of such an extent as to invest the entire convexity of the spleen, and to present a thickness of several (two, three, and five) lines.

β. It occurs very rarely in the shape of fibroid tumors of the parenchyma of the spleen.

b. Anomalous osseous growths.—We find them occurring—

a. As ossification of the fibroid laminæ just described, of the same extent and thickness as the latter; they are rarely found except in very old people;

β. As cretified fibrine in the cellulo-fibrous callus, subsequent to primary and secondary inflammation of the spleen;

γ. As round unattached concretions, or phlebolithes, in the venous channels of the spleen.

c. Formation of cysts.—Encysted tumors of the spleen are very remarkable, and as unusual as cancer, a fact which is interesting on account of the contrast with the frequency of tubercle. The acephalocyst is either found in the spleen alone, or concurrently with one in the liver; it rarely attains the size it reaches in the latter organ, but is otherwise not distinguished by any peculiarity. Cysts with other contents are still less frequent.

d. Tubercle.—Tubercular disease affects the spleen only less frequently than the lungs and the lymphatic glands. It always characterizes an advanced stage of tuberculosis, which had previously only appeared as chronic disease in some other organ, as the lungs, the brain, or the lymphatic glands, or had merely existed in a latent form, and is now converted into acute general tuberculosis. Splenic tubercle is consequently always complicated with tubercle in the most various organs, and very frequently with universal tubercular deposit.

Tubercle of the spleen, when acute, commonly appears in the shape of numerous densely-sown granulations of the size of a pin's head or millet-seed, resembling gray transparent vesicles, or of an opaque white color; or as yellow cheesy masses, varying in size from a millet-seed to a pea. When chronic, it presents the shape of crude, originally gray, granulations of the size of a millet- or hemp-seed, which subsequently are converted into a cheesy substance.

The parenchyma of the spleen is the seat of tubercle ; we not unfrequently find a small central cavity within the tubercle, and the latter is occasionally surrounded by a cyst or capsule of fibro-lardaceous texture, a fact which demands special investigations for its elucidation.

For the same reasons that apply to hepatic tubercle, tubercle of the spleen scarcely ever passes beyond the stage of commencing ramollissement.

The spleen appears swollen in proportion to the quantity, and also to the size of the tubercles ; in acute tuberculosis its turgescence and the relaxation of its parenchyma strongly resemble the typhous condition.

e. Cancer.—Cancer occurs very rarely; we have as yet only met with the medullary variety in combination with cancer of other organs, especially of the liver and the lumbar glands. The structure of the spleen appears to afford a satisfactory explanation of the fact, that cancer occurring in it is frequently invested by a fibrous sheath, within which it passes into a state of ichorous solution. The sheath is formed by the displaced fibrous tissue of the spleen, which, in the case of the adventitious growth attaining a considerable size, is strengthened by the fibrous investment of the spleen.

SECT. IV.—ABNORMITIES OF THE PANCREAS, AND THE OTHER SALIVARY GLANDS.

We shall first examine the abnormities affecting the parenchyma of the above-named glands, and then proceed to examine those of their efferent ducts, and of their contents. We may observe, generally, that these organs are not very liable to become diseased.

§ 1. *Abnormities of the Pancreas and the Salivary Glands.*—1. *Defect and excess of formation.*—Absence of the pancreas and the salivary glands is only observed in very imperfect monstrosities; salivary glands sometimes present an arrest at a very low stage of development, inasmuch as they may be blended with one another and with the thymus and thyroid glands, so as to form one mass. Excess of development occurs very rarely in the shape of a double pancreas, or of an extravagant development of accessory appendages.

2. *Deviation in size.*—Enlargement of the above-named glands, in consequence of hypertrophy, is altogether unusual; but when it does occur it affects not so much the acini themselves, as the interstitial cellular tissue. The gland therefore almost invariably becomes more compact and drier, and then presents simple non-malignant induration.

A diminution of the pancreas is the result of atrophy. Occasionally, and particularly at an advanced age, this takes place spontaneously, or it may be induced, secondarily, by other anomalies, such as chronic inflammation and adipose infiltration, or the deposition of calcareous matter in the efferent ducts. The atrophic state is accompanied by variations of consistency, the organ sometimes presenting coriaceous tenacity, at others a lax and pultaceous condition.

3. *Deviations of consistency.*—We meet with the most various degrees of consistency in the pancreas. The two extremes only come within the

range of pathology; they are on the one hand extreme cartilaginous dryness of the tissue, and induration which is generally coupled with enlargement; on the other extreme softening, relaxation, and succulence of the tissue.

4. *Diseases of the tissues.* *a. Inflammation.*—Inflammation of the salivary glands is either acute or chronic, and it is either primary or secondary; in the latter case it is metastatic. Inflammation of the pancreas, at all events in the acute form, is extremely rare: this is not the case with the other salivary glands, especially with the parotis; here the inflammation is very often primary, and still more frequently metastatic.

The acute form is characterized in the following manner: in the first instance there is tumefaction of the gland, reddening, congestion, relaxation, and succulence, i. e. infiltration of the interstitial cellular tissue; in the progress and in the higher stage of the disease, a sarcomatous condensation of the cellular tissue follows as a consequence of plastic exudation into its areolæ; the congestion and reddening attack the acini, which appear to be fused with the former, and the entire gland is enlarged and indurated. Unless the inflammation pass into resolution, small punctiform abscesses result, which enlarge, become more numerous and coalesce; the gland, and particularly the cellular tissue, is now found uniformly infiltrated with yellow pus, which exudes from it as from a sponge, whilst the acini appear as small, red, lax, friable corpuscles, which fuse at a later period; or suppuration is established at distinct spots so as to form an abscess, which may discharge itself in various directions, subsequent to the destruction of the adjacent tissues.

Chronic inflammation induces condensation, induration of the cellular tissue, obliteration of the acini, and either permanent enlargement or subsequent atrophy of the gland.

The metastatic forms of inflammation not unfrequently pass rapidly from the stage of hyperæmia with livid redness, into sanious ulceration, with sudden disappearance of the turgor.

b. Adventitious growths.—The salivary glands are not very subject to the formation of morbid growths; tubercle is never discovered in them, and carcinoma rarely attacks them primarily. We find the pancreas liable to—

a. Excessive accumulation of fat, which may terminate in a conversion of the entire organ into one mass of fat. This affection rarely occurs without a coincident accumulation of fat in the abdomen. The disease proceeds from without inwards, and in very obese persons a direct communication may be traced between the surrounding fat and the pancreas; the cellular tissue gradually absorbing the lax greasy fat, the acini, which are of a dirty yellow color, being reduced and gradually disappearing. When the disease has attained its extreme limits, a mere pultaceous strip of fat retaining the general outlines of the gland is found in its place; only scattered remains of the acini are discoverable, and in the delicate and thinned duct there is a whey-like fatty fluid. The disease occurs frequently in drunkards, associated with fatty liver and the formation of biliary calculi.

β. Cysts.—Serous cysts are occasionally formed in the pancreas, as

well as in other salivary glands. They are to be carefully distinguished from dilatations of the ducts and their terminations, which put on a similar appearance.

γ. Fibrous tissue, cartilaginous and osseous growths. Tumors of this description occur but very rarely in the parotid.

δ. Carcinoma.—Carcinomatous disease occurs, in the pancreas and salivary glands, and especially in the parotid, in the shape of scirrhus and medullary cancer. In the parotid it sometimes appears as a primary disease; in the pancreas we have only found it, and even then exclusively at its duodenal end, as a complication of extensive carcinoma of numerous other organs. The secondary affection of the salivary glands by an extension of the disease from adjoining organs, and in the case of the pancreas especially, by an extension from the scirrhous pylorus, is very common. Cancer appears in the shape of infiltration of the interstitial cellular tissue of the gland or of nodes. Dr. Berg has, during his residence in Vienna, discovered carcinomatous induration of the entire pancreas in a new-born child.

§ 2. *Abnormities of the different Ducts and of their Contents.*—Next to salivary fistula subsequent upon injuries and ulcerative destruction of the tissues, which occurs chiefly at the ductus stenonianus, but which we have also seen in the shape of pancreatic fistula (see p. 39) discharging by a perforating ulcer of the stomach, we find dilatation of the excretory ducts and of the ductuli salivales to be the chief and most frequent affection that has to be noticed under this head.

Dilatation depends mainly upon retention and accumulation of the secretion, and may either affect the entire duct or one portion uniformly, or small detached points, so as to form saccular or varicose dilatations; in the latter case, again, the duct may present single fusiform or vesicular dilatations at intervals, or numerous closely-set expansions, which are partially separated from one another by valvular folds formed by the coats of the duct. The coats may be either considerably thickened or considerably attenuated.

The cause is generally to be found in some mechanical impediment, such as compression and complete obliteration of the duct external or internal to the gland by morbid growths of various descriptions. In the pancreatic duct it may be induced by gall-stones occupying the orifice of the ductus choledochus, by a sudden curve or angle of the duct brought on by cancerous induration and shrivelling of the normal tissue, with change of position, such as we often observe in the pancreatic duct near the head of the pancreas. It may be induced by tumefaction of the internal membrane, by a mucous plug, and especially by calcareous concretions (salivary calculi). In rare cases the dilatation of the pancreatic duct is, like that of the bronchi, brought on by induration and atrophy of the gland. In morbid softening of the gland, and especially in the adipose metamorphosis, the duct is deprived of its contractility, and dilatation with a marked attenuation and relaxation of its parietes ensue; lastly, dilatations of the duct may take place without any mechanical obstruction, in consequence of scirrhoid disease of its duodenal end; the duct in this case fuses with the scirrhous portion of the gland; it is thus fixed, the

scirrhus involves its tissue, whereby its vital contractility becomes impaired, and the secretion is allowed to stagnate in its cavity.

The dilatations of the pancreatic duct enlarge to the size of a goose's or swan's quill; the saccular expansions may reach the size of a hazelnut or pigeon's egg. In Wharton's duct the dilatation occurs in the shape of a fluctuating tumor, and is known as ranula. Dilatation of the ductuli and their terminations sometimes puts on the shape of serous cysts.

The contents of the salivary ducts, i. e. the saliva of the mouth and stomach, occasionally offer rather remarkable anomalies in reference to quantity, color, consistency, and probably, as indicated by the taste, and especially by its acid or alkaline reaction, in reference to chemical constitution. Not unfrequently calculous concretions, the so-called salivary calculi, are generated in the saliva, and this is more especially the case in the ducts of the sublingual gland and the pancreas. They are white, friable, and either round, oblong, cylindrical, or obovoid; in size varying from that of a millet-seed or a pea, to even that of a hazelnut; they are either solitary, or if small, frequently very numerous (twenty and more); and they are composed of phosphate and carbonate of lime, held together by animal matter. These calculi give rise to obturation of the ducts, and consequent accumulation of the secretion and dilatation.

At times, blood, pus, cancerous sanies, is found in the salivary ducts; bile is not unfrequently discovered in the pancreatic duct; in one case of migration of lumbrici into the biliary vessels, two were found to have crept into the latter.

PART II.

ABNORMITIES OF THE URINARY ORGANS.

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ABNORMITIES OF THE URINARY ORGANS.

UNDER this head we comprise the morbid anatomy of the kidneys and the efferent apparatus, viz. the calices, the bladder, and the urethra; the two are of course very intimately related to one another. The abnormities of the suprarenal capsules will be considered in an appendix.

SECTION I.—ABNORMITIES OF THE KIDNEYS.

§ 1. *Defect and Excess of Formation.*—The urinary apparatus is very rarely entirely deficient; it is generally found even in very imperfect monstrosities. One kidney is frequently absent, or individual portions of the system are, as we shall have occasion to see, more or less imperfectly developed.

When one kidney only is present, it is important to distinguish between the unsymmetrical and the solitary kidney. The former is represented by a right or left kidney, which is normal in regard to position and conformation, and occasionally rather enlarged, its fellow being deficient. The solitary kidney is the result of a fusion of the two organs, and therefore offers the characters peculiar to this arrangement in a greater or less degree. The lowest degree of fusion is seen in the horseshoe kidney (ren unguliformis); the two kidneys are united at their inferior portions by a flat, riband-like, or rounded bridge of tissue, which crosses the vertebral column. In the higher degrees the two lateral portions approach one another more and more, until they reach the highest degree, in which a single disk-like kidney, lying in the median line and provided with a double or a single calyx, represents complete fusion. The more intimate this union is, the more the hilus of the kidneys is directed forwards, so that whereas, in the lowest degree, it is indicated by an evidently increased development of the posterior labium of the hilus, the hilus of the solitary kidney occupies the anterior surface. The more considerable the fusion is, the more the kidneys descend along the vertebral column, and the solitary kidney is commonly situated at the promontory, or even at the concavity of the sacrum. In exceptional cases only the solitary kidney is placed, like the unsymmetrical kidney, at the side of the vertebral column, on one side of the median line.

Excess of development occurs very rarely, except in the case of biventral monsters, in the shape of a third kidney, situated in the median line, and generally placed at the promontory; or in the shape of a single symmetrical kidney, which is composed of two kidneys united into one.

§ 2. *Deviations of Size.*—The kidneys are found enlarged or diminished in various degrees, and under various circumstances.

1. Enlargement is observed—

Firstly. Occasionally in one kidney, after its fellow has been deprived of its functions; this is a case of hypertrophy which may be considered as analogous to the increase of size in the unsymmetrical kidney ;

Secondly. As congestive turgor ;

Thirdly. As inflammatory swelling ;

Fourthly. As a consequence of infiltration of the renal tissue induced by or independent of inflammation; various forms of Bright's disease belong to this subdivision ;

Fifthly. as arising from morbid growths, in which case the enlargement corresponds to their number and size ;

Sixthly. As originating in dilatation of the pelvis and calices of the kidneys; the greater in this case the increase of size, the more will the renal substance become atrophied in consequence of pressure from within. Rayer states the left kidney to be normally of greater weight and larger dimensions than the one on the right side.

Abnormal smallness is either congenital, or the result of atrophy. Spontaneous and primary atrophy occurring independent of contraction, or complete occlusion of the artery, is very unusual, and belongs almost exclusively to old age ; secondary atrophy, resulting from and complicated with disease of the tissues, is much more frequent. In the case of extreme dilatation of the renal pelves and calices atrophy and enlargement appear combined.

2. Atrophy may affect the two substances of the kidney uniformly; or it may involve the cortical substance only ; the latter is the more frequent case in secondary atrophy, on account of the greater proclivity to disease in the cortical substance. The tissue is rendered pale, or it may be distinguished by its darker color, and the vessels are often found varicose. We very often find an unusual amount of fat accumulated round the atrophic kidney.

We shall have occasion to enter more fully into the subject of secondary atrophy, at a future period.

§ 3. *Deviations of Form.*—Besides the anomalous forms of the kidneys, resulting from fusion of the two organs, which we have already described, we may point to the lobulated kidney as an interesting conformation. It occurs as an arrest of development in the foetal state, or if acquired, as atrophy of the cortical substance, accompanied by dilatation of the calices. There are other congenital malformations of the kidneys, which are of less importance, as, for instance, the elongated kidney, which appertains to the foetal state, the round, prismatic, triangular, cylindrical kidneys, the kidneys with a transverse furrow (separation into an upper and lower half) ; and also various acquired malformations, which are caused by external pressure, by partial loss of substance, and atrophy.

§ 4. *Deviations of Position.*—Here too we must first point to an anomaly resulting from the various degrees of fusion of the two organs,

i. e. the descent of the kidneys to a lower part of the abdomen. This may, however, occur independently of the malformation alluded to, and we sometimes find one, sometimes both kidneys, as low down as the brim of the pelvis, or even as the hollow of the sacrum. The anomalies in the origin of the renal vessels which correspond to the original deviation of position deserve attention, as well as the increase in their number and the diminution of the ureter in proportion to the descent of the kidney.

The kidneys, and especially the right one, may be depressed by an enlarged liver, and the consequence is, that the hilus of the former is turned upwards, as the upper portion of the kidney is necessarily most depressed.

We have lastly to allude to the occasional movability of the kidneys, which is owing to insufficient fixation by means of the adipose fascia, and apparently also to an elongation of the vessels; we sometimes find that the kidneys may be moved from one to two inches along the spinal column.

§ 5. *Deviations of Consistency.*—The kidneys sometimes offer a diminution of consistency, or relaxation, or an increase of resistancy or toughness, without any apparent change of texture. The former occurs concurrently with a similar condition in other parenchymatous organs, and is the result of cachexia, anæmia, and marasmus, and of defibrination of the blood, from excessive exudations; the organs, in this case, are very pale and friable. Both an increase and a diminution of consistency are much more frequent as complications of textural alterations, and we shall examine them more in detail under this head. Genuine softening of the entire kidney, or of a portion of the organ, in the shape of spots of various sizes, of a dirty brown, chocolate-colored, rusty pulp, is a very rare occurrence.

§ 6. *Solution of Continuity.*—This is produced not only by cutting instruments, but may occur in the shape of rupture, from concussion, or in consequence of falls or blows, received in the region of the kidneys. After a fall from a considerable height, rupture of the kidneys is very frequently complicated with laceration of other abdominal viscera. It gives rise to hemorrhage, inflammation, and suppuration; the latter terminates in the manner that we shall have occasion to delineate when speaking of renal abscess. Concurrent injury of the calices and of the pelvis of the kidney, causes extravasation of urine into and beyond the adipose covering of the kidneys: if the peritoneum has also suffered, a fatal termination ensues rapidly; if not, a permanent or temporary cure, with a residuary fistula, may follow.

§ 7. *Diseases of the Tissues.* 1. *Hyperæmia, apoplexy, anæmia.*— Hyperæmia of the kidneys not unfrequently occurs in the active form accompanying an exaltation of the renal functions; or as passive congestion in consequence of general marasmus, and especially in consequence of paralysis of the spinal and ganglionic nerves, such as we find in the torpid condition of the sympathetic in the insane, connected with abdo-

minal plethora and congestion, and in paraplegic cases; it also occurs in
the mechanical form as a consequence of impeded circulation in con-
nection with hyperæmia of other organs. The effects, are swelling of
the organ (congestive turgor) and increase of size, greater depth of
color of the tissues, increased density and resistancy, and loose attach-
ment of the fascia propria. In children the tubular portion is frequently
the chief seat of hyperæmia. When it has reached a high degree, it is
apt to give rise to spontaneous hemorrhage (renal apoplexy), which, both
in children and adults, has its main seat in the pyramids. We then
find in the place of the pyramids, a spot of various dimensions, which
has pushed aside a proportionate amount of parenchyma, and contains
besides coagulated dark blood, the broken-up remains of the tubular sub-
stance. A cure undoubtedly ensues occasionally; the effusion gradually
loses its color, and assumes a rusty and a yellow tint; it is then absorbed,
and the calyx becoming obliterated, a fibro-cellular cicatrix closes up the
cavity. Minute hemorrhagic spots, in the shape of ecchymoses of the
tissue resulting from an acute disorganization of the blood, as well as small
extravasations under the tunica albuginea, are of much more frequent
occurrence.

Hyperæmia accompanied by increase of size (hypertrophy), is, accord-
ing to the few cases we have been able to examine, the only anomaly of
the kidney, demonstrable in diabetes by the pathological anatomist.

Anæmia of the kidneys occurs not only in connection with general
impoverishment of the blood, but it is found as a more or less character-
istic symptom, in all those cases in which the renal parenchyma has
become impermeable from being infiltrated with coagulable matter, either
owing to inflammation or deficient nutrition; this is particularly the case
in that disease which is commonly cited as the type of the class, Bright's
disease of the kidney.

2. *Inflammation.*—Inflammation of the kidneys is either primary,
secondary, or metastatic; in the first case it results from injury, concus-
sion of the intestines, cold, or specific irritation (turpentine, cantharides,
&c.); in the second it follows acute or chronic diseases, and it then pre-
sents a more or less remarkable type, corresponding to the general
dyscrasia; in the third instance it arises chiefly from inflammations of
the pelvis and calices, or from inflammations of the fascia adiposa and
adjoining organs. The inflammation runs an acute or a chronic course;
the idiopathic variety being particularly liable to the former.

The cortical substance is the chief seat of the first two varieties, as of
textural alterations generally; when the inflammation commences at the
pelves of the kidneys, the tubular substance is naturally implicated also.
In the former case we often find one or both kidneys, either simultane-
ously or in rapid succession attacked throughout their substance;
whereas the latter commences in spots from which it extends through the
renal tissue.

The following are the anatomical characters of acute inflammation of
the kidneys, modified of course by the degree and the acuteness of the
affection.

Hyperæmic tumefaction and redness of the organ are followed by
a uniform discoloration of the parenchyma which appears of a dirty

brown or purple color, and filled with a dark sanguinolent fluid; it is either turgid and resistant, or collapsed, flabby, and very friable; or it may be turgid and friable, and the discoloration less uniformly grayish-red, or dirty white, accompanied by infiltration of a denser, coagulable, fibrinous substance, the texture is granular, the surface scattered over with an injected, asteroid, and polyhedral vascular network, and the fractured surfaces or sections made in the direction of the hilus, are streaked with striated vessels.

The general result of the infiltration is, that the organ is more or less swollen and discolored, and that its consistency is variously diminished. In accordance with what has been above remarked, we find the cortical substance chiefly affected; the affection is general or partial, and in the latter case it occupies particularly the superficial layer; in the first instance the swollen cortical substance is found to have forced its way into the basis of the pyramids, between the fasces of the tubuli, and they consequently appear unravelled and fimbriated.

The process not unfrequently extends to the tubular portion itself, or the latter is involved in the inflammation propagated from the pelvis. The pyramids then appear enlarged, swollen, pale; their color changed to a dirty brown, or grayish-red, and softened or indurated according to the nature of the inflammatory products; the inner membrane of the calices and pelvis is in both cases injected as in catarrhal inflammations, reddened and relaxed, and filled with an opaque, flaky, grayish, or yellowish-brown fluid.

Externally we find the fascia propria, and even the adipose covering of the kidneys involved in the inflammatory process: the former is easily detached from those portions of the surface which present the vascular injections above spoken of, its tissue is more or less injected and tumefied; the latter is infiltrated with serum, and softened.

This inflammation occasionally affects one kidney only, but very often both are simultaneously attacked: in the latter case, especially, it is liable to terminate fatally, in consequence of paralysis of the renal function with typhoid symptoms, resulting from retention of the urea in the blood; this is frequently complicated with serous effusion into the ventricles of the brain, or into the pulmonary tissue, followed by putrescence; or if the inflammation reaches a certain degree of intensity, suppuration, or an excessive retrograde process, or atrophy may result; or, lastly, the affection may become chronic.

Suppuration is not a frequent consequence. The inflammatory product which has been infiltrated in detached sections, or uniformly throughout the organ, is converted at first into small punctiform or millet-sized spots of white, creamy, or yellow pus, which subsequently coalesce into a small abscess. In its vicinity a renewed reactive process is set up, and we find a red injected halo, varying in size, which gives rise to a similar fusible product leading to an extension of the abscess. The original small abscesses are sometimes found scantily dispersed through the kidney, at others they are grouped together, at others, again, they are thickly sown through the entire kidney; they are then characterized by the surrounding inflammatory halo, and this renders them conspicuous though individually almost imperceptible.

They are always incomparably more numerous in the cortical substance; they here generally retain their rounded shape, even whilst enlarging, whereas in the tubular substance they are converted into elongated striated abscesses.

In the manner just described, as well as by the coalition of several abscesses, we see an extensive purulent accumulation brought about, which may increase so as to occupy one-half or two-thirds, or more, of the kidney. Moreover, there may be one or more of these accumulations, and their existence establishes phthisis renalis.

Renal abscess extends in the most various directions from the inflammation and suppurative fusion spreading through the kidney, and even beyond its sheath; we most frequently find it presenting excavations or sinuses, backwards and downwards; it causes death by exhaustion, or if the progress of the fusion is stopped, the surrounding parenchyma may become obliterated, or in the case when suppuration has extended beyond the latter, the fasciæ of the kidney may become converted into cartilaginous tissue, and the abscess thus be enclosed and be borne for a long period; it may be reduced in size, and may even heal up, leaving a cicatrix; this is particularly liable to result after an opening and a discharge have been effected in a favorable direction.

This discharge may take place:

Firstly, into the cavity of the renal pelvis; the pus is then discharged by the urinary passages;

Secondly, into the peritoneal cavity;

Thirdly, externally in the lumbar region, by means of sinuses of various dimensions;

Fourthly, after previous agglutination of the intestine to the walls of the abscess and perforation, into the cavity of the former; it is evident that the ascending and descending colon, and the sigmoid flexure, are particularly liable to be thus involved, and in second order the duodenum.

Fifthly, renal abscess has also been seen to communicate with the lungs after perforation of the diaphragm; its contents are then expectorated in the shape of urinous-purulent sputa.

These discharges may sometimes take place in various directions at once; a combination of the discharge into the urinary passages with elimination of urine by a false passage—renal fistula, is of especial interest.

Termination in gangrene or gangrenous suppuration is extremely rare; it is more usual to find acute inflammation passing into the chronic form.

Chronic inflammation of the kidney either commences in that form, or is the result of acute inflammation, or, as is most frequently the case, it is the consequence of inflammation of the urinary passages, and especially of the calculous variety. It is distinguished from acute inflammation by a lower intensity of the symptoms, by its smaller extent, and by the variety of stages presented by the coexisting and consecutive inflammatory spots. Chronic inflammation also not unfrequently terminates in suppuration, which is particularly the case with the variety originating in calculous irritation of the renal pelvis; it also frequently terminates

in induration and obliteration of the parenchyma, or induces atrophy of the kidney.

In the former case the coagulable portion of the infiltrated and accumulated product of inflammation is converted into a whitish, fibro-lardaceous, cartilaginous callus, in which the renal parenchyma has entirely disappeared. The kidney is often found increased in bulk, and appears altered in shape, from the irregular accumulation of the product, giving rise to various tuberculated projections. This tissue may here, as elsewhere, subsequently become shrivelled and condensed, and is also, in a few cases, the seat of bone-earth deposit, osseous transformation, ossification.

Chronic inflammation is, like the acute form, frequently followed by atrophy of the kidney; inasmuch as not only its product but the original tissues themselves become absorbed. This secondary atrophy attacks either the entire kidney or sections of the organ, and the consequence is, accordingly, a uniform reduction of its size, or a partial contraction, which gives the kidney a shrivelled and uneven, lobulated surface. The contraction sometimes advances to such a degree, that the kidney appears reduced to the size of a hen's or even a pigeon's egg, it is surrounded by the tunica albuginea, that has become thickened by the inflammatory deposit, and by contraction, and forms a callous sheath of several lines in thickness; on closer examination we find the cortical substance reduced to a mere vestige; the pyramids are diminished to a size corresponding to the dimensions of the organ; the tissue generally is of a pale-red, or here and there of a slate-gray color, denser, tough, and fibro-cellular; occasionally, however, it is unusually dark-red, vascular, and congested, and all the vessels dilated. The calices and pelves are uniformly enlarged, the ureters contracted, their parietes shrivelled and thickened, and here and there approaching to obliteration, or actually obliterated.

Inflammation of the kidneys, with its consequences, has occasionally been discovered in new-born infants; but its frequency and importance are much more considerable at maturity and at the advanced periods of life.

3. *Bright's Disease of the Kidney.*—This affection of the kidneys, which has been named after its discoverer, Bright, and has of late been extensively investigated, is of extreme importance. It has been termed granular degeneration, by Christison, and néphrite albumineuse, by Rayer. We treat of it in connection with inflammatory affections of the organ, for reasons which will appear in the sequel.

It is generally a chronic disease; however there are numerous cases that incline to an acute course, and some equal, or even exceed, acute inflammation in rapidity.

It assumes very different forms, which have reference either to the degree and rapidity of the disease, or to its stage of development; the former bear a close relation, first, to the amount of local reaction in the renal tissue, and, secondly, to the dyscrasic state of the blood. We shall commence by describing the various phases which the disease presents as distinct forms; we shall then examine its complications, their course, stages, degrees, and transitions, and lastly, arrive at a general analysis of the disease.

The cortical substance is that which is primarily and chiefly affected; in the course of the disease, however, the tubular substance also becomes involved in the manner which will be immediately delineated.

First form.—The kidney appears enlarged, swollen, heavier; the cortical tissue is almost uniformly infiltrated with dirty brownish-red, turbid fluid, and the bloodvessels, with the tissue immediately surrounding them, are delineated on this background in the shape of spots, or streaks of a darker red. Other red spots may be visible, which are owing to extravasations of blood into the tissue,—ecchymoses. The pyramids, however, present a similar though darker discoloration, with dull-red striæ. The entire parenchyma, but more especially the cortical substance, is peculiarly pulpy and friable, and the surface, presented by section or fracture, yields a reddish-brown, limpid, delicately flocculent and opaque, sanguinolent and slightly viscid fluid. The organ generally is characterized by a turgid though flabby state. The fascia propria, from the injected state of its vessels, but more from the exudation of blood into its tissue, is of a dirty red color, and is easily detached; the mucous membrane of the calices and pelvis is similarly reddened and tumefied; and their cavity contains a thin, muco-sanguinolent, turbid, urinous fluid.

Second form.—Besides the increase in size and weight found in the first variety, the cortical substance presents an infiltration of a grayish or grayish-red, or yellow, viscid, and turbid fluid, which pervades it uniformly or in diffused spots; the color of the tissue corresponds, and if more carefully examined, an indistinct, dotted, or linear arrangement is perceived. At the same time, small punctiform or striated ecchymoses are found, which are the more conspicuous the paler the color of the infiltrated tissue. The tissue frequently presents the infiltrated and pallid appearance in some parts, whilst the hyperæmia and ecchymoses predominate in others; this constitutes the combination of partial anæmia and hyperæmia, alluded to by authors as a special variety. The organ appears of diminished firmness, but this character is less marked here than in the first form. The renal fascia observes the same bearing, the mucous membrane of the pelvis and calices of the kidney is of a roseate hue, and tumefied; and the latter contain a flocculent, turbid, yellowish or reddish-white fluid.

Third form.—There is considerable enlargement and increase in weight; the cortical substance is completely anæmic; and only a few solitary dilated vessels, bearing an asteroid, convoluted, or striated appearance, are seen in it. The cortical portion presents an increase in diameter of from five to nine lines; its surface is smooth and slightly glossy; it is tense, friable, and infiltrated with a large quantity of opaque, milky-white, or yellowish fluid. The superficial layer more particularly, but also the deeper-seated parts, are found to be made up of white or yellowish-white, loose, tense granules (Bright's granulations), of the size of a poppy-seed, or a pin's head; in the neighborhood of the pyramids these granulations assume a linear appearance.

The increase of the cortical substance either extends to the base of the pyramids only, or affects those portions also that dip down between the latter; by this means the pyramids, and more particularly their apices, become compressed. The pyramids are of a pale-red color, and

from the granular cortical substance forcing its way between the tubuli and separating them, the basis of the pyramids presents a frayed or unravelled appearance, resembling a plume with dependent feathers, or a sheaf of wheat.

The renal fascia is easily detached; its tissue is swollen and opaque, the mucous membrane of the calices and pelvis of the kidney is reddened, and there is a milky, turbid, viscid fluid in their cavities.

Fourth form.—The increase in size and weight is very considerable, and the consistency of the tissues is much diminished; the cortical substance is very tense, and here and there appears almost fluctuating; its tissue is completely anæmic and very friable, and gorged with a large quantity of milky-white or yellowish juice. The granulations exceed the size of millet-seeds, and equal that of hemp-seeds; and as this enlargement is chiefly effected in the peripheral layer, they project from the surface of the organ, and give it a racemose appearance. Occasionally, we find this increase of size occurring with great rapidity in sections, and we then have an accumulation of granulations shooting like a cauliflower from the surface, and producing irregularities and nodulated protuberances upon the kidney. The granulations are very soft, tear and dissolve upon the slightest touch; the renal sheath is almost unattached, the pyramids are of a pale-red color and undefined, and the reddened calices and pelves contain a viscid creamy fluid.

Fifth form.—The kidneys are enlarged or of the normal size, or they may be reduced in size; their surface is granular and racemose, or whilst certain portions present the nodulated and prominent appearance, others are irregularly furrowed, indented, and cicatriform. The cortical tissue is coarsely granulated, looser in texture, very vascular and congested, and the vessels are varicose; or else we find it, as in the case of a diminution of the organ, of pale-yellow or ashy hue, exsanguineous, of coriaceous density, and mainly of a fibro-cellular texture; the indentations at the surface here and there present a similar tissue, of a whitish or slaty color. We also not unfrequently see cysts, containing the most various substances, and varying in size from that of a poppy-seed to that of a pea or nut and more, scattered through the cortical structure.

In the former case the attachment of the fascia propria is slight, in the second it is more intimate: the fascia is thickened, and the adipose layer indurated. The pyramids are small and atrophied, of increased density, and generally of a dirty brown color; the calices and pelvis are slightly contracted.

Sixth form.—The organ is but little increased in size and weight, the cortical substance only presents a few undefined patches of a paler color, and the prevailing hue is either pale red, or it is found on closer examination to offer transitions of a pale red, a white, yellow, or ashy color. It is infiltrated with inspissated matter, resembling thick cream or coagulated albumen; and not only does not present greater laxity of texture, but is of the normal or even of increased consistency.

The fascia propria is but slightly less adherent at these points than in the healthy condition, and the pyramids, as well as the calices and pelvis, are normal.

Seventh form.—The increase of size is commonly trifling ; occasionally there is partial atrophy and diminution. There is increase of density and consistency. As in the last variety, the cortical substance only presents patches of a dull white color, which have no defined borders, and are often very extensive ; it arises from a coagulated, albuminous, larda-ceous-looking substance, in which no trace of the renal tissue remains. We here find considerable swelling of the kidney, owing to the copious deposition of the morbid growth ; or the organ otherwise seems shrunk, and presents the appearance and consistency of fatty cartilaginous tissue. One or more of the pyramids occasionally undergo a similar metamor-phosis. The fascia propria is agglutinated to the diseased portions of the kidney, and thickened ; the lining membrane of the calices and pelvis is tumefied.

Eighth form.—The kidney presents but a slight increase of size, or is of normal dimensions, but always considerably indurated. The general hue is a dirty red or brownish-yellow, and the cortical substance pre-sents a fatty waxy gloss, is unusually hard and brittle, and infiltrated with an albuminous, lardaceous, and transparent substance. Occa-sionally a whitish flocculent deposit is seen in the tissue, of the shape of fine granular dots and lines, giving to the surface and sections a marbled appearance.

We have thus enumerated the forms which, in a general point of view, we think it proper to class under Bright's disease. The first seven forms undoubtedly belong to the latter, if the totality of the symptoms, as they appear in the living subject, be considered : they also occur complicated with one another, and the second, third, fourth, and fifth forms more particularly represent Bright's disease and Christison's granular degene-ration of the kidney. In the latter form the disease is generally chronic, though with an acute tendency and occasionally exacerbations ; the se-cond, third, and fourth forms represent progressive stages of degrees of the metamorphosis occurring in Bright's disease : they vary in duration, and pass from one to the other either gradually or, as is frequently the case, with very tumultuous symptoms. Each of these stages may prove fatal. The fifth form is the last link of the metamorphosis ; with it the process becomes retrograde, and the disorganized tissue of the viscus presents the condition of secondary atrophy. The different varieties are not un-frequently complicated with one another ; and we thus find the first de-gree (second form) attacking one kidney, or a section of one kidney, whilst the other kidney, or the other sections, present the metamorphosis of the third or fourth degree (third and fourth form). The peripheral layer of the cortical substance is generally in a more advanced stage than the deeper-seated layers.

The sixth and seventh forms represent the less frequent or chronic varieties of the disease ; the latter (the seventh) must be looked upon as the terminal point of the metamorphosis, as the product of the disease is retained in a state of condensation and organization, and subsequently shrivels up. It is sometimes complicated with the varieties previously spoken of.

The first form is extremely rare, and runs an acute course ; on the occurrence of powerful exciting causes, very tumultuous symptoms are

sometimes induced, which speedily reach their climax, and may terminate fatally on the fourteenth day.

The eighth form is invariably chronic; we shall for the present exclude its consideration from the following remarks, and advert to it subsequently, for reasons that will then be apparent.

The nature of the disease, and the scientific exposition of its characteristic symptoms, have been the subject of numerous discussions, and we neither venture to assume that our remarks will add great weight to the arguments of those who consider it inflammatory, nor do we wish to anticipate further investigations and statements of depth and originality.

We consider the nature of Bright's disease to consist in an inflammatory process, which proceeds from a stage of hyperæmia to one of stasis, and then gives rise to a product, which is not only remarkable by its peculiar character, but which, in well-marked cases, by its excessive accumulation, causes a singular alteration in the appearance and structure of the kidney. It commonly runs, as we have already stated, a chronic course, with occasional exacerbations, but it is sometimes acute. In the latter very important cases, in which, from the tumultuous violence of the exudation, the product is mixed with a large amount of serum, and is generally reddened by the coloring matter of the blood, and in which the characteristic milky or creamy or coagulated substance of well-marked Bright's disease is not formed, we should be obliged to consider the condition as one of very acute simple inflammation of the kidneys, were it not that the characteristic general symptoms and the constitution of the urine established it as a case of Bright's disease.

The whitish or ashy, milky or creamy product, which may resemble albumen in its various degrees of coagulation, and consists of solitary and accumulated molecules, or of more or less globular fibrinous coagula and pus-corpuscles (Gluge), is an albumino-fibrinous substance, with a predominance of albumen; the amount in which it occurs is proportioned to the amount of granular degeneration.

The product may, as in simple inflammation, be deposited at every point of the renal parenchyma external to the vessels, but we find it more particularly in the Malpighian bodies (glands), and subsequently in the urinary tubuli; the granulations of Bright's disease are therefore in reality the Malpighian corpuscles charged with the above-named substance. The more the latter accumulates, the more it interferes with the circulation, hence the peculiar pallor or anæmic condition of the organ.

The cause of the peculiar character of the product is the more obscure, since the question is generally evaded. As the amount of reaction that takes place in the renal tissue does not suffice to explain it, we are led to seek the cause in an anomalous constitution of the blood, consisting in an excess of albumen, which may originate in a decomposition of the fibrine. This becomes the more probable, when we consider that the most frequent exciting cause (cold) appears peculiarly adapted to give rise rather to a change in the blood, than to a disease of the kidneys, and that the infiltration of the kidney, which we have examined as the eighth form, is evidently developed as a sequel of the cachexiæ which we shall shortly investigate, and in complication with similar affections

of other organs (liver, spleen). Although we might offer numerous observations on this connection, the real cause of the development of the renal disease from the crasis of the blood, which often takes place with such extreme rapidity, is to us an enigma. We look upon the anomalous condition of the blood in Bright's disease as the primary affection, which, from a peculiar relation to the kidneys, is followed by the secondary and visible disorganization of the renal tissue; this need not however always ensue, at all events it does not follow as rapidly as the structural disease of the kidney, consequent upon the vegetative disturbance that causes diabetes mellitus. By this means we explain how it happens that the two kidneys are generally attacked at the same time or at brief intervals. Graves is of opinion that the change of texture is induced by the free acids of the urine (phosphoric and nitric acids) coagulating the albumen as it passes into the urinary tubuli.

Bright's disease is distinguished in the dead and the living subject by the following symptoms :

a. We may briefly enumerate the following as occurring in well-marked cases in the kidneys themselves,—increase in the size and weight of the organ, and especially of the cortical substance (the hypertrophy of French authors, a term which may easily give rise to a misapprehension); anæmia, pallor, laxness of the tissues, development of peculiar granulations, inflammatory sympathy of the renal fascia, on the one hand, and of the mucous membrane of the pelvis and calices, on the other.

β. The so-called consecutive symptoms : a constant and considerable amount of albumen in the urine, accompanied by a diminution of its specific gravity (Gregory), a symptom considered by Rayer as belonging to the chronic form only ; a reduction of the solid constituents, viz. the salts and urea, a milky turbid appearance, or if tinged with blood and blood-corpuscles, dark discoloration, eminent serosity of the blood arising from the removal of the albumen, and accompanied by a diminished specific gravity of the serum ; dropsy, which is chiefly manifested as anasarca, marked pallor of the surface, and secondarily as serous effusion into the serous cavities, and especially of the pleura and peritoneum. Of the latter symptoms the albuminuria and the dropsy have long since been the special objects of explanatory attempts.

Albuminuria is considered by Gregory as pathognomonic only when the specific gravity is simultaneously diminished ; it seems to ourselves to consist in a disturbance of the catalytic function of the kidney arising from the homologous infiltration of the renal tissue ; albumen is in part deposited in the channels of the urinary tubuli themselves, as a product of the reaction. There is not, however, a proportionate relation between the degree of the albuminuria and the amount of renal disease, as we may even find the former existing without the latter.

Sabatier, whose views are not materially controverted by Rayer's objection, attributes the dropsical affections to an attenuation of the blood produced by the removal of the albumen. This crasis of the blood must, therefore, be viewed as secondary.

The lower degrees of Bright's disease are curable by resolution, without leaving any traces, like other moderate inflammatory processes. In the advanced stages a cure may be effected, but only with considerable altera-

tions of texture, as manifested in atrophy of the kidney with a racemose surface, varicosity of the vessels, cellulo-fibrous condensation of the tissue, fibro-lardaceous thickening of the renal fascia, and contraction of the pelvis and calices, in induration of the product, and its conversion into a contractile callus. A fatal termination is induced, with a greater or less rapidity, by dropsy, and especially by serous accumulations in the large cavities of the body, by the slow or sudden supervention of serous effusion into the ventricles of the brain, into the cerebral substance, and into the pulmonary parenchyma, by anæmia, by the retention of urea in the blood, or by morbid conditions of other tissues and organs, which present accidental or essential complications with the renal disease and its predisposing cause.

In the case of retention of urea in the blood, the resulting symptoms are owing to the antagonism between the urea and the nervous matter; they consist in coma, delirium, convulsions, and tetanus, and are not unfrequently caused by urinous effusions within the cavity of the cranium.

The complications are chiefly dependent upon causes that operate suddenly or repeatedly, and for a considerable period, such as catarrhs, and particularly bronchial catarrh, rheumatism, with or without endocarditis, and their sequels; the complications may also originate in the secondary disorganization of the blood, and here again we meet with catarrhs, and also with extensive exudative processes, both on the mucous membranes (serous diarrhœa, pneumonia),[1] and, more especially, on the serous membranes, the arachnoid, pleura, peritoneum, and internal coat of the blood vessels (phlebitis). Hemorrhage and apoplexy are of rarer occurrence. There is great difficulty in accounting for the complication with granular liver, and with the ascites resulting from the latter affection. The supervention of Bright's disease as a new complication may probably be accounted for by the greater liability of a previously diseased subject to the reception of noxious influences, whether operating continuously or temporarily; we allude more particularly to the abuse of spirituous liquors, and to cold.

The commonest and most evident cause of Bright's disease is cold, the sudden or constant influence of cold damp air, more especially; at all events, the occurrence of Bright's disease after scarlatina in children and adults, is most frequently due to this cause; the abuse of spirituous liquors is also considered as a cause, though chiefly in connection with the previously mentioned influences; diuretics, though they do not originate, undoubtedly promote the disease.

Numerous dyscrasic momenta are of considerable importance. We advert to the development of Bright's disease, subsequent to exanthematic fevers, particularly scarlatina, to typhus, to tubercular disease and tubercular suppuration, e. g. pulmonary phthisis, to cancerous diathesis, and to the affections which we are about to consider in connection with the eighth form.

The eighth form invariably sets in without reaction, and springs from inveterate scrofulous or rickety disease, but especially from syphilitic and mercurial taint. It presents itself as a constitutional infiltration of

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[1] [Qy. Bronchitis?—ED.]

the kidney, and is associated with analogous affections of the spleen and liver, in the shape of lardaceo-albuminous infiltration; both the nature of this product and the anomalies of the blood and the urine as yet remain a perfect enigma. We have once noticed the complaint as a sequel of intermittent fever combined with a similar condition of the spleen.

4. *Deposits in the kidneys.*—The same circumstances that give rise to deposits or metastases in the lungs, the liver, and the spleen, may induce them in the kidneys. They follow inflammations of the endocardium, and of the lining membrane of the vessels brought on by infection of the blood, arising from absorption of the inflammatory product, or they result from suppuration and gangrene of membranous and parenchymatous tissues produced in a similar manner, or lastly they originate in spontaneous pyæmia. We would again direct especial attention to the deposits arising from endocarditis, as they have not only been overlooked, in the same way as those occurring in the spleen have been, by the most distinguished inquirers, but as of late Rayer has interpreted them falsely, and has viewed them as symptoms of rheumatic nephritis.

They are found in endocarditis, generally coexistent with similar deposits in the spleen, consequent upon primary phlebitis with a purulent exudation, or upon the absorption of pus or sanious matter from ulcerating surfaces or abscesses; they co-exist with deposits in the lungs, the liver, the brain, the subcutaneous, and intermuscular cellular tissue, the interstitial cellular layers of the intestines, and with secondary phlebitis, in the most different portions of the venous system.

There may be only a few, and in endocarditis we generally find one only, or they are as under the last-named conditions, very numerous; in rare cases the kidney is entirely gorged with them.

They occur chiefly in the cortical substance, and here again mainly in its peripheral strata; so that they are at once apparent on the removal of the fascia albuginea; it is only in exceptional cases, and when they are very numerous, that they occur in the pyramids. They vary much in size, from that of an almost imperceptible poppy-seed, to that of a millet- or hemp-seed, of a pea, a bean, or of a walnut; the larger ones present the peculiar form described in the section on the spleen, as exhibiting a pyramidal shape, the base of which is directed towards the surface, the apex towards the interior of the organ; the smaller ones appear as rounded nodules. The intermediate sizes are the most frequent, but when very numerous, they generally remain so small as scarcely to exceed the size of millet-seeds.

They commence in the renal parenchyma as dark-red indurated spots, which correspond in extent to the above-mentioned sizes; they gradually assume a dirty brown, yellow, or yellowish-white color, and are surrounded by a light-red inflammatory halo, which indicates the reaction set up in the adjoining tissue, or if the disorganization advances to a high degree, by a dark-red, discolored ecchymosis. The latter appearance is coincident with a very large number of the deposits, and as we have seen that these must then be very small, we find the renal tissue presenting in the advanced stage of the disease very numerous small red spots, in the centre of which an almost imperceptible yellowish-white spot is discovered.

The further progress of the disease consists in a conversion of the de-

posit into a purulent or sanious fluid, and the abscess may be enlarged by an analogous transformation of the inflammatory halo; the metamorphosis may, however, be benignant, and the deposit become pale, and shrivel up; it may then, together with the involved tissues, be absorbed, or partially retained as a pulpy or cretaceous mass, having a cicatriform cavity with a fibro-cellular investment, or a fibro-cellular callus, which corrugates and draws down the surrounding parts; a greasy yellow substance or chalky concretion is found buried in the callus, and like the investment of the first-mentioned cavity, this is agglutinated to the tunica albuginea.

The deposit is essentially an exudative process, the product of which undergoes the described metamorphoses; or it depends upon stasis and coagulation of the blood in the capillary vessels, and a conversion of the fibrine in the manner above described,—a secondary angioitis (phlebitis) capillaris. Both metamorphoses are known to be induced by something that is taken up by the blood; and we thus generally see deposits in the kidneys resulting from endocarditis, which go through the second metamorphosis, and heal with loss of substance of a small section of the cortical tissue.

In the case of solitary deposits, the parenchyma, with the exception of that adjoining the morbid product, does not participate in the local process; when they are very extensive, reaction takes place throughout the organ, and is evidenced by tumefaction, enlargement, softening, and and infiltration of the parenchyma; even the mucous membrane of the urinary passages appears congested, reddened, and softened.

5. *Morbid growths. a. Fatty deposit in the kidneys.*—We shall examine this subject under the head of Hypertrophy of the Fascia Adiposa.

b. Formation of cysts.—Although we explicitly exclude the consideration of all encysted tumors which have their origin in a dilatation of the urinary passages, and especially of the calices, we think it necessary at this place to discuss—

a. Cysts, that occur frequently in the renal parenchyma, and which we cannot positively state to be new formations. We allude to cysts which vary in size from that of a millet-seed, pea, or bean, to that of a walnut or even a goose's egg, and which contain a clear, colorless, or yellowish, serous, alkalescent matter, or a substance of a yellowish or brownish color, and of a melicerous or mucilaginous consistency, or again, of a lateritious, chocolate-colored or inky (melanotic) tint. They are formed by a serous membrane, in which a branched vascular network may be traced. They vary in number; sometimes there is a solitary cyst of one of the above-named sizes; generally there are several of different sizes; and in rare cases, they are so numerous, that the kidney, being proportionately enlarged, appears converted into a collection of cysts varying both as to size and to contents, the renal tissues having given way to them. In very well-marked cases a diminution of the urinary secretion, and its consequences, have been observed. These cysts are chiefly developed in the peripheral layer of the cortical substance, and project above the surface of the kidney, so as to be at once perceptible on the removal of the tunica albuginea.

They occur at every period of life, and are sometimes even congenital.

They acquire additional importance if developed in consequence of renal inflammation, especially when this arises from lithiasis, and more particularly in consequence of Bright's disease.

Our own view, and that of German authors generally, is that they are not the dilated terminations of the Malpighian capillary tubes, but that they consist in a conversion of the cellular layer in the Malpighian corpuscles into serous cysts, resulting from the pressure exerted by the Malpighian corpuscles when tumefied and gorged with the inflammatory product of these diseases upon the surrounding strata. The latter during their metamorphosis take up the vessels of the renal coil (Nierenknäuel) for the purpose of the new secretions. It would not be surprising if their contents were occasionally urinous, but we have never been able to discover a trace of urinous precipitates or concretions in them. We have once found a cyst that was seated at the circumference, and was of considerable size, inflamed and ruptured, and its contents effused into the adipose layer.

β. The acephalocyst is a morbid product that occurs in the kidney; less frequently certainly than in the liver, but more frequently than in any other organ. We have no particular remarks to offer in reference to the relations of this variety of encysted tumor, to its contents, or to the surrounding tissues, except that it occasionally reaches the extraordinary size of a fist or a child's head, and that it may discharge its contents in various directions. The following modes of discharge are important:

aa. Communication of the cyst with, and its discharge into, the colon (the ascending or descending colon), and consequent evacuation per anum, and

ββ. The communication of the cyst with, and its discharge into, the cavity of the renal pelves and calices. Small acephalocysts, or ruptured larger ones, may thus be conveyed by the ureters to the bladder, and be evacuated, as is particularly the case with females, by the urethra (mictus acephalocysticus), or they induce obstruction and dilatation of the urinary passages by their size.

γ. The composite cystoidea rarely occur in the kidneys; though when they are formed, they attain a considerable size. We have in our museum an illustrative specimen, in the left kidney of a boy of five years of age.

c. *Anomalous, fibrous, and osseous tissue.*—We find fibroid masses of various extent and shape developed in the products left by inflammation and Bright's disease; and in rare cases a deposition of osseous substance is effected within them, in the same manner as we find occurring in the fibrous exudations of serous membranes. The calcareous concretions are not however in this case laminæ, but irregular tuberculated masses. We also find that a fibrous tissue of recent formation constitutes the external layer of the acephalocysts and composite cystoidea, as well as the base and fundamental structure of cancerous growths in the kidneys.

d. *Tubercle.*—Tubercle exists in the kidneys under two distinct conditions; in both, however, the cortical substance is the chief seat of the deposit.

a. In one case, it is the result of a very high degree of tubercular

dyscrasia; a partial symptom of the development of tubercular disease in many or the majority of organs, and, in that case, frequently the product of a very tumultuous process of deposition. The tubercles are found to exist in great numbers, and occur in the shape of grayish-white, delicate vesicular, or larger, i. e. miliary granulations, surrounded by congested and ecchymosed parenchyma. The entire viscus is swollen, gorged, and softened; it is hyperæmic, and either darker than ordinary, or paler and infiltrated, and the mucous membrane of the urinary passages is reddened and injected. If the morbid process takes place with less intensity and has a more chronic duration, the tubercular matter is found in less quantity, of the size of millet- or hemp-seeds, and surrounded by pale tissue, which presents no trace of reaction either in the vicinity of or at a distance from the tubercular deposit.

This form of renal tubercle occurs as a complication of tubercular deposit in most parenchymatous organs and membranous expansions; and especially in conjunction with tuberculosis of the abdominal viscera, and more particularly of the spleen, the liver, and the peritoneum. Even when occurring under violent symptoms, it is rarely fatal by itself by paralysis of the renal functions, but it becomes so by the universal affection and by the coexistent disease of other organs. This variety of renal tubercle, even when its progress is less rapid, rarely proceeds further than to a yellow discoloration, and never advances to actual fusion. Both kidneys are commonly attacked uniformly.

β. In the other case, renal tubercle is a partial appearance of tubercular disease that is limited to the male urinary and sexual organs. It then generally attacks the testes and the allied lymphatic and prostate glands primarily, and extends from these to the urinary apparatus, i. e. the mucous membrane of the entire tract, to the kidneys, and, lastly, to the supra-renal capsules. It is commonly viewed as possessing a blennorrhoic character or as gonorrhœal tubercle; but post-mortem examinations have not established the fact by demonstrating any peculiarity in the tubercular deposit. It very often supervenes upon a previous tubercular condition of the lungs, or the latter, as well as tubercle in other organs, allies itself to the advanced stage of renal tubercle. This variety of renal tubercle frequently reaches a high degree as regards the number of the tubercles, and their gradual accumulation into extensive groups and coalition into large masses. The viscus is found to have increased in size and is nodulated, and the tissues in the vicinity of the tubercle, or throughout the organ, are in a state of chronic reaction, and appear pale and dense, and infiltrated with lardaceous matter, and the tunica albuginea is thickened. This form of renal tubercle frequently passes more or less rapidly into the stage of softening, giving rise to tubercular ulceration (vomica renis tuberculosa), tubercular suppuration, and tubercular phthisis of the kidneys.

The disease generally attacks one kidney only in a very extensive degree.

e. *Carcinoma.*—Carcinomatous growths occur frequently in the kidneys, and in the primary form. This is particularly the case with medullary cancer, which we find attaining a very large size, whereas areolar and hyaline cancer are extremely rare. Of these, we have observed the

former only twice, in combination with medullary cancer, and the latter only as a secondary affection accompanying universal cancerous deposit.

Medullary cancer appears either in the shape of more or less numerous distinct, rounded, circumscribed masses, varying in size from that of a pea to that of a walnut and a hen's egg, of dense or soft texture (encephaloid), white or variously colored (melanotic); these circumstances generally attend the rapid development of universal carcinomatous deposition, and therefore indicate *secondary* cancer of the kidney; as a *primary* affection, it appears in the shape of a carcinomatous tumor, accompanied by partial infiltration and degeneration of the adjoining tissues; this tumor rapidly increases to the size of a child's or adult's head, forming rounded nodulated masses, which perforate the fibrous sheath, extend to the peritoneum, the lymphatic glands of the lumbar plexus, and involve the periosteum and ligaments of the abdominal vertebræ; the diseased tissue thus becomes fixed, after which occurrence it grows into the cavity of the renal pelves and calices, the renal veins and the vena cava, and causes their obturation.

The latter variety generally remains the focus of the carcinomatous cachexia and the sole cancer occurring in the body, on account of its extreme vegetative power; yet we not unfrequently discover in its vicinity and especially on the peritoneum, the diaphragmatic pleura of the diseased side, and in the liver, isolated cancerous deposits.

An important complication, and one that points to an analogy with tubercular disease, is that with medullary cancer in the testicle of the same side. The two commonly coexist, or the renal cancer is developed shortly after that of the testis.

We have noticed the disease not only in the middle period of life, but both in advanced age and in early youth (as early as in the fifth year). Both kidneys appear equally liable to the affection.

When the growth is effected with great violence, hyperæmia and hemorrhage not unfrequently occur in medullary carcinoma of the kidney, and when it extends into the urinary passages, we find that blood is effused into them also.

6. *Anomalous Contents.*—Besides the anomalies already alluded to, we have to advert to the following morbid contents of the urinary canaliculi.

a. The formation of calculous urinary concretions, which appear in the shape of delicate granular crystals, dispersed through the substance of the kidney, and which consist of lithic acid.

b. Entozoa; these are, besides the animalcules inhabiting the acephalocyst, the cysticercus and the very rare strongylus gigas.

§ 8. *Special disease of the Investments of the Kidneys.*

1. *Hypertrophy of the adipose layer.*—The adipose tissue which surrounds the kidneys may increase in quantity coincidently with a universal increase of the fat of the body, or it may become hypertrophied by itself; in the latter case it may increase to such an extent as to force its way into the hilus of the organ, impede its nutrition, and cause a fatty infiltration of the kidney, accompanied by anæmia and pallor. It appears that rare cases of this description have been occasionally taken for Bright's disease, and this has given rise to the latter being thought ana-

logous to fatty liver. When it has advanced to the highest stage, the kidney presents the appearance of a mere piece of fat surrounded by a mass of adipose tissue, and without the slightest traces of renal organization ; the urinary passages at the same time are atrophied and obliterated.

Independently of universal adipose deposit, we find a larger or smaller excess of fat enveloping the kidneys of old people, accompanied by atrophy of the organ ; it also accumulates when the kidney is affected by moderate but lasting inflammatory irritation, especially that caused by calculi, and in secondary atrophy, and obliteration of the kidney.

2. *Perinephritis.*—This comprehends inflammation of the tunica albuginea and of the fascia adiposa of the kidney. It results from wounds, concussion, and urinous infiltration, and accompanies both the inflammation of the kidneys and that of the pelves and calices.

Inflammation of the tunica albuginea is characterized, as we have already had occasion to state, by development of the vessels of the cortical substance, by congestion and softening, succulence and condensation of its tissue, and by the facility with which it may be detached. It is always combined with inflammation of the cortical substance of the kidney. It is only when the latter terminates in suppuration that the disease in question has a similar issue ; but it frequently leaves a fibroid thickening of various degrees, combined with induration, atrophy, and obliteration of the kidney, resulting from inflammation of the organ.

Inflammation of the fascia adiposa, which is particularly apt to supervene upon the tedious inflammation of the kidneys and their pelves, induced by calculous irritation, has the general characters of inflammation of fatty tissues ; it induces condensation and rusty discoloration ; atrophy and conversion of the fat into a white or slate-colored cellulo-fibrous tissue, which forms adhesions with the thickened albuginea and the peritoneum ; in some cases suppuration and abscess may ensue.

SECT. II.—DISEASES OF THE URINARY PASSAGES.

§ 1. *Defect and Excess of Formation.*—It is self-evident that where one kidney is deficient, the corresponding portion of the urinary passages must be entirely, or at least partially, absent ; but when the kidneys are present, exceptional cases occur in which the ureters terminate in a cul-de-sac in the vicinity of the bladder, and also in the neighborhood of the pelvis of the kidney ; or we may find in addition to a perfect ureter, a rudimentary one developed at the bladder ; or finally, the apparatus may have undergone an arrest of development, and be very narrow, and have very delicate coats.

If the kidneys are increased in number, the urinary channels are also multiplied ; but more frequently the apparent excess is owing to fissure ; the calices opening into two or three pelves, which, in their turn, discharge themselves into two or three ureters. In a less marked degree there is a single pelvis, which is divided inferiorly so as to open into two ureters ; occasionally, these are also found to form partial subdivisions. This malformation, and particularly the fissured pelvis, which is then found partially detached from the organ, frequently accompanies a defective

development of the hilus of the kidney; it also coexists with an elongated state and a transverse division of the kidneys.

The relation of the vesical orifice of the fissured ureters to the bladder varies. They generally coalesce in the neighborhood of the bladder, or within its coats, so as to form a single channel, which communicates with the cavity of the bladder by a single mouth; they rarely open by separate orifices placed behind one another at one side of the trigonum Lieutaudi.

When the kidney occupies an irregularly low position, the length of the ureter is correspondingly diminished.

§ 2. *Deviations of Calibre.*—The deviations of calibre consist in dilatation of the urinary passages, caused by accumulations of urine, which result from obstacles to its discharge, and frequently favored by an inflammatory condition of the mucous membrane, which paralyzes the external contractile layer. It will depend upon the position of the impediment whether the dilatation affects a larger or smaller section of the apparatus. If the former occupies the vesical orifice of the ureter, the entire ureter, the pelvis, and lastly, the calices, become gradually dilated; it is evident, as we shall subsequently examine more fully, that more distant impediments, as, for instance, those placed in the urethra, must also induce dilatation.

The degree in which the dilatation occurs is very various; the higher degrees offer on their own account, as well as on account of various consecutive anomalies, numerous points of interest. Dilatation of the pelves and calices, by exerting pressure upon the renal substance, induces atrophy of the latter. The papilla is first reduced; it becomes condensed and coriaceous, and gradually disappears in the arch of the expanded calyx; the superimposed renal tissue at the same time diminishing in thickness, becoming denser, and assuming a leathery toughness. At an advanced stage the substance of the kidney may be only one, or a few lines in thickness, and even disappear altogether, being converted into a mere membranous sac (hydrops renalis, Rayer's hydronéphrose), with an external lobulated appearance, presenting cells within, and filled with a urinous, variously sedimentary fluid, or with clear serum; the loculi may intercommunicate with one another, in consequence of atrophy or rupture of the contiguous parietes. These sacs sometimes attain, especially in cases which are unaccompanied by inflammation, the size of a child's or an adult's head; but there is no doubt that, after the urinary secretion has ceased, in consequence of atrophy of the renal tissue, and especially of previous inflammation, they may be reduced.

Dilatation of the ureters exhibits every possible degree; the ureter may even attain the size of the small intestine. It is then found hypertrophied, inasmuch as its parietes not only present the average but even increased thickness; and as it is increased in length, and consequently, instead of being straight, appears coiled or bent. At the same time the dilatation is not uniform, as several portions of the ureter are narrower than others, the external cellulo-fibrous tissue accumulating at these points during the dilatation, and offering resistance. To this fact, also, is owing the peculiar direction the ureter assumes, as the curvature or

flexure always occurs at these spots. It may also be observed that the tube rotates upon its axis at these points, a circumstance which further adds to the diminution of its calibre, and offers a new obstacle. The parietes of these cavities and canals always bear, as we 'have already remarked, that proportion to the dilatation, that they must be considered hypertrophied; they only attain a remarkable and extravagant thickness, however, if there is concurrent inflammation.

The following circumstances may induce the occurrence of dilatation: Compression of the ureter at different points by morbid growths, by the impregnated uterus, especially by cancer of the womb which extends to the bladder, by fibroid tumors of the uterus, by enlarged, and particularly by dropsical, ovaries, by accumulation of urine in the bladder itself, or by lasting contraction of the bladder consequent upon hypertrophy of its coats;—contraction of the ureter from tumefaction of its coats, consequent upon inflammation and its results;—obliteration of the ureter, and obturation of the calices, the pelvis, and ureter, by calculous concretions;—cancerous growths forcing their way inwards from without; and, finally, numerous morbid conditions of the bladder, the prostate, and the urethra, which impede the discharge of the urine into the bladder, or the evacuation of the latter.

These dilatations are consequently generally acquired in advanced life, though in the case of original occlusion (blind termination) of the urinary passages, they may be congenital.

In a particular case that we have observed, the pressure exerted by an irregular branch of the emulgent artery, of one line in diameter, that descended from the upper end of the hilus, so as to form an arch over the convoluted transition of the pelvis to the ureter on the right side, caused a dilatation of the former.

The contractions of the urinary passages are sufficiently explained in the above; they are also the result of renal atrophy, and may amount to complete obliteration and closure of their calibre.

§ 3. *Anomalies of Position.*—As a congenital anomaly, we mention the detached position of the single or multiplied pelvis of the kidney accompanying an imperfectly developed state of the renal labia, and especially occurring in cases of anomalous formation and position of the kidney: acquired anomalies of position are brought on by pressure exerted upon the ureter by irregularities of the neighboring organs.

§ 4. *Anomalies of Texture.*—1. Inflammation of the urinary passages have to be first mentioned, and especially—

a. Catarrhal inflammation, both on account of its frequent occurrence, as on account of its consequences and its transition to the substance of the kidneys. As a primary disease, it appears in the shape of inflammation of the renal pelvis and the calices (pyelitis), with inflammation of the kidney, as may be gathered from the description of nephritis and Bright's disease; it may be secondary, owing to irritation exerted by accumulation of urine and urinary concretions on the mucous membrane of these parts (pyelitis calculosa); and it may also be and very often is metastatic, the inflammation of the bladder being transferred to the ureters, the pelves, and calices.

It is either acute, as in the case of complication with acute nephritis, or more commonly chronic, being maintained by lasting and repeated noxious influences, or being the result of a chronic morbid process in the bladder, in which case we meet with temporary acute exacerbations. It is of extreme importance, and renders the following details necessary.

The characters are, in proportion to the degree of intensity and duration, a dusky reddish or brown-red congestive state, similar or ashy discolorations in the shape of solitary spots or islands, or of extensive connected patches, tumefaction and villosity of the mucous membrane, and secretion of a yellow puriform mucus, blennorrhœa.

The longer the inflammatory condition lasts, the more the gradual dilatation of the urinary passages, with hypertrophy of the membranes, increases, both in consequence of the paralysis of the external contractile and irritable layer as from the accumulation of the renal and the morbid mucous secretion.

At an advanced degree, as in the temporary exacerbation of chronic inflammation, the mucous membrane, particularly when subject to irritation by gravel and calculi, which chiefly affect the calices and pelves, appears of a saturated red color, considerably swollen, spongy, and friable; a purulent, more or less sanguineous, fluid is secreted (superficial suppuration), the surrounding cellular and adipose tissues are traversed by varicose vessels, and infiltrated. We find that moderate catarrhal inflammation of the ureters gradually extends to the kidney in the shape of chronic inflammation; it equally attacks the kidney with tumultuous symptoms as acute inflammation when it has reached this advanced degree, and thus proves fatal.

The above-mentioned high degree of inflammation is also found to pass into suppuration of the urinary passages, which spreads from the calices to the tissue of the kidneys, and causes in the latter the formation of abscesses or extensive ulcerative destruction, occasionally urinous infiltration of the renal parenchyma, gangrenous ulceration, and gangrene of the calices and pelvis. We thus find it gradually proceeding in the ureters to perforation, slow infiltration of urine in the adjoining tissues, inflammation, suppuration, necrosis, and in fortunate cases, formation of circumscribed abscesses with indurated parietes.

In these various conditions, the urinary passages contain an alkaline urinous fluid of a pungent odor, which is variously discolored; it is mixed up with puriform mucus or true pus, sanies, blood, and portions of broken-up tissue, and it frequently deposits a sedimentary incrustation upon the inflamed mucous membrane.

In rare cases the advanced stages of the disease terminate favorably in obliteration of the urinary passages. After the cessation of the urinary secretion, consequent upon complete atrophy of the renal tissue, from pressure exerted by the dilated renal calices, or more frequently consequent upon the coexisting chronic inflammation of the kidney, the tissues contract, the parietes become thickened, and the calibre of the passages is gradually reduced, till complete obliteration results. The fluid contained in the cavity of the calices, which consists of blennorrhoic mucus, pus, and urine, the latter being strongly impregnated with alkalies, salts of lime, and particularly with phosphates, first causes an incrustation on

the parietes of the calices, and then becomes inspissated, so as to form a grayish or yellowish-white, greasy, and chalky pulp, which fills the calices; the kidney thus presents the appearance of a loculated cyst, the compartments of which contain the pulp, and radiate from the hilus to the circumference. This pultaceous substance is in due course converted into a dry mortar-like, gritty, dense, calculous mass, and the tissues contracting at the same time, the sac is reduced, the kidney and the efferent channels are obliterated. Occasionally this metamorphosis is observed to take place in one or more detached calices.

Occasionally laminated, corded, nodulated, and amorphous bony concretions are formed in the membranes of the renal calices and pelves, after these have been previously converted into a fibroid or cartilaginous tissue by the inflammatory process; the same may occur in the ureter, though we have not observed it ourselves.

b. Exudative inflammation.—This is on the whole an unusual occurrence, and as far as we are able to judge, invariably a secondary affection; we have never met with a case of idiopathic croup of the urinary organs. It is found complicated with products of the most various plasticity, following typhus, exanthematic diseases, more especially variola and scarlatina, exudative processes in other tissues, as diphtheritis and acute tuberculosis, and purulent infection of the blood; it is very frequently the consequence of extreme disorganization of the blood (especially the so-called status putridus), and then appears as hemorrhagic exudation with purple or dark-red discoloration, sanguineous infiltration, friability and solution of the mucous tissue, and hemorrhage. It may extend over a large surface, or be confined to isolated spots, and it not unfrequently implies gangrene.

2. *Morbid growths.*—*a.* Fibroid tissue and calcareous concretions result from chronic inflammation of the urinary passages in the manner above described.

b. Cysts appear to be more frequent in the urinary passages than they are in and upon other excretory ducts. Without referring to older cases, we may notice two that have been observed in the Vienna Hospital. They represent cysts of the size of millet-seeds or peas, developed under the mucous membrane, and either grouped together or solitary, containing a colorless or yellowish serous fluid, in which is found a soft glutinous or hard nodule, varying in size, and resembling amber or horn; these cysts and the mucous covering occasionally burst, which is proved by the concretions having been discovered unattached in the bladder. They were found chiefly occupying the ureters, and in one case the pelves and calices of the kidneys.

c. Tubercle.—This occurs as tubercular affection of the mucous membrane, and is always a symptom of tubercular disease that has spread from the male genitals to the urinary organs. The earlier stages and the chronic course of the disease are marked by gray millet-sized granulations in the submucous cellular tissue, which speedily become yellow, soften, and after perforating the mucous membrane within a ring of reactive inflammation, give rise to small circular ulcers, which but rarely enlarge to the dimensions of a pea or a bean. When the disease sets in with great violence, the mucous membrane is attacked in larger sinuous

or annular patches, or becomes infiltrated throughout with the tubercular product of inflammation, which is at once detached as a cheesy purulent mass. The mucous membrane is, under these circumstances, converted into a thick, yellow, fissured, and purulent layer, the external cellulo-fibrous layer of which presents a lardaceous character; the calibre of the canal is enlarged. At those parts which are not affected by this degeneration, we not unfrequently find numerous aphthous erosions, re-sembling those observed in pulmonary and laryngeal phthisis.

Tubercular suppuration occasionally passes from the pelvis of the kid-ney to its parenchyma, and it here not unfrequently meets with soften-ing tubercles, or even with tubercular abscesses.

d. Cancer.—Cancer occurs very rarely as a primary disease of the mucous membrane of the urinary passages, and never except in company with one or several cancerous formations in other organs already in a process of development; in these cases it affects the calices and pelvis of the kidney, and chiefly assumes the medullary or fungoid form.

The parietes of the urinary passages are very often involved in a secon-dary degeneration by the encroachment of cancerous growths from with-out; the calices and pelvis being attacked by carcinoma of the kidney, the ureters by cancer of the uterus. Their cavities are narrowed by the cancerous products, and even entirely closed up.

SECT. III.—ABNORMITIES OF THE URINARY BLADDER.

§ 1. *Defect and Excess of Formation.*—Arrest of development occurs in various forms and degrees.

Complete defect is a very rare occurrence; we may meet with it accom-panying a very imperfect development of the kidneys, with absence of the urethra, and commonly also as a complication of formative defects of other organs. If, under these circumstances, the ureters are well formed, they open at the navel, into the rectum, or the vulva.

Occasionally the bladder is very small, whilst the other portions of the urinary apparatus are of normal size; its parietes are then imper-fect; it is, in fact, represented by a delicate mucous bag, a mere dilata-tion of the ureters.

The various fissures of the bladder are other forms of arrest of deve-lopment. We allude, first to the very rare cases of fissure or division of the bladder by means of a perfect or an imperfect partition in the median line, the so-called double bladder. That variety of this species of defect is much more frequent, which has been termed, from its appearance, ectrophia or inversion of the bladder. It is the result of a fissure, or a defect of the anterior vesical parietes, and is not unfrequently associated with fissures of adjoining viscera in the mesian line. It is more parti-cularly accompanied by a defect of the symphysis pubis—in the female sex by absence of the anterior commissure of the labia and the clitoris; in the male sex, by fissure of the urethra on the dorsal surface of the penis, or epispadiasis. In the case of inversion of the bladder, we find in the hypogastrium, immediately beneath the navel, which is always placed very low, a red, mucous, dilated spot, the edges of which coalesce with the common integument: in the male sex it passes downwards, so as to

terminate in the fissure of the urethra; in the female it is surrounded by two diverging tumors which represent the labia, and it terminates in the lamina of the general integument which invests the rima vulvæ. The ureters open upon this mucous surface, and their orifice is generally found at the inferior half.

The exposed vesical mucous membrane and, owing to the constant stillicidium of urine from the ureters, the neighboring cutaneous surface, become irritated, reddened, and excoriated. In a very old preparation taken from an adult, which has been transferred from the Anatomical Museum of the University to the Pathological Collection, I find the former in a state of fungoid degeneration.

When the fissure of the urinary bladder occurs in an opposite direction, and is accompanied by fissure of the genital cavities and the rectum, we obtain the formation of cloacæ in their various degrees. The urachus may remain patent to a certain distance from the bladder, or throughout its entire extent.

We have also to allude to defective development occurring in the shape of unusual contraction of the vesical orifice, or atresia vesicæ.

In biventral monsters, the bladder is found more or less competely double.

§ 2. *Deviations of Size and Form. Hypertrophy and atrophy of the bladder.*—With the exception of the above-mentioned congenital smallness of the bladder, and the congenital dilatations of the organ from contraction or atresia of the urethra, the anomalies to be classed under this head are all acquired; they are the conditions of permanent and excessive dilatation and contraction.

Dilatation of the bladder is seen under various forms. It may be uniform and general, and in solitary cases attains such an extent, that the bladder is represented by a fluctuating paralyzed sac, with relatively thickened parietes, filling the entire pelvis and hypogastric region. It is caused by accumulation of urine, consequent upon insensibility and paralysis of the bladder, but more particularly by mechanical obstacles in the neck of the bladder and in the urethra; in the last case especially, that extreme degree is developed which is always accompanied by hypertrophy of the parietes.

Dilatation of the ureters is a consequence of this affection; it proves fatal by inflammation resulting from the influence of the stagnating and decomposed urine upon the mucous membrane, by the consequent suppuration and gangrene, and especially by peritonitis.

Dilatation occasionally affects in a greater or less degree certain portions, or predominates in certain directions; thus we find lateral expansions at the fundus vesicæ, and saccular indentations produced by the pressure of calculi at or posterior to the triangle of Lieutaud.

An important variety of partial vesical dilatation is presented to us in the hernial dilatation, or acquired diverticulum of the bladder. It is always developed in a bladder the muscular coat of which is hypertrophied, and this hypertrophy, being accompanied by increased irritability of the bladder, affords an evident and intelligible explanation for the predisposition. The vesical mucous membrane insinuates itself between the

fissures left by the rounded or hypertrophied fleshy columns, is gradually forced through them, and forms saccular appendages to the bladder, which increase by degrees, and attain a size varying from that of a walnut or hen's egg to that of a fist or a human head. Their cavity at first communicates with the bladder by means of an elongated rhomboidal opening, and the more they increase, the more the latter, being enlarged at the same time, is converted into a round sphincter.

These diverticula occur principally at the lateral portions and near the vertex of the bladder; they are also found at the posterior surface, and may frequently be seen at all these points at once. The diverticulum is very rarely developed in the triangle near the perineum. Its parietes are formed of the mucous membrane of the bladder, which, under certain self-evident circumstances, is invested by the peritoneum. Sometimes a few muscular fibres traverse the diverticulum, which circumstance may cause it to be viewed as congenital.

If there happens to be concurrent calculous disease of the bladder, the diverticula acquire additional importance, as the calculi may pass into them, or be formed within their cavity, and either be firmly grasped or float unattached. The mucous membrane of small diverticula is frequently the seat of chronic inflammation, causing a muco-purulent secretion, and followed by ulcerative perforation and the formation of sinuses between the vesical coats; these sinuses traverse the trabecular structure of the muscular coat in the most various directions.

Permanent contraction of the bladder occurs in various degrees as a consequence of enduring irritation, e. g. by a calculus; or of increased irritability of the mucous membrane from inflammation. The longer these influences last, the more the parietes increase in thickness and hardness, so that they not unfrequently present the appearance of a ball contracted to the size of a duck's or hen's egg.

The contraction is at times partial, and may then give rise to a permanent coarctation of the bladder at one or even at several points. The bilocular vesicæ, noticed by ancient anatomists, probably took their origin in a morbid contraction of this nature.

As regards the diameter of the vesical parietes, we pass over numerous morbid conditions which give rise to thickening, and which will be investigated subsequently, and have now to examine the states of hypertrophy and atrophy.

Both are most apparent in the muscular coat; hypertrophy of the mucous membrane is chiefly seen in connection with chronic congestion and catarrh of the bladder, and we shall examine into it more fully in speaking of these affections.

Hypertrophy of the muscular coat takes place in consequence of catarrhal affections of the vesical mucous membrane; of repeated and enduring irritation, especially from urinary concretions; of excessive efforts made to overcome obstacles to the discharge of the urine. The latter may affect either the neck of the bladder or the urethra, and be caused by the pressure exerted upon these parts by enlarged or dislocated organs in the vicinity; as by prolapsus, tumors, and degenerations of the uterus, uterine, vaginal, and rectal cancer, by the enlarged prostate, strictures of the urethra, &c. The muscular fasciculi are found

thickened, so as to form rounded trabeculæ, which project from the inner surface of the bladder in the shape of a trabecular network, comparable to the inner surface of the right ventricle of the heart (*vessie à colonne*), the mucous membrane insinuates itself within its meshes, unless the bladder be permanently contracted, and finally forces its way through them in the shape of diverticula.

The bladder is at the same time either dilated, or if the irritability of the mucous membrane is increased, it is contracted. In the latter case especially, the entrance of the urine from the ureters is variously impeded, and thus a dilatation of the urinary passages ensues.

We must, however, be cautious not to mistake a bladder with thick walls, which is perfectly contracted after it has been completely emptied, for a case of hypertrophy.

Atrophy of the vesical parietes occurs rarely. The mucous membrane may be reduced to a very delicate, shining membrane, resembling the arachnoid, and the muscular coat disappears, with the exception of a few almost imperceptible pale traces; the contractile power of the bladder ceases, its parietes are in a state of permanent relaxation, soft, thin, transparent, pale, anæmic, and friable. We have twice observed atrophy of the vesical parietes of this description as a substantive disease.

The shape of the bladder is liable to numerous deviations. All the congenital malformations that are connected with the above-mentioned anomalies of development belong to this head, and as acquired malformations, we may mention those accompanying dilatation, especially when effected in one direction, and causing diverticula, those resulting from irregular and constant contraction, and those assuming the cylindrical, cuneiform, or cordate form, in consequence of hypertrophic conditions.

§ 3. *Anomalies of Position.*—These involve the dislocation of the bladder from its normal position, and in various directions, by enlarged neighboring viscera, and voluminous morbid growths in the pelvic cavity, by contraction and malformation (especially that resulting from mollities ossium) of the pelvis; the dragging down of the bladder by dislocated viscera in its vicinity, especially by the prolapsed uterus, and by large morbid growths in the perineum, the position occupied by the bladder in large inguinal, perineal, and vaginal herniæ; the intussusception of the bladder in the urethra, and its prolapsus through the latter in females; the eversion of the bladder in consequence of a rupture affecting both it and the vagina.

§ 4. *Solutions of Continuity.*—We class under this head—

1. Injuries of the bladder by means of cutting instruments, including the surgical wounds caused by cystotomy and puncture of the bladder; the contusions produced by the head of the child during parturition, by obstetric instruments, by splinters of bone arising from pelvic fractures, or by concussion received by a fall or a blow; rupture of the bladder accompanied by more or less diffused infiltration of the vesical membranes and the surrounding cellular tissue, and hemorrhage.

2. The very rare spontaneous ruptures of the bladder resulting from excessive repletion and distension of the latter.

In both cases the termination may vary; in favorable circumstances a cure may result; extravasation of urine into the peritoneal cavity and peritonitis, or urinous infiltration of the cellular tissue, with diffuse inflammation, suppuration, gangrene, and under these circumstances commonly a fatal issue, may take place; or if the secondary processes are circumscribed, abnormal openings may be established, and vesical fistulæ form.

3. The ulcerative solutions of continuity occurring from within as well as from without, together with the consequent and frequent constricted or patulous communications between the bladder and neighboring cavities and channels, the intestinal tube, and particularly the rectum, the uterine and vaginal cavities, abscesses, &c.

§ 5. *Anomalies of Texture.*—Here too the diseases of the mucous membrane are of main interest, as those of the muscular coat are rare in themselves, and when they occur are generally consecutive or secondary. We shall consider them in their proper places.

1. *Hyperæmia of the Bladder.*—Besides the congestion existing as a stage preparatory to and associated with inflammation, we find hyperæmia occurring not unfrequently as a result of mechanical impediments to the circulation in the pelvic veins and the vena cava. It is commonly complicated with hyperæmia of the neighboring pelvic viscera, of the rectum, the uterus, and the vagina; it gives rise to a more copious secretion of mucus in the bladder, to hypertrophy of the mucous membrane, and is followed by a permanent dilatation of the vessels, and habitual congestion. The condition accompanying stases in the hemorrhoidal vessels of the rectum, in the shape of vesical hemorrhoids, is one of this nature.

Extravasation or apoplexy of the vesical membranes, and hemorrhage into the cavity of the bladder, as a consequence of hyperæmia, is a very rare occurrence. Even in those rare cases it is always limited to a few small spots, and they must be carefully distinguished from the dark-red suffusions of the vesical mucous membrane, into which the hyperæmic condition which is followed by secondary exudative processes and gangrene frequently degenerates.

2. *Inflammation. a. Catarrhal inflammation.*—This occurs in the acute form, but more frequently as a chronic affection; it is commonly presented to the morbid anatomist in the latter shape.

Both generally offer the symptoms common to catarrhal inflammations. Relatively to the chronic form, we have the following observations to make:

It may be developed gradually in consequence of repeated attacks of acute inflammation, or be left as a residuary affection after the incomplete cure of the latter; or, as is very frequently the case, catarrhal inflammation results from an extension of gonorrhœal catarrh to the bladder. It may also be induced by the continued irritation of long-retained and decomposed urine, as is the case when the discharge of the urine is impeded; or lastly, by the irritation arising from calculi.

It offers various degrees; from a pale circumscribed redness, occasionally surrounding the crypts only, slight opacity and thickening,

increase of villosity and secretion of a grayish-white liquid mucus, to a dark reddish-brown, slaty or bluish-black discoloration, accompanied by considerable spongy tumefaction, and the secretion of mucus, which is partly vitreous and clotted, partly yellow and puriform (blennorrhœa). The longer the disease lasts, the more the mucous membrane, from its increased irritability and from the permanently increased innervation of the muscular coat, becomes hypertrophied; the cavity of the bladder is diminished in consequence, and if this condition attains a certain point, paralysis of the muscular fibres and consequent dilatation of the bladder ensue.

In this secondary condition, after the affection has lasted a considerable period, a rapid exacerbation of the chronic catarrh is frequently brought on by the irritation exerted upon the vesical mucous membrane by the accumulation of decomposed alkaline urine. The inflammation speedily attains a high degree, and terminates in exudation, fusion of the mucous tissue, suppuration, and gangrene.

Under these circumstances the bladder is found dilated, and filled with decomposed, intensely alkaline urine, mixed up with blood of a brown color, viscid mucus and pus, sanies, lymph, and detached portions of mucous tissue in the shape of discolored flocculi or larger patches. From this liquid, which offers a pungent ammoniacal odor, a soft, pulverulent, mealy sediment, consisting of calculous matter bound together by lymphatic exudation, is deposited upon the internal surface of the bladder. The parts themselves are discolored, and present a dark reddish-brown, greenish-gray, or bluish-black hue. The mucous membrane, when presenting a dark-red color, appears spongy, softened, and pultaceous, is easily detached and bleeds; when chocolate-colored or greenish it is found purulent, infiltrated with sanious matter, or converted into a friable flocculent tissue, which is traversed by the urinary sediment; or if the process of solution is completed, and the mucous membrane has become detached, the surface of the cellular and muscular coats is exposed in larger or smaller sinuous patches, appears frayed and pulpy, infiltrated with purulent sanies, discolored, softened, and friable. Finally, the muscular coat is involved in the suppurative and gangrenous destruction, and general peritonitis ensues; or even before this takes place sinuses are formed between the vesical membranes, the parietes of the bladder are eaten through, and present a cribriform appearance, and the urine exudes into the surrounding cellular tissue and into the peritoneal cavity. The bladder is converted into a paralyzed sac, the coats of which are thickened, though they yield on slight pressure, they are discolored, and infiltrated with pus and sanies.

The disease commonly proves fatal, either directly or by extension of inflammation to the ureters and kidneys.

In other cases the disease has slight exacerbations from time to time, being limited to a more or less circumscribed spot, which undergoes a slower process of suppuration, and at last becomes perforated. If, under such circumstances, the tissues external to the bladder have become the seat of inflammatory action previous to the occurrence of perforation, a diffuse extravasation of urine is prevented in one direction by inflammatory condensation of cellular tissue—in another, by free peritoneal exu-

dation and agglutination to an adjoining organ. The circumscribed suppuration progresses slowly, and induces fistulous destruction of the tissues, and communications between the bladder and the external surface of the body, or with other hollow organs.

Catarrh of the bladder is of importance, under all circumstances, from its extension to the ureters; and, in bad cases, from its complication with renal inflammation. It may also extend to the seminal ducts.

A very important variety of vesical inflammation is that developed in the course of paraplegia; it generally passes into gangrene, and terminates fatally. The mucous membrane becomes the seat of extensive congestion and suffusion, which spread to the submucous cellular tissue and the muscular layer; the bladder assumes a dark-red hue, is friable, dilated, and filled with urine; or it is empty and collapsed, and the mucous membrane is then partly invested with a coat of ill-looking lymph, partly infiltrated with pus, partly fused into a pulpy sanious tissue. The muscular fasciculi are pallid, ash-colored, and friable, and the cellular tissue is infiltrated with pus and sanies. The cavity of the bladder contains a sanguineous, dirty brown, or chocolate-colored urine, of a pungent ammoniacal odor; this is mixed up with the various products of the process, and deposits a white, soft, pulverulent sediment.

This affection presents an extremely asthenic character, and although we are ready to admit that in many cases it originates, together with the concurrent inflammation of the kidneys, in paralysis, we consider that in others the irritation produced by the alkaline urine stagnating in the bladder, is to be viewed as the chief or as a collateral cause.

b. Exudative processes.—Primary croup of the vesical mucous membrane is extremely rare; but secondary exudative processes are by no means as unusual as is commonly thought. The latter occur during the course of exanthematic diseases, especially of scarlatina and variola, during typhus as a symptom of an anomaly and degeneration of the typhous process, in consequence of absorption of pus in the blood, and associated with exudative processes in other mucous membranes.

The affection gives rise to a more or less coagulable fibrinous exudation of varying thickness, or to a viscid, gelatinous, discolored, purulent, or sanious product; it rarely involves the entire bladder, or even a large portion of it, but is generally limited to round spots or striæ. The mucous membrane presents the most various degrees of injection and redness, varying from an almost imperceptible change to complete saturation of some portions, with considerable thickening and tumefaction, and an induration proportionate to the coagulability of the deposit. According to the character of the process, the diseased tissue becomes softened and converted into a pale or dark-red, reddish-brown pulp, or a gelatinous, purulent, or sanious mass; the local process not unfrequently assumes a gangrenous character, and the tissues are then resolved into a putrescent sanies, or become detached in the shape of an eschar.

As the exuded matter coagulates, it not unfrequently takes up urinary sediments, or these are subsequently deposited, and give rise to an incrusted appearance of the coagula or of the bladder.

We see the typhous process occurring in the vesical mucous membrane under various forms:

a. It is rarely presented in the genuine shape, i. e. characterized by a product resembling that formed in the intestinal follicles and in the mesenteric glands.

β. It is frequently met with as a degenerate exudative process in the shape of scattered, insulated, and soft exudations.

γ. It is seen degenerated to an exudative process resembling a gangrenous eschar. Opportunities of observing the complete metamorphosis of the products and their subjacent strata, in the shape of softening, fusion, and separation, are but rarely offered, as the general disease commonly proves fatal prior to these events.

c. Pustular inflammation.—We advert to the rare formation of variolous pustules upon the authority of other observers. We have ourselves not seen pustules in the bladder, even in cases in which the urethral mucous membrane was intensely affected by the variolous disease.

We may at the same time mention the occurrence of small millet-seed vesicles containing a clear serosity, and resembling a miliary eruption; they accompany catarrhal inflammation and slight exudative processes in the vesical, in the same manner as in other mucous membranes, and are noticed chiefly at the fundus and neck of the bladder. It is also an interesting fact that we have found them in many cases of Asiatic cholera, accompanied by painful dysuria, for which alkaline fomentations afforded considerable relief.

d. Pericystitis.—We have already alluded to the more or less diffused inflammation of the cellular tissue surrounding the bladder, which supervenes upon intense inflammation of the muscular coat and suppuration of the bladder (vide p. 173), or is the result of infiltration of urine after accidental or intentional wounds of the bladder, of ulcerative perforation, and of an extension of inflammation from adjoining cellular structures; but we have besides these a spontaneous inflammation of the cellular tissue surrounding the bladder, which is designated as *pericystitis*. Like the inflammatory, suppurative, and gangrenous processes of the subcutaneous cellular tissue, or of the cellular tissue surrounding the cæcum or rectum, it may be idiopathic, though it is more frequently a secondary process; it is to be considered as a localization of pyæmia, which was either spontaneous or dependent upon an absorption of pus, or of a degenerate typhous or anomalous exanthematic process. It spreads with facility through the cellular tissue of the pelvis, to the cellular septum of the rectum, to the anus, and into the scrotum; it attacks the submucous tissue of the bladder, and having passed into suppuration and necrosis, causes an exfoliation of the mucous membrane and perforation of the vesical parietes.

The affection is sometimes of a chronic nature, and then gives rise to induration, callosity, and rigidity of the bladder.

3. *Gangrene of the bladder.*—Gangrene is the result of intense inflammation, brought on by the contact or imbibition of anomalous urine in the affected tissues, in which cases it assumes the appearance of sphacelous fusion (vide p. 173); or it results from contusion, and then we find an eschar formed (vide p. 174),

4. *Softening.*—Besides the fusion of the mucous membrane accompanying the exudative process, we have but once observed a gelatinous

softening of the vesical mucous membrane. It occurred in a case of typhus which had reached the ulcerative stage, and the bladder was found to contain a large quantity (three pounds) of putrescent urine.

5. *Adventitious growths.*—*a.* We have never observed the formation of cysts between the coats of the bladder, or in its mucous membrane, though from their occurrence in the ureters, pelvis, and calices (vide p. 167), we are not inclined to dispute the possibility of the former. We have to remark that the accounts of a discharge of hydatids or acephalocysts from the bladder for the most part depend upon a descent of these growths from the kidneys, or from other organs (e. g. the liver), that have formed adhesions with the urinary passages, to the bladder, from which they are eliminated.

b. Tubercle.—Tubercle of the vesical mucous membrane is a very rare occurrence, and is not even always found as a complication of tubercular affection of the urinary apparatus, which, as we have already seen, is combined with and results from tuberculosis of the sexual organs. When it presents itself on the vesical mucous membrane, it is commonly also associated with tubercle of the urethra and prostate gland.

It assumes the form of discrete granulation only, and is deposited, with more or less reaction and vascularity, under the mucous membrane; it becomes softened with greater or less rapidity, and after perforating the mucous membrane within a vascular area, leaves a small circular ulcer. According to our observations, and owing probably to the rapid development of the tubercular disease in the other segments of this and the sexual system, as well as to the high degree of the universal cachexia, secondary tubercular deposition and secondary enlargement of the tubercular ulcer in the bladder, are found to be very unusual. The cervix and fundus of the bladder are the main seat of tubercle; we sometimes however notice that the bladder is involved in secondary tubercular ulceration by an extension of the disease from the prostate gland.

c. Carcinoma.—The bladder is either attacked primarily by cancer, or the disease is consecutive, having spread from neighboring organs, especially the uterus, the vagina, and the rectum. The latter is by far the more common case.

We have observed the following varieties of cancer:

a. Fibrous cancer occurs but rarely in the shape of cancerous degeneration of the vesical membranes with thickening, cartilaginous induration, and the characteristic metamorphosis of the muscular layer; we have seen it spread over large surfaces, at the side of the bladder, both upwards and downwards, accompanied by carcinomatous degeneration of the female urethra.

β. Medullary cancer occurs in the shape of nodulated morbid growths between the coats of the bladder, and is commonly associated with cancer in the adjoining sexual organs of the female, and with cancer of the rectum. It perforates the mucous membrane, and occasionally gives rise to a characteristic carcinomatous ulcer with raised edges.

γ. The most frequent form of cancer occurring in the bladder presents the appearance of soft, furred, cauliflower-like, vascular, and generally bluish-red vegetations, which bleed on the slightest touch, and are at-

tached by a rounded flattened peduncle; they arise from the mucous membrane and the submucous cellular tissue with delicate fibres, and develope a very fine membranous tissue, within which a whitish or reddish-white, creamy or medullary (encephaloid) mass is formed. They are either isolated or grouped together, and at last coalesce so as to form a very large, loose, fissured, succulent, globular mass (fungus), which fills out the bladder in proportion as the latter becomes hypertrophied and contracted, in consequence of the permanent irritation. They chiefly occupy the neck and fundus, the trigonum and the parts near the urethral orifices; they are sometimes spread over the entire inner surface of the bladder, but they generally make their first appearance at the above-named spots, and it is there too that the large fungoid growths are found. Of several cases we may mention one in which these vegetations occupied and nearly filled the cavity of a diverticulum of the size of a duck's egg, which descended from the fundus of the bladder to the rectum and perineum.

The more they are developed, the more they are liable to produce considerable hemorrhage from their extreme vascularity; with consequent cachexia and exhaustion; they are occasionally found inflamed, covered, and interlaced with lymphatic exudation, and gangrenous.

This variety of cancer is frequently complicated with cancer in other organs; it is especially allied to the cauliflower excrescences occurring upon anomalous serous and fibro-serous membranes, and upon the inner surface of the compound cystoidea or of the peripheral follicles of areolar cancer that have been converted into large sacs; as also to erectile tumors or epithelial formations on other mucous membranes.

SECT. IV.—ABNORMITIES OF THE URETHRA.

§ 1. *Defective Development.*—The urethra is absent in those rare cases in which the entire uropoietic system is wanting, as also in those in which the bladder is deficient; it is also wanting in those cases in which there is a partial deficiency of the bladder, as in cases of fissure, of ectrophy in the female sex, and of cloacal formation. The urethra may be imperfectly developed, presenting on the upper (epispadiasis) or lower (hypospadiasis) surface of the penis, a fissure which extends either along its entire length, or only to a short distance from the external orifice; fissure of the entire dorsal surface of the penis occurs as a complication of eversion of the bladder, that of the inferior surface with fissure of the scrotum. The latter malformation causes a resemblance to the vagina. In other cases a portion of the urethra is deficient, and the latter then terminates in a cul-de-sac, placed at a greater or less distance from the usual point of the orifice in the glans penis; total absence of the urethra equally gives rise to an imperforate penis.

The urethra may, in consequence of a congenital arrest of development, not open externally, but communicate with the cavity of the rectum, or in the female sex with the vagina; or vice versa, it may receive the rectum or vagina at the lower or posterior portion of its parietes.

§ 2. *Deviations of Size.*—They affect, with exception of congenital shortness of the urethra, its calibre only. We find a more or less dilated or contracted condition of the urethra occurring in both sexes as a congenital anomaly, and affecting its entire extent or small portions only; it is of especial importance in the male sex.

Dilatations as well as contractions of the urethra, the latter being particularly frequent and important, occur as acquired conditions.

Dilatation affects the entire canal uniformly or detached spots only; this depends upon the locality of a mechanical impediment, and upon the extensibility of various portions of the urethra. The pars membranacea of the male urethra is liable to the largest fusiform and pouchy dilatations; a uniform dilatation of the entire canal is often brought on by the continued use of bougies.

Contractions of the urethra originate in primary, but more frequently in secondary, textural changes of the urethral mucous membrane of the corpus cavernosum and its fibrous sheath, and we shall have to examine them more carefully when speaking of urethral inflammation and its consequences.

Contractions of the urethra are also brought on in either sex by the pressure of morbid growths, in man by the enlarged prostate, in the female by neighboring organs that have been dislocated, e. g. the uterus, the prolapsed vagina, &c. The passage of the urethra may also be more or less permanently or dangerously narrowed or closed up by products of its own mucous membrane, as well as that of the bladder, e. g. a mucous plug, croupy exudation, renal and vesical calculi, acephalocysts, &c.

§ 3. *Deviations of Direction.*—Among these we reckon the serpentine, angular or inflected, and variously altered course given to the urethra by voluminous herniæ in either sex, by large morbid growths in the vicinity, by the dislocation of neighboring organs (the uterus) in the female, and especially by the enlarged prostate in man; the latter causes a contraction of the urethra, and pushes it aside, or divides it into two passages, which diverge in the direction of the bladder.

Both the pressure which the urethra suffers, as well as the anomalous direction, and particularly the inflection induced, diminish the calibre of the urethra at various points.

§ 4. *Solutions of Continuity.*—We enumerate under this head, wounds of the urethra, contusions and rupture brought on by a concussion or fall, particularly upon the perineum; rupture produced by the passage of large angular calculi, perforations brought on by rude efforts at catheterization, and ulcerative destruction. In all these cases incomplete recovery very often takes place, leaving urinary fistulæ of varying extent, length, direction, and course.

§ 5. *Diseases of the Tissues.*

1. *Inflammation. a. Catarrhal inflammation.*—It commonly commences with a more or less acute or inflammatory stage, and subsequently passes into a protracted or chronic (blennorrhoic) stage. It results from

chemical or mechanical irritation by substances that have been introduced from without, or it may be developed spontaneously in children from a scrofulous, or in aged people from a gouty diathesis, and in either it may be connected with impetigo ;[1] though it has its origin most frequently in gonorrhœal contagion (gonorrhœal catarrh).

We find the anatomical characters to be those belonging to catarrh generally ; in the acute stage there is, according to the violence of the process, redness, injection, tumefaction of the urethral mucous membrane, or secretion of puriform mucus ; in the chronic stage there is tumefaction of the mucous membrane, enlargement of the follicles, relaxation of the sinuses, and a white or colorless secretion. The inflammation is either uniformly diffused over the urethra, or is limited to one or more spots. The latter is especially the case in genuine gonorrhœa of the male urethra ; we here find not only the navicular fossa, but every point as far as the prostatic portion, and especially the vicinity of the bulb of the urethra, liable to become the seat of the disease. When the gonorrhœa is very violent and obstinate, a small tubercular swelling, which results from the deposition of fibrinous matter in the spongy tissue of the urethra, is found at these points of the urethra. This subject has not hitherto received the attention it deserves, either in regard to gonorrhœa itself, or in reference to the pathology of stricture consequent upon gonorrhœa, and to the gonorrhœal ulcer of the urethra.

The terminations and consequences of gonorrhœa are various. The most common result, which is caused by great violence of the affection, by improper dietetic and therapeutic treatment, and by repeated attacks, is condensation and hypertrophy of the submucous tissue, fusion of the latter with the mucous membrane, and conversion of the corpus cavernosum into a white, resistant, fibrous, cartilaginous tissue. The entire urethra sometimes undergoes this metamorphosis, subsequent to repeated and mismanaged attacks of gonorrhœa, but more commonly detached portions only are affected, and this gives rise to partial contraction or stricture.

Stricture of the urethra occurs in various shapes : the urethra is sometimes contracted to the extent of several lines, the parietes presenting a cartilaginous appearance, and the lining membrane being either smooth or having nodulated projections, or longitudinal folds ; sometimes the stricture forms a rounded protuberance or an angular band encircling the entire canal or only surrounding a portion of the circumference ; at others, again, it appears in the shape of an irregular cicatrix, which causes the surrounding mucous membrane to be puckered up.

The strictures may be solitary, or after a recurrence of gonorrhœal attacks, there may be two, three, four, and more. Their seat corresponds to the seat of the previous inflammation. We have a unique preparation in the museum of Vienna, of a urethra of a man who had repeatedly been affected with gonorrhœa; it presents numerous cartilaginous protuberances from the size of a millet-seed to that of a pea, in part coalescing and scattered over the inner surface, as far back as the bulb, leaving the passage however of adequate dimensions.

[1] [See note, p. 22.—ED.]

The degree attained by the stricture varies; we not unfrequently find it so excessive, that the contracted part scarcely permits the passage of the finest bristle.

The essential character of stricture consists in the same alterations of this submucous and mucous tissue which we observe accompanying and following violent inflammation of the mucous membranes, when it involves the submucous cellular tissue; it does not bear any specific character. The inflammation attacks the spongy substances of the urethra at those spots at which the diseased action was most developed, and gives rise to a deposit of the fibrinous matter in its meshes, which induces the above-mentioned swellings in the urethra. If resolution does not ensue this product remains, and the corpus cavernosum is converted above it into a wheal, varying in extent, shape, and thickness, and consisting of fibrous and fibroid tissue; this is the more liable to induce a narrowing of the urethra, as it possesses a great tendency to contract, and the liability increases in proportion as the sound layer of the corpus cavernosum diminishes. The stricture is most considerable when the corpus cavernosum is involved throughout its entire thickness. It is evident that when the metamorphosis affects the innermost layer of the corpus cavernosum only, the gonorrhœa may be followed by dilatation of the urethra, and we actually find this to be the case in violent though diffused gonorrhœa.

The stricture, consequently, consists of the corpus callosum urethræ, which is converted into a fibroid callus with which the mucous membrane, including its epithelial and submucous layer, has become identified. It is in no way related to cancer, and particularly not to so-called scirrhus. However, mechanical irritation frequently brings on excoriation, inflammation of the tissue, and ulceration, which in favorable cases may be put a stop to after the passage of the urethra has been re-established, though it often involves the deeper parts, destroys the urethra, and induces urinary fistulæ.

Strictures maintain a tendency in the urethral mucous membrane to inflammatory attacks, which gradually extend to the bladder, the urinary passages, and the seminal ducts. They also lead to a dilatation of the urethra beyond the contracted part, to dilatation and hypertrophy of the bladder, and dilatation of the ureters.

Those excrescences which are termed warts by medical practitioners, and which are probably polypous or condylomatous growths of the urethral mucous membrane, and which are said to be particularly liable to accompany stricture, are another consequence of gonorrhœa. We have observed them very rarely.

Lastly, we find gonorrhœal inflammation degenerating into ulceration, causing the gonorrhœal ulcer, which has not been as yet sufficiently investigated in the dead subject, and which not unfrequently gives rise to very fine capillary fistulæ.

True polypi, particularly of the female urethra, probably occur as a consequence of repeated and tedious catarrhal affections. I have found them in one preparation in the prostatic portion of the male urethra.

b. Exudative processes.—In very rare cases we find primary croup occurring on the urethral mucous membranes; it induces a circumscribed

or a tubular exudation, according to the intensity of the process, and occurs chiefly in children.

In the course of hectic fever, brought on by suppuration in the vicinity, we occasionally see more or less numerous aphthous exudations and erosions on the urethral mucous membrane.

c. Pustular inflammation.—We frequently observe variolous pustules in the urethra, when the disease is very intense on the general tegumentary surface. As in other mucous membranes, it is accompanied by an exudative process of varying intensity.

2. *Ulcerative processes.*—Besides the gonorrhœal ulcer, the ulcerating stricture and the ulcerative processes, with which the urethra is attacked from without (the prostate), and to which it is more or less exposed in conjunction with the penis, we have to notice the primary syphilitic ulcer—chancre of the urethra. Cicatrices left by ulceration, and especially by the last variety, must be carefully distinguished from gonorrhœal stricture, though this is rendered extremely difficult, as the cicatrix almost invariably induces stricture.

3. *Adventitious formations.*—In addition to the fibroid tissues occurring after gonorrhœal inflammation, and especially in strictures, to the problematic carunculæ or warts of the urethra, we find that tubercle and tubercular ulceration (Tuberculosis urethræ) are formed in the urethra, though only in conjunction with tuberculosis of the entire urinary apparatus. The urethra is also attacked by cancer and cancerous ulceration; in the male sex this accompanies, or is the consequence of, carcinoma of the penis, and especially of the glans.

§ 6. *Anomalous Contents of the Urinary Passages.*—The anomalous contents of the urinary passages are very various, and may be classified as follows:

1. The products of the organic affections of the secretory as well as the efferent apparatus; they are the more intimately mixed with the urine, the nearer the point of their formation is to the place where the latter is secreted, and the greater their capability of suspension and their solubility.

2. The deviations which the urine presents, independent of the first-mentioned admixtures, whether accompanied by a demonstrable disease of the renal texture, or unassociated with any traces of structural disease: they result from an anomaly in the vegetative sphere, and especially in the blood; they may also occur as a passing effect of certain indulgences, and they relate to the quantity and quality, and particularly to the physical characters of the urine.

In reference to 1, we have to notice:

a. The blood and certain of its component parts. The former (hæmaturia) is found in the urinary passages, to a larger or smaller amount, in the shape of rounded or cylindrical coagula of varying consistency, or mixed with the urine in a fluid condition. It appears in consequence of various injuries involving the kidneys and the urinary apparatus, produced by means of cutting instruments, concretions, ruptures, apoplexy of the kidney, the bursting of an aneurism into the urinary passages, or of varicose veins into the bladder, ulcerative corrosion of a vessel, or

bleeding carcinomatous growths in the urinary organs. It results from
hyperæmia, nephritis, Bright's disease, hemorrhagic inflammation of the
passages, and from disorganization of the blood. Sometimes it is not
true blood—blood-globules—but mere hæmatosine, which passes into the
urine from the serum in the kidneys. We also find other constituents of
the blood, such as albumen and fibrine, in the urine.

Albumen is discovered in the course of numerous diseases both accom-
panied by and unassociated with renal disease. In many acute diseases,
albuminous urine is secreted with an excess of lithic acid, and lithate of
ammonia. Albumen is sometimes found with sugar in diabetic urine; it
always occurs in hemorrhage into and inflammation of the urinary pas-
sages, in hyperæmia, nephritis, &c. It is found to a large amount in
Bright's disease of the kidney, frequently mixed up with blood-globules,
or hæmatosine. Its presence is demonstrated by milky turbidity of
urine, by the urine foaming when air is blown into it, by coagulation of
the albumen on the application of heat, the addition of alcohol or nitric
acid, &c.

Fibrine is said to have been found in the urine in some cases of
dropsy; in the case of hemorrhage into the urinary apparatus it forms
coagula of various shapes and sizes, which are easily recognized.

b. Exudations in the urinary passages, assuming the shape of flocculi,
laminæ and tubular concretions.

c. Grayish, milky, vitreous, colorless, purulent yellow (blennorrhoic)
mucus, pus and sanies, may be intimately blended with the urine, caus-
ing it to be variously discolored or turbid, or forming flocculent concre-
tions, and loose, crummy, viscid, glutinous sediments. Mucus appears
in the urine as the effect of acute, but more frequently of chronic catar-
rhal inflammation of the urinary passages. Pus and sanies are the
result of suppuration of the kidneys, with discharge of the abscess into
the urinary passages, and of suppuration, and the formation of sanies in
the latter; or these fluids reach the urinary cavities from neighboring
organs by ulcerated communications; they may also be the consequence
of gangrene, tubercular or cancerous degeneration. We also find in the
urine, besides the above-mentioned substances, epithelial lamellæ, tuber-
cular matter, elementary cells of cancer, &c.

d. It is stated that the urine contains a substance resembling cerebral
fat, when the kidney is affected with medullary cancer. The immediate
condition of this occurrence has not as yet been determined; it is pro-
bably essential that the morbid growth should have forced its way into
the urinary passages, or that it should project into them.

e. Ancient and modern observers have noticed that hairs are some-
times evacuated with the urine; they may be formed within or external
to the urinary organs.

f. Within the most recent period, Curling has discovered a new ento-
zoon, the dactylius aculeatus, in the bladder. A very recent case is
also given of the discharge of cysticerci with the urine; acephalocysts
are frequently carried into the urinary passages both from the kidneys
and from other organs, and are evacuated with the urine.

In reference to 2, we observe that the deviations of the urine, as re-
gards quantity, may consist in excessive or diminished secretion; if the

quantity found in the dead subject be small, it is requisite to ascertain the evacuations that have taken place before death; if considerable, the obstacles to its discharge must be inquired into. Urine presents various anomalies as to quality, affecting both its physical and chemical properties.

a. The color of the urine is either too intense, owing to a large amount of coloring matter, which is generally combined with lithic acid or urea; or it is very pale, and, at the same time, less acid or neutral. The urine assumes a red color from an admixture of blood or its coloring matter; if there is at the same time an excess of acid, it may become reddish-brown, brownish-black, or in very rare cases, which are probably dependent upon an alteration in the hæmatosine, it may even become perfectly black. Biliary matter produces a yellow, yellowish-brown, or even greenish discoloration. We must finally allude to those anomalous appearances of the urine produced by the consumption of various substances that are rich in coloring matter, as beet-root, madder, rhubarb, gamboge, chelidonium, indigo, ink. The urine may at the same time be transparent or turbid; the latter, in so far as it is independent of the above-mentioned foreign admixtures, is proportionate to the lithic acid or lithate of ammonia contained in acid, or to the phosphates in alkaline urine.

b. The odor of urine is either more or less powerful than in the normal condition; thus the pale watery urine is frequently almost without smell, whereas the saturated urine of acute rheumatism or of pneumonia smells very strongly. Occasionally the urine presents the odor of broth or of whey; in diabetes mellitus it has a spirituous smell, owing to the commencement of fermentation, or its odor resembles that of decomposed straw, of putrid matter, or is very pungent. Different odors are perceived after the consumption of asparagus, turpentine, the balsams, leek, assafœtida, &c. In diabetes mellitus, the urine has a sweet taste.

c. Specific gravity.—This is either above or below the normal standard. It is excessive in diabetes mellitus, and very low in diabetes insipidus. In the chronic form of Bright's disease it is diminished, as the proportion of urea and of the urinary salts is diminished, at the same time that the albumen increases; in the acute form it is not unfrequently increased.

3. As regards the chemical composition of the urine, we find that the normal constituents exist in irregular proportions, or that there are new and unusual substances.

a. The watery portion of the urine is in excess in numerous affections of the nervous system, in hysteria, in diabetes insipidus, and according to Rayer and older observers, in advanced age; its quantity is too small in proportion to the solid constituents in the saturated urine of acute diseases, especially at the period at which critical discharges occur.

b. The urea does not, as was formerly believed, bear a direct relation to the coloring matter of the urine, a fact that has been distinctly proved by Prout in some cases of diabetes insipidus. It is more frequently morbidly diminished, as in diabetes mellitus, in Bright's disease, and numerous other diseases that have not as yet been clearly diagnosed, and in which, as the urea disappears, albumen is substituted.

Original deficiency of urea is to be carefully distinguished from that deficiency which results from its decomposition in consequence of stagnation in the urinary passages, from the influence of mucus, purulent secretion, and pus.

c. Uric acid, either free or combined with a base, and especially in the shape of urate of ammonia, is deposited in the form of small crystals, or of a yellow or lateritious powder. It is increased in quantity in rheumatism, gout, and inflammatory affections; in hysterical urine, in the urine voided during the cold stage of intermittent fever, and in numerous other diseases, it is diminished in quantity. If free acid is present in the urine it may be precipitated in the shape of gravel, though not itself in excess.

d. The phosphates (phosphate of lime, phosphate of magnesia, and triple phosphate of ammonia and magnesia) are often present in excess. Phosphate of lime is deposited in the absence of a free acid, and phosphate of ammonia and magnesia, as a basic salt; these form the phosphatic sediments. As the latter salt is formed in consequence of the development of ammonia, it occurs principally in urine containing much mucus, pus, seminal fluid, and other animal substances that are easily decomposed. The lithic acid is, at the same time, proportionally diminished, and the urine is neutral or alkaline.

e. The alkaline state of the urine is of extreme importance; in many cases that have not as yet met with a sufficient explanation, it appears to be the result of a morbid secretion, or it depends upon decomposition of the urine, and presents various degrees. The urine in this condition is commonly pale and turbid. It is particularly alkaline in chronic inflammation of the kidney, and in numerous diseases of the urinary passages; it is so sometimes in a slight degree, and temporarily, in Bright's disease. The alkaline state of the urine in diseases of the spinal cord, in paraplegia, has attracted some attention, and has given rise to the question, whether this alkalescence is the result of a simple derangement of the act of secretion, i. e. whether the urine is secreted as an alkaline fluid; or whether an acid urine becomes alkaline in consequence of decomposition, by means of the products of coexistent cystitis or nephritis. The question has not received a satisfactory reply. Post-mortem examinations have generally demonstrated the existence of the latter series of causes of alkaline urine; the examinations of the urine in living subjects have been either neglected in the class of cases that come under this head, or they have but little value, on account of the insufficient diagnosis of existing inflammation of the urinary passages and the kidneys. The only proofs in evidence of alkaline urine being secreted by the kidneys, are afforded by the vivisections of Krimer and others, in which, after the division of the spinal cord, urine of the appearance of pure water was secreted; and by the clear neutral or alkaline urine passed in hysterical or epileptic attacks. Rayer has found the urine acid in cases of recent paraplegia, unaccompanied by retention of urine.

The following substances are rarely found as constituents of the urine:

a. Purpuric acid, a modification of lithic acid, produced by the presence of nitric acid, and purpurates (purpurates of ammonia and soda), which are said to give a red color to the urinary sediments (Prout).

β. Hippuric acid (Liebig), which has been found in children in the shape of hippurate of soda, and in diabetes.

γ. Oxalic acid is, according to Prout, the result of a decomposition of lithic acid, and occurs as oxalate of lime, in the form of a greenish or blackish sediment, or of gravel or calculous concretion.

δ. Benzoic, butyric, and cyanic acid, cyanurin and melanurin in blue and black urine, xanthic oxide (Marcet), and cystin (Wollaston).

ε. Sugar, in varying proportions, in diabetes mellitus.

ζ. Cholesterin.

η. Numerous medicinal substances.

The formation of calculous concretions in the urinary organs is a matter of extreme importance; it takes place within the kidneys, in the pelvis and calices of the kidneys, in the ureters, the bladder, the urethra, the urachus, and even externally to these passages. The pelvis and calices of the kidney and the bladder are, however, the parts in which calculi are most frequently formed. The latter present considerable varieties, both as regards their physical properties and their chemical composition.

αα. When the concretions are very small they are termed gravel, and may be very numerous or few in number. Gravel may be formed at any part of the urinary apparatus, and even in the kidney. The red variety consists chiefly of lithic acid, the white of phosphates. Calculi are larger concretions, which again differ much as to volume and weight. In size they vary from that of a millet-seed to that of a goose's egg, or a fist.

ββ. Vesical calculi are generally of a globular, ovate, or oval form; they are frequently flattened so as to present a discoid or lenticular shape; if two or more coexist, friction planes are formed giving the calculi when numerous, a polyhedral shape. Large renal calculi are moulded according to the form of their nidus, and assume a branched appearance. In rare cases the calculi are hollow, forming tubular or conchoid concretions. Their surface is either smooth or rough, angular or fissured; or it appears decaying, gnawed, granular, of a mulberry form, or set with sharp, prickly projections, crystalline, &c.

γγ. The number of the calculi present varies; there are generally several renal calculi, whereas vesical calculi are commonly solitary; however, there are cases on record in which fifty, a hundred, nay, several hundred calculi, especially of the phosphatic variety, were found.

δδ. In color, consistency, and texture, they vary much, and these qualities depend upon their chemical composition.

The substances entering into the chemical constitution of urinary calculi are numerous; sometimes one only form the calculus or predominates, at others several are mixed up together, or disposed in layers. They are not all equally frequent.

á. Lithic acid enters into the composition of most calculi, inasmuch as many consist entirely of it, many in part, and as it forms the nucleus of the majority. Lithic-acid calculi are commonly of considerable hardness, smooth, light or dark brown, rounded, and often flattened.

β′. Lithate of ammonia and lithate of soda rarely enter into the composition of calculi. Those consisting of the former are yellow, and of a loose texture; those composed of the latter are white and chalky.

γ. Phosphate of lime rarely forms a calculus by itself.

δ. Phosphate of ammonia and magnesia forms small, friable, white calculi, that have a shining crystalline investment.

Calculi consisting chiefly of the two last-named substances and carbonate of lime, are very frequent. They are white, of a loose texture, and often of a considerable size; they are generally formed in consequence of inflammatory affections of the kidneys and urinary passages, which in their turn are frequently induced by the presence of a lithic-acid calculus, or some other foreign body, which serves as a nucleus for the calculous deposit.

ε. Oxalate of lime forms the mulberry-shaped, nodulated, dark-brown or black, and very hard calculi.

ζ. Xanthic oxide and cystine are very rare. The latter we generally find combined with fat, resin, coloring matter, iron, silica. In rare cases we also find fibrinous coagula, in the shape of carneous or fibrous elastic masses, entering into the formation of calculi.

Vesical calculi are either contained free and unattached in the bladder or are firmly grasped by the bladder, which has become hypertrophied in consequence of catarrhal attacks. They are found encysted in hernial diverticula of the bladder, or lie in saccular expansions of the vesical parietes, which they form for themselves during the contractions of the bladder; they sometimes become agglutinated to these and other parts by means of fibrinous exudations.

Urinary calculi offer mechanical obstacles to the conduction and discharge of the urine, and give rise to inflammations of the kidneys and urinary passages, proportionate to the size of the calculi, and the roughness and irregularity of their surface. They are sometimes, even when of considerable magnitude, discharged by the natural passages, especially in the female; still they more commonly cause severe injuries of the urinary channels, rupture of the urethra, &c. At other times they make their way by inflammation and suppuration into neighboring cavities, as into the rectum, the vagina, or into abscesses, and from these by unnatural passages outwards.

In very rare cases we find urinary calculi enclosed in cartilaginous capsules external to the urinary passages, having either forced their way out of the latter by rupture or ulcerative perforation, or having been formed at the spot where they are discovered, in urine that has been previously extravasated.

Appendix.—Diseases of the Suprarenal Capsules.

The suprarenal capsules are occasionally deficient, especially when there is a deficiency in other organs also. They are not always absent in acephalous monstrosities; and as their absence generally involves the absence of numerous other organs, the fact suggests no distinct interpretation as to their functions. They are, moreover, generally present when one kidney is absent, and this proves that they are perfectly independent of the kidneys and the sexual organs (Meckel); their diseases place them in a more distinct relation with the lymphatic glands.

The fusion which often occurs in the kidneys is not found to take place in the suprarenal capsules.

Accessory suprarenal capsules, indicating an apparent excess of development, are of frequent occurrence. Several flattened acessory suprarenal capsules are then found in the renal and solar plexuses, and on the ganglion of the latter, varying in size from a millet- or hemp-seed to that of a pea.

They are occasionally of great magnitude, a circumstance which calls their fœtal condition to mind, though it may result from morbid affections. On the other hand they may be small; and this may equally be the consequence of a congenital or an acquired anomaly. A reduction of size occurs in the shape of marasmus in advanced age, or at an earlier period of life; the organ shrivels up, becomes tough and coriaceous, its cortical substance assumes a dirty yellow color, its vascular medullary substance is obliterated; or in some cases it becomes friable, of the color of the lees of wine, or of a rusty brown, so as to resemble the spleen of old persons. The atrophy may also be the consequence of textural changes, appearing after inflammation in the shape of induration or obliteration.

The form of the suprarenal capsules is subject to various unimportant deviations; in reference to their position we have to remark, that they do not follow the congenital dislocations of the kidneys, but in these cases invariably retain their normal position.

Their textural diseases have hitherto met with little consideration. Hemorrhage not unfrequently occurs in them, on account of the vascularity of their medullary substance. The suprarenal capsule is found distended in proportion to the amount of extravasation caused by the rupture of a vein; and according to the period that has elapsed since the occurrence of the hemorrhage, we find the blood, more or less discolored and changed in constitution, enclosed within the cortical substance, which has become pale and atrophied, and is finally converted into a fibroid layer.

We scarcely ever have an opportunity of observing inflammation of the suprarenal capsules, except in its terminal stages, suppuration and induration. Some observers have found the suprarenal capsules converted into purulent pouches in the new-born infant, and even in the fœtus (Andral).

The morbid growths not unfrequently seen, are: tubercle and cancerous degeneration; both, and particularly the latter, are found complicated with similar affections of other organs, and especially of the lymphatic glands.

Tubercle commonly appears deposited in the suprarenal capsules in large masses, and either fuses into pus enclosed in a callous sac, or is converted into a chalky concretion, invested by a fibroid tissue, in which all traces of the proper tissue of the organ have disappeared.

Cancer commonly appears in the form of medullary carcinoma, which very frequently involves the neighboring glands of the lumbar plexus, and the kidney, and causes a considerable enlargement of the suprarenal capsule. Hemorrhage occasionally takes place within the parenchyma

of the cancerous growth, and causes it to be broken down into a chocolate-colored pulp.

It frequently happens that the suprarenal capsules become adherent to the kidneys in consequence of inflammation, or of other diseases associated with inflammatory reaction. A much rarer, though very interesting, occurrence is congenital union of the two organs, in which case one tunica albuginea invests the two, and the concave surface of the suprarenal capsule adheres to the kidney by means of short, tense, vascular, cellular tissue.

PART III.

ABNORMITIES OF THE SEXUAL ORGANS.

CHAPTER I.

ON ABNORMITIES OF THE SEXUAL ORGANS GENERALLY.

THE sexual organs are occasionally entirely absent; a defect that is commonly associated with imperfect development of other parts, and especially with acephalia; a more or less important section of the apparatus is often defective, and one of the symmetrical organs, or one half of those organs which unite in the mesial line, may be absent; or again, one of these organs, or halves of organs, may be imperfectly developed, and its cavity contracted or closed up; or the apparatus may be complete in its different constituent portions and not have been duly developed, remaining permanently small and inefficient, so that the individual presents no sexual character.

Another defect of the sexual organs assumes the form of fissure, which is an arrest of various stages of embryonic development. The highest degree of this malformation is presented in the cloaca, which is to be explained as a persistence of the original sinus urogenitalis, or an imperfect separation of the parts that form the latter. A lower degree of this species of deformity is presented in the fissured condition of the sexual organs, in which case the fœtal or female character predominates; we allude to the various fissures of the uterus, of the vagina, the penis, the urethra, or the scrotum, with or without a residuary trace of the urogenital sinus.

From these latter, apparently hermaphroditic formations, which depend upon an arrest of development, those pseudo-hermaphroditic formations, which consist in an excessive development of certain portions of the female organs of generation according to the male type, form a transition to true hermaphrodisia, i. e. hermaphrodisia *per excessum;* in which case certain portions of the sexual apparatus of an opposite sex are superadded.

In addition to the just-mentioned excess of formation we meet with another form in the shape of a repetition of certain sections of the apparatus, which may either present itself as excessive development of volume, or as precocity.

Besides congenital deviations of size, we find many that are acquired; in addition to those varieties which depend upon textural diseases, and particularly upon adventitious growths, they occur in the shape of hypertrophy and atrophy. The uterus in the female, the prostate in the male sex, are particularly liable to be affected by the former; the latter, independently of the process of involution (tabes senilis), which more or

less uniformly involves the generative system, especially attacks the testes and the ovaries, and in a second degree the uterus.

The sexual organs are subject to numerous congenital deviations as to form; the uterus and its cavity are peculiarly liable in the female, the prostate in the male sex, to acquired malformations.

The position of the external sexual organs depends upon the congenital or acquired degreee of inclination of the pelvis, and other malformations. The most important congenital deviation of position of single organs affects the testes; the uterus presents very important acquired irregularities of this class.

Diseases of the tissues are peculiarly frequent in the female organs of generation; and among them the adventitious growths are most remarkable. We shall have occasion to advert in detail to many points of interest, relative to the morbid growths occurring in the sexual organs of either sex.

CHAPTER II.

ABNORMITIES OF THE MALE ORGANS OF GENERATION.

SECTION I.—THE TESTES AND VASA DEFERENTIA.

§ 1. *Defect and Excess of Formation.*—The testes are absent when the entire sexual apparatus is absent; sometimes they are wanting when the other parts are defectively developed, or are represented by a few coils of a seminal duct: lastly they may be in existence, but of small size, and incapable of further growth. In this case the epididymis is particularly small, its ligament elongated, and the entire organ apparently broken up. This is very commonly the case when the testes remain in the abdominal cavity or in the inguinal canal, and there is an apparent absence of testicles (cryptorchis).

The vas deferens may present a malformation, and after diminishing gradually, terminate blindly at some distance from the vesiculæ seminales and generally in the inguinal canal.

Excess of development, in the shape of a plurality of testicles, is undoubtedly very rare: the fact itself is not supported by sufficient proofs.

§ 2. *Deviations of Size.*—Increase of size of the testicles depends upon hyperæmia, upon inflammation and its consequences, i. e. upon the inflammatory enlargement itself, and the residuary product of inflammation and induration upon hypertrophy of the cellulo-fibrous stroma, and upon morbid growths and degenerations of the organ.

Enlargements of the testicle are to be carefully distinguished from distension of the tunica vaginalis.

Besides congenital smallness of the testicle, dependent upon arrest of development, we not unfrequently meet with atrophy of the testicle. It

occurs not only in the shape of marasmus senilis, accompanied by flabby texture of the organ and a dirty yellow color of its tissue, but is found at earlier periods of life as a consequence of exhaustion, of gonorrhœal neuralgia of the testis, and from unexplained influences in the tropics (Larry). The testicle also becomes atrophied in consequence of pressure exerted by effusion in the vaginal sac, by large herniæ, by exudations within its substance, and by morbid growths.

§ 3. *Deviations of Position.*—We have to notice the fœtal position of the testicles within the abdominal cavity, or in the inguinal canal (crypt-orchis). It is important both from being commonly associated with defective development of the testicle, and on account of the doubt arising as to the sex of the individual, as well as on account of the descent of the testicle, which commonly occurs about the period of puberty, and the consequent occurrence of (congenital) inguinal hernia.

In rare cases the descending testicle does not pursue its regular course ; it either passes under the crural arch, or sinks into the pelvic cavity.

§ 4. *Diseases of the Tissues.*
1. *Inflammation.*—*a.* Inflammation of the testicle is a common occurrence ; but nevertheless, rarely a subject of cadaveric investigation. It may be either primary, secondary, or metastatic.

It may also be acute, or, as is more frequently the case, chronic ; it either attacks the entire testicle, or the epididymis, or single lobules of the former chiefly. Accordingly, the tumefaction of the organ is either uniform or irregular ; its tissue is at first more or less reddened, injected, and according to the coagulability of the inflammatory product, either firmer or looser than in the normal condition.

Acute inflammation not unfrequently passes into suppuration ; the chronic form more frequently ends in induration and permanent enlargement of the organ. The orchitic abscess not unfrequently discharges externally by one or more openings, after inducing perforation of the tunica albuginea, and of the agglutinated lamellæ of the tunica vaginalis. The inflammatory product becomes more or less organized, and converted into a fibroid cartilaginous mass, and the resulting induration induces atrophy of the testicle.

b. Chronic inflammation affecting the tunica albuginea, and its processes, in rare cases induces considerable thickening of this fibrous sheath, hypertrophy of the fibro-cellular tissue within the testicle, enlargement and morbid induration of the latter, and finally atrophy of its proper tissue.

The progress of inflammations of the testicle would appear to be sometimes impeded, and a cure brought on, by the pressure which an effusion into the tunica vaginalis exerts.

2. *Morbid growths.*—*a.* We have already found that fibroid tissue occurs as a consequence of chronic inflammation, and its termination in induration.

b. The formation of cysts is very unusual, a fact that acquires special interest from the frequency of their occurrence in the ovaries.

c. Enchondroma is equally rare.

d. An anomalous osseous substance is sometimes developed in the indurated testicle, i. e. in the fibroid tissue ; and assumes the shape of round, tuberculated, or tendiniform concretions.

e. Tubercle.—Tubercle not unfrequently attacks the testicle primarily, and its chief seat is the epididymis. From this point it not only spreads to the vasa deferentia, the vesiculæ seminales, the prostate, and the glands that are connected with the organs of generation generally ; but also to the lymphatics of the abdomen, the thorax, and even of the neck, on the one hand, or on the other to the urinary organs, in the manner previously described (p. 161). In the former case we find the glands aggregated or strung together in large, shapeless, nodulated masses, and infiltrated with cheesy tubercular matter.

Tubercle is developed in young subjects who are predisposed to tubercular affections, in consequence of excessive or unnatural gratification of the sexual desires. The pathological anatomist has been unable to demonstrate its connection with gonorrhœa, or, in other words, to prove the blennorrhoic character of the general morbid affection, as well as of tubercle itself ; and we, therefore, consider the gonorrhœal theory of orchitic tubercle to be wanting in a most essential point.

The affection proves fatal, either by the universal atrophy induced by the effusion of tubercle throughout the lymphatic system, or by the supervention of more or less acute tubercular deposition in the urinary organs, in the lungs, on the peritoneum, and in the spleen.

Orchitic tubercle generally appears in the shape of rounded nodules, of the size of a millet- or hemp-seed, or a pea, which coalesce into larger masses ; they scarcely ever undergo a retrograde metamorphosis, but fuse, and thus establish tubercular suppuration or phthisis orchitica. The increase in size of the testicle varies according to the number of the individual tubercles, and more still according to the size of the tubercular conglomerations. Its surface is irregular and nodulated. The tissue surrounding the tubercle and the tubercular abscess becomes cartilaginous, lardaceous, and tough.

In the same manner as elsewhere, and especially in the lungs, we find inflammation of the serous investment supervening upon tubercular affections ; thus the tunica vaginalis testis is liable to attacks of inflammation, accompanied by tuberculizing exudation of various forms.

Tubercle of the testicle is of extreme interest as contrasted with the immunity from tubercle enjoyed by the ovary.

f. Cancer.—All the varieties of cancer undoubtedly occur in the testicle, but both according to my own observations and those of others, medullary carcinoma is the most frequent. It always gives rise to very extensive degeneration, is very soft, and presents fluctuation ; sometimes it perforates the tunica vaginalis and the skin, and is thus converted into an open cancerous sore.

It generally so completely takes the place of the proper orchitic tissue that no trace of the latter is left ; still many cases occur in which it occupies the interstices of the hypertrophied fibro-cellular stroma of the testicle. It is peculiarly liable to a complication with renal cancer, and also with medullary growths in the cellular tissue surrounding the pelvis and the hip-joint, with medullary retro-peritoneal growths, and finally with universal cancerous cachexia.

The frequency of its occurrence in the testicle, especially as a primary affection, is of interest when contrasted with the rarity of its appearance in the ovary, and with the frequency of cysts and the allied form of areolar cancer, in the latter.

The vas deferens is generally attacked by disease extending to it from the testicle, or the vesiculæ seminales; it is found to be affected by induration and thickening of its coats and ossification, which probably result from inflammation, by tubercle, and cancerous degeneration.

Appendix.—Abnormities of the Tunica Vaginalis Testis.

In consequence of an arrest of development, the cavity of the tunica vaginalis may remain in communication with the peritoneal cavity, and thus give rise to congenital inguinal hernia.

All the diseases affecting the tissue of serous membranes are found to occur here; inflammatory affections of every degree and variety, followed by the most various effusions, are common; and of the sequelæ, adhesion by means of various tissues of new formation, and ossification of the fibroid exudations, are not unfrequent. Among the morbid growths we notice the anomalous fibroid and osseous tissues in the form just mentioned, as well as subserous, fibro-cartilaginous, and osteoid formations, which we sometimes find as free corpuscles in the tunica vaginalis, and tubercle, occurring especially as tubercular exudation; this must be distinguished from tuberculosis of the testicle, with which, however, it is often coincident.

Dropsy of the tunica vaginalis, or hydrocele, is a common disease, occasionally brought on by varicosity and stasis in the venous network of the testicle and the spermatic cord, in which case it has the character of a passive accumulation; sometimes it is the result of slight inflammatory affections of the serous membrane.

SECT. II.—ABNORMITIES OF THE VESICULÆ SEMINALES.

§ 1. *Arrest and Excess of Development.*—The vesiculæ seminales are absent when the testicles are deficient, and are more or less abortive when the testicles are imperfectly developed.

It is stated that they have been found increased in number in cases in which there were supernumerary testicles.

§ 2. *Deviations of size. Of calibre.*—Under this head we class, on the one hand, the dilatations of the vesiculæ seminales and ductus ejaculatorii, resulting from continued catarrhal irritation, which, according to Lallemand, accompanies spontaneous discharges of semen, and on the other, the atrophy and obliteration of the vesiculæ seminales, which may, but does not necessarily, follow removal or atrophy of the testicle.

§ 3. *Diseases of the Tissues.*

1. *Inflammation.*—We not unfrequently have opportunities of observing, in the dead subject, the effects of chronic catarrh and its sequelæ,

upon the vesiculæ seminales; they are, especially, tumefaction and re-laxation of their mucous membrane; secretion of a grayish or yellow purulent mucus (blennorrhœa), dilatation, and, finally, thickening of the parietes. In rare cases we find those portions of the inner surface in which the mucous membrane has been destroyed by suppuration, covered by a whitish or slate-colored, reticular pulp, of a cellulo-fibrous texture, the parietes considerably thickened and cartilaginous, and the cavity contracted and obliterated. This inflammation as rarely degenerates into ulcerative perforation of the vesiculæ seminales, the formation of abscesses in their cellulo-fibrous nidus, into destruction of a neighboring coil, or communication of two contiguous tubuli.

Chronic catarrh occurs chiefly in advanced age, accompanying mechanical hyperæmia of the pelvic veins, stasis, varicosity, and the formation of phlebolithes; as a consequence of chronic vesical catarrh, as a result of repeated gonorrhœal catarrh of the urethra and the neck of the bladder, of excessive venery, and especially of masturbation.

2. We find a low state of irritation developed in a similar manner in the cellulo-fibrous substratum of the vesiculæ seminales; this induces condensation and hypertrophy in the latter, and causes its adhesion to the vesiculæ seminales, which thus become fixed.

§ 4. *Morbid Growths.*
1. *Bony matter* is sometimes deposited in the indurated coats of the vesiculæ seminales, as well as in the terminal portion of the vas deferens (ossification).
2. *Tubercle.*—Tuberculosis of the mucous membrane of the vesiculæ seminales is not an unfrequent disease. When seen in the dead subject, the disease has generally attained such a degree that the mucous membrane appears converted into a thick, yellow, cheesy, lardaceous, fissured, purulent layer of tubercular matter, filling up and closing the passage of the seminal vesicles, whilst the superficial layer of their coats is considerably thickened, and infiltrated with a lardaceous substance. The external investment occasionally becomes the seat of tubercular deposit, and, as this fuses, suppuration and perforation of the seminal vesicles are induced.

Tubercular disease is associated with tubercle of the prostate, the testicle, and the lymphatic glands that belong to the sexual apparatus, as well as with tubercle of the uropoietic system. It prevails during the prime of life, and appears never to occur before puberty; in this it differs essentially from tubercular disease of the uterus and the Fallopian tubes.

3. Cancer affects the vesiculæ seminales only by extension from neighboring organs.

§ 5. *Anomalies of the Contents of the Vesiculæ Seminales.*—The seminal fluid may present various irregularities; it is found mixed with a greater or less quantity of colorless, vitreous, grayish, yellow, puriform mucus, and with pus; if the inner surface of the vesiculæ seminales has undergone any change of texture there may be hemorrhagic exudation, tubercular pus, cancerous sanies, and, lastly, calculous concretions. The pus and sanies may, as in the ductus ejaculatorii, be introduced from

neighboring abscesses, especially of the prostate, after perforation has taken place.

<div align="center">SECT. III.—ABNORMITIES OF THE PROSTATE.</div>

The prostate is generally found to be small when the organs of generation are in an imperfect condition. Its most important anomalies consist in :—

§ 1. *Abnormities of Size.*—And of these the most common is enlargement, resulting from hypertrophy. It is one of the most frequent causes of the urinary obstructions occurring in advanced life. The substance of the gland in these cases appears normal, occasionally a little softened, of a spongy elastic consistency, and succulent, i. e. its ducts contain much secretion ; in other cases it appears tough and coriaceous, without visible alteration of structure. The formation of fibroid tumors (vide p. 198) is often complicated with this benignant variety of enlargement.

The enlargement varies much in degree ; occasionally it is so considerable that the gland attains the size of a fist. The lateral lobes are the chief seat of the enlargement, which affects both uniformly, or predominates on one side ; but the development of a so-called middle lobe (Home) is of greater importance, in reference to the impediment it offers to the discharge of the urine ; it not unfrequently predominates in a most remarkable manner, even when the hypertrophy affects the entire gland. It rises from the posterior section of the prostatic ring, between the two lateral lobes, and, according to its size, projects more or less into the cavity of the bladder. It presents the appearance of a rounded tumor, of the size of a bean, or hazel-nut, which projects into the neck of the bladder ; it may increase to the size of a walnut, hen's or duck's egg, or more, and then protrudes into the cavity of the bladder in the shape of a smooth or rough, nodulated, slightly lobular, rounded or cordiform, pyramidal or cylindrical tumor.

All enlargements of the prostate impose an obstacle to the passage of the urine, both by narrowing the neck of the bladder and the prostatic portion of the urethra, as well as by inducing a change in the direction of the channel, by diminishing its calibre, and by dividing it. The last two malformations are more particularly the result of unilateral development of the gland, and of increase of its middle lobe. The former not only produces a lateral contraction and deformity of the canal in the vertical direction, so as to produce a sickle-shaped fissure, but forces it out of the mesial line to the opposite side ; the middle lobe not only obstructs the internal orifice of the urethra, but often narrows the neck of the bladder by pushing it on one side, or divides it into two diverging passages, which reunite in the prostatic portion of the urethra.

The results of this enlargement are hypertrophy of the bladder, dilatation of the urinary passages, &c.

A diminution of the prostate, with relaxation of the glandular tissue, is observed in rare cases, as accompanying atrophy of the testicles.

§ 2. *Diseases of Tissue.*
1. *Inflammation.*—An opportunity is scarcely ever presented of study-

ing inflammation of the prostate in the dead subject, except in its results, suppuration and abscess, or induration. The former occurs not unfrequently as the issue of chronic inflammation, which exacerbates from time to time. The abscesses, which vary in size and number, generally discharge themselves into the bladder, into the prostatic portion of the urethra, in which case the ejaculatory ducts are destroyed, into the vesiculæ seminales, the surrounding cellular tissue, or the rectum; or they force their way along the urethra to the penis, or into the scrotum.

2. *Morbid growths.*—*a.* We have never observed the formation of cysts in the prostate.

b. Fibroid tumors occur frequently, and generally induce considerable hypertrophy of the gland. They are commonly of the size of a pea, a bean, or a hazel-nut, round or oval, and when deposited in the peripheral layer of the gland, give rise to nodulated protuberances. Although they do not attain an extraordinary magnitude, they are of interest, on account of the relation they bear to analogous growths in the uterus.

c. Tubercle.—Tubercle of the prostate is always complicated with tubercle of the testis, of the vesiculæ seminales, and of the allied lymphatic glands. The softening process gives rise to tubercular abscesses, which are enlarged by the fusion of secondary tubercular deposits and thus extend beyond the gland, causing the devastations spoken of under the head of abscess.

d. Cancer.—Cancer in any shape rarely occurs in the prostate, which is curious as contrasted with the frequency of its occurrence in the uterus. Medullary carcinoma is occasionally found to attack the prostate, and to give rise to considerable enlargement of the gland; it may sometimes perforate the fundus vesicæ, and sprout into its cavity, causing a cancerous ulcer with raised edges, and of varying size.

3. *Anomalous contents of the prostatic ducts.*—The prostatic ducts, in advanced age, very often contain calculous concretions; they are generally very minute, resembling fine sand or poppy-seeds, rarely attain the size of millet-seeds, and still less frequently form conglomerations of the size of hemp-seeds or peas. They present a black, blackish-brown, or yellowish-brown color, are very hard, and generally glossy. Their number varies, but is often considerable, and a section of the gland shows them more or less uniformly scattered through its tissue. The gland at the same time appears very juicy, and the ducts are more or less dilated.

SECT. IV.—ABNORMITIES OF THE PENIS.

§ 1. *Defect and Excess of Formation.*—The penis may be smaller than usual, whilst the remainder of the sexual organs are normal, or themselves imperfectly developed, or it may present some further anomalies depending upon an arrest of development; in the latter case it is reduced in length, as is the case in hypospadiasis and hermaphrodisia; the penis then bears a resemblance to the clitoris.

Fissures of the penis, or rather of the urethra, which sometimes extend to the glans, and to the penis itself, are important. They are termed hypospadiasis and epispadiasis, the former of which is by far the most

common. Both present various degrees, but the first is particularly liable to variations. We here find the fissure affecting a greater or less extent of the urethra from the glans backwards, or even involving the entire penis together with the scrotum; the penis remains in a corresponding state of imperfect development as to size and form; the prepuce is also fissured and small, the glans divided; in higher degrees, the smallness of the organ, the total absence of foreskin, the retraction of the scrotal fissure, and the imperforate condition, induce a resemblance to the clitoris; and mistake as to the sex of the individual will be the more likely to occur if the scrotal fissure leads to a cul-de-sac simulating the vaginal passage. Epispadiasis is a very unusual occurrence, and is either limited to the glans or extends over the entire urethra; in the latter case it is complicated with eversion of the bladder (fissure of bladder).

Excess of development, except as more or less remarkable enlargement of the penis, is very rare; the few observations recorded of two perfect penes placed beside or above one another are not to be credited.

§ 2. *Deviations of Size.*—Atrophy of the penis, accompanied by obliteration of the tissue of the glans and the corpora cavernosa, deserves notice; it is probably always associated with atrophy of the testicles.

An apparent diminution of the penis is presented in the retracted state, induced by large scrotal herniæ, sarcocele, hydrocele, œdema of the scrotum, &c., in consequence of the relaxation and advance of the common integument.

§ 3. *Diseases of the Tissues.*—They affect the glans and the corpora cavernosa of the penis.

We meet with mechanical hyperæmia of all the spongy tissues as an accompaniment of most of the advanced stages of organic heart diseases; we find a similar tumefaction of these parts in cases of asphyxia, especially when produced by strangulation.

Inflammation of the cutaneous investment of the glans, which is generally complicated with inflammation of the internal lamina of the foreskin, gives rise to excoriation, exudation of coagulable lymph, adhesion of the prepuce to the glans, suppuration, and ulceration; when chronic, it induces exuberant formation of epidermis, and if the deeper parts of the parenchyma of the glans are involved, obliteration, cartilaginous induration, and atrophy follow. Inflammation of the coronal follicles induces increased secretion of a fluid, corroding smegma, and follicular ulceration. Ulcers of a specific character present deep, white, striated, more or less hard, cartilaginous cicatrices, which vary according to the size of the ulcerated surface, and the intensity of the surrounding reaction.

Inflammation of the corpora cavernosa, though of rare occurrence, is brought on by contusions or by gonorrhœal metastases; it occasionally terminates in obliteration of the cells, and, by means of the inflammatory product, in the conversion of the latter into a cellulo-fibrous cicatrix; the uniform turgescence of the penis in erection is thus permanently impeded.

Among the morbid growths, we have to notice the warts occurring on the glans, and carcinoma, and carcinomatous ulcers on the glans and the

corpora cavernosa; the former occur frequently, the latter very rarely. Cancer appears chiefly to assume the medullary form; it gives rise to considerable malformation and enlargement, and to ulcerative destruction of the penis.

We find an anomaly in the secretion occurring in the shape of abundant discharge of sebaceous matter, which, in the case of phimosis or a neglect of cleanliness, accumulates on the glans and round the corona in the shape of lamellæ and tubercular masses, and, after long stagnation and decomposition, brings on inflammation, excoriation, and ulceration, or becomes inspissated, so as to form calculous concretions (calculi glandis).

SECT. V.—ABNORMITIES OF THE CUTANEOUS COVERING OF THE PENIS AND THE SCROTUM.

§ 1. *Defect and Excess of Formation.*—As a defect of formation, we notice the occurrence of extreme shortness or contraction (phimosis) of the prepuce; fissure and entire absence of the foreskin in hypospadiasis, and the clitoroid arrest of development of the penis. The scrotum is small when the sexual apparatus is imperfectly developed, and in cryptorchis, and is sometimes only represented by a slightly corrugated cutaneous fold, which shows an almost imperceptible raphe, and occasionally contains adipose cellular tissue. In hermaphroditic formations it is fissured and resembles the labia of the female genitals, in those cases especially in which the two halves are empty, viz., when the testicles have been retained in the abdomen or in the inguinal canals.

Excess of development occurs in the penis in the shape of exuberant formation of skin, as a very long foreskin (occasionally characteristic of a particular race), in the scrotum as considerable enlargement, and in either as extreme thickness of the common integument, with an unusually well-marked and projecting raphe, which is continued upwards on the penis; there is also an accumulation of the tissue of the tunica dartos and of the subcutaneous cellular tissue.

§ 2. *Anomalies of Size.*—Besides the congenital anomalies we have to notice the acquired enlargement of the scrotum resulting from hypertrophy of the tunica dartos, sarcocele, or elephantiasis, accompanied by fibrous induration; in Egypt more especially it attains the most enormous dimensions.

§ 3. *Diseases of the Tissues.*—The common integument of these parts is liable to the primary and secondary diseases to which the skin generally is subject; but it is also liable to primary and secondary inflammatory process of a specific character, to ulcerative disorganization, to induration and condensation, and even to gangrenous destruction. Paraphimosis of the prepuce resulting from inflammatory swelling, and the ulceration which causes the glans to pass through the ulcerated opening, and denudes the glans of its foreskin, deserve special mention. The scrotum is frequently attacked by metastatic processes and by gangrene; it is remarkable for the facility with which it is reproduced; it is also subject to leprous degeneration, discoloration, and to chimney-

sweeper's cancer. The tunica dartos is variously affected in the above-mentioned processes; it is also found to be the seat of œdema, of sanguineous effusion (hæmatocele), of urinary infiltration, suppurative inflammation, fibrous induration, which is sometimes confined to the septum scroti, of urinary fistulæ, and of various morbid growths.

CHAPTER III.

ABNORMITIES OF THE FEMALE SEXUAL ORGANS.

THE EXTERNAL GENITALS.

SECT. I.—ABNORMITIES OF THE PUDENDA.

ARREST of development occurs in the shape of total absence of the pudenda; absence or defective development, i. e. unusual smallness of individual parts, the labia majora and minora, or the clitoris; absence of the rima or of the commissures, i. e. unusual fissures, such as we see at the superior commissure, accompanied by eversion of the bladder and separation of the symphysis pubis.

Excess of development is met with as uniform or partial congenital enlargement of the labia, nymphæ, and clitoris, causing the latter to resemble a penis; as increase in the number of individual parts, as of the nymphæ, and as precocious or extravagant development during puberty.

Congenital anomalies of form affect particularly the nymphæ; like the acquired anomalies, they present several varieties.

The diseases of tissue are primary or secondary; they consist in metastatic inflammatory processes, varying in degree and rapidity, accompanied by increased sebaceous secretion, great epidermal development, excoriation, œdema, superficial and profound suppuration, condensation and induration, gangrene of the external and internal labia; we meet with specific circumscribed inflammation and ulceration of the latter; among adventitious products, condylomatous excrescences occur in them and on the clitoris, varying in size and number, and occasionally producing extreme deformities. We also find hemorrhagic effusion occurring within the labia spontaneously, or in consequence of external violence (sanguineous tumors), and, besides steatomatous (fibroid) tumors, all the adventitious growths occurring in the cellular tissue at large.

SECT. II.—ABNORMITIES OF THE VAGINA.

§ 1. *Defect and Excess of Formation.*—The vagina may be totally absent, or partially deficient; in the latter case there is a cul-de-sac opening externally, or the vagina terminates blindly at a greater or less distance from the labia, or opens posteriorly into the urethra—in this instance the development takes place from both points, but an intervening

portion is defective, thus forming a transition to congenital atresia. When the other parts of the sexual apparatus are atrophied, or certain of its sections, as, for instance, the clitoris, approach the male type, or in cases of hermaphrodisia per excessum, the vagina is not duly developed, and is found rather narrow than short, smooth, and without rugæ.

We must here allude to an apparent excess of development, called the double vagina, or division of the vagina into two channels which lie in juxtaposition to one another. It is produced by a vertical septum that descends along the mesial line of the vagina; and in a low degree is indicated by a more ridge-like elevation of the columnar rugæ. The division of the vagina may be complete, and is then associated with division of the uterus and its orifice, and with a double hymen; or it may be incomplete, and in this case the septum ceases above, and the fornix vaginæ is common to both passages, the os tincæ being at the same time single or double; or else the septum does not reach down to the vaginal entrance, which is protected by a single hymen, and the vagina is single to a greater or less extent; or, lastly, the septum is incomplete, inasmuch as it presents partial defects. The deviation of the septum from the mesial line, which occurs in rare cases, is of interest and importance; the passage on one side may then be imperfect, or have a blind termination above or below. The following case, taken from our collection, is an instance:

Sexual organs of a very imperfectly-developed female of fifteen, who was covered with scrofulous ulcers and cicatrices, and died of tubercular phthisis of the lungs and the intestines. Two very delicate, elongated, fusiform uteri, each provided with one Fallopian tube and one large ovary, unite at the point of the internal orifice at an obtuse angle (uterus bicornis), and are from this point separated by a vertical septum, so that each cervix has its distinct vagina. The two vaginæ descend on both sides of a septum, which is a continuation of the septum uteri, down to the external pudenda, which are closed by a single hymen, the left vagina being considerably wider and presenting larger rugæ than the right. The latter terminates at about the middle of the entire vagina, in a blind sac formed by the septum; the left vagina immediately bulges out to the right in the shape of a single canal. The external organs are, like the uterus, in an extremely undeveloped condition. It is a curious coincidence that the right kidney was absent, the left being at the same time enlarged, and its hilus directed forwards.

The hymen is often too large, owing to excess of development, so as almost to close up the entire passage; it deviates at the same time from its normal shape and mode of attachment, inasmuch as it is generally connected with the internal labia by a small round column, by which means two orifices are formed which lead into the vagina.

§ 2. *Anomalies of Size.*—The congenital anomalies involve a greater or less dilatation, such as we find to be peculiar to some nations; and the contraction which we have spoken of above, the highest degree of which is complete closure.

Congenital atresia, which we have above classed with partial defect of the vagina, is commonly produced by an enlarged hymen, or, in excep-

tional cases, by a horizontal or obliquely placed membrane, which occupies different parts of the passage; if carefully examined we should probably find that it was formed by the adherent parietes of a vagina, ending above and below in a cul-de-sac. This form of atresia would, in that case, have to be considered as partial (and slight) deficiency of the vagina.

The acquired irregularities appear, on the one hand, as unnatural elongation or dilatation; on the other, as shortening or narrowing, amounting even to complete obturation.

The vagina is liable to a uniform or partial elongation, with disappearance of the rugæ and diminution of its arch, in consequence of traction exerted by the uterus or ovaries, owing to uterine tumors or enlarged ovaries that mount into the abdomen, or to morbid growths that force those organs upwards. Prolapsus uteri, tumors projecting into its cavity, especially fibroid tumors, polypi of the uterus, pessaries, and the like, induce dilatation of the vagina.

Shortening or narrowing is the result of injury and loss of tissue that has been intentionally or accidentally induced, of ulceration and the resulting cicatrices. The vagina is also narrowed when the passage is elongated by traction, and its cavity is diminished when the cervix uteri becomes atrophied.

Acquired atresia may be complete or incomplete, and result from adhesion of the anterior and posterior walls of the vagina to a greater or less extent, in consequence of excoriation or ulceration; or it may be produced by flat or rounded cords that pass horizontally or diagonally across the vagina and reduce its calibre. The latter may consist of vaginal folds brought on by traction, or of the membranous bands left after the cure of ulcerative loss of substance.

§ 3. *Deviations in Position and Form.*—The form of the vagina is modified in a manner corresponding to the anomalies which we have first examined, and in a medico-legal point of view we have to notice the unusual forms presented by the hymen after it has been ruptured. Instead of the carunculæ myrtiformes, a more or less considerable annular tumor remains; or if the hymen was inserted into the nymphæ, one half is left so as to form a species of valve, or it is entirely torn out in the shape of a ring.

Among the deviations of position we notice intussusception and prolapsus of the vagina, which affect mainly the anterior wall of the vagina, and the eversion of the anterior or posterior vaginal parietes in vaginal hernia (cystocele vaginalis, hernia vaginalis posterior).

§ 4. *Solutions of Continuity.*—Besides the injuries inflicted by means of cutting instruments, which generally implicate various neighboring organs, and the ruptures caused by concussion and contusion, we have to mention the contusions and ruptures of the vagina occurring during parturition, whether or not occasioned by operative interference, and the loss of substance by ulceration. The contusions or lacerations affect the vagina alone, either superficially or throughout its tissues, or they are associated with contusions and lacerations of the uterus; in the last

case, the injury affects the vagina and the uterus simultaneously, or a laceration of the latter is carried down to the former to a greater or smaller extent. Neighboring organs, and especially the bladder, may also be involved in the solution of continuity.

In difficult or hurried parturition, when the parts have not been properly supported, the vagina, the posterior commissure, and the perineum may be ruptured, and when the parturition is effected by the perineum, the vagina is perforated above the sphincter.

Ulcerative destruction is not always limited to the vagina, but frequently gives rise to communications between the cavities of the vagina, the bladder, or the rectum, or with both at the same time by means of fistulæ or large cloacæ.

§ 5. *Diseases of the Tissues.*

1. *Inflammation.*—*a.* Catarrh affects the vagina very frequently in the protracted acute, or, if blennorrhoic, in the chronic form, and presents the most various characters. It may be a simple benignant catarrh, or have the specific qualities of the scrofulous, arthritic, syphilitic, impetiginous, or gonorrhœal catarrh; it is sometimes complicated with blennorrhœa of other mucous membranes, and is either idiopathic or symptomatic, accompanying various local inflammatory, ulcerative, or degenerate processes in the vagina, the uterus, and neighboring organs.

The vagina appears flabby, its mucous membrane tumefied and pale, invested with a pale thick coating of epithelium, or excoriated and reddened, with enlargement of the follicles, which are surrounded by a vascular ring. It contains and discharges a secretion varying in quantity and quality, and mixed up with the products of the associated inflammatory and ulcerative processes. In its pure condition it is a white, thin, milky, or creamy mucous, which is commonly secreted in considerable quantities, and indicates an abundant formation of epithelium and desquamation, or it appears as a vitreous, grumous, and viscid, or as a yellow puriform mucus.

Catarrh of the vagina is an important disease, not only on account of the extreme loss of fluids which it often entails, but also on account of the imminent danger of its extension to the uterus and the Fallopian tubes, and the consequent morbid affection of these organs. It predisposes to intussusception of the vagina, owing to the relaxation it induces; it leads to excoriation and superficial ulceration, both of the vagina, the external pudenda, the parts in their vicinity, and of the cervix uteri, to closure of the os tincæ, to follicular suppuration, atresia vaginæ, permanent hypertrophy of the follicles, and dilatation of the vaginal vessels. It follows that a cure is effected with extreme difficulty, and that relapses occur very frequently.

b. Exudative processes.—In rare cases primary croup occurs on the vaginal mucous membrane alone; but it exists more frequently in complication with an exudative process on the internal surface of the uterus, in the shape of puerperal disease. As the latter generally predominates, the affection is usually found to have spread from the uterus to the vagina. Exudative processes with various products occur more frequently in patches, or throughout the vagina as secondary diseases, both

as a result of puerperal affection of the uterus, as well as in consequence of an infection of the blood proceeding from other causes, or from a degeneration of the typhous and various exanthematic processes. They correspond to the condition of the blood and its products, and accordingly produce a solution of the mucous membrane and the submucous layer, varying in shape and depth, and not unfrequently resembling gangrenous destruction. A loss of substance may ensue, and to this cause undoubtedly many cicatrices found in these parts are to be attributed. They also not unfrequently extend to the pudenda, the perineum, and the nates, and give rise to extensive disorganization.

We must make special mention of the secondary form of typhus occurring in the vagina. It does not appear to exhibit itself in the vaginal mucous membrane in its genuine form, but is often found degenerated into croup and gangrene. It is remarkable that an existing blennorrhœa, especially if of a gonorrhœal or syphilitic character, exerts a powerful attraction upon it.

c. Inflammation of the submucous cellular tissue of the vagina.—It very rarely appears in the chronic form; it leads to considerable thickening and coriaceous induration of the vaginal parietes; the latter at the same time become less movable, so as to seem agglutinated to the adjoining parts.

2. *Ulcerative processes.*—We here meet with the simple (catarrhal) follicular ulcer, the circumscribed or diffused solution of the tissues resulting from exudative processes, the syphilitic ulcer, the phagedænic ulcer of the os uteri, which generally spreads from the cervix uteri to the vagina, and the true cancerous ulcer. At the cervix we find some other ulcers, of which we shall have occasion to speak more fully at a future period.

3. *Gangrene of the vagina.*—Gangrene is the result of pressure and contusion produced during difficult parturition; it also occurs in the shape of gangrenous eschar and gangrenous or putrid fusion of the mucous and submucous layers.

4. *Morbid growths.*—Their occurrence is altogether unusual, and even the fibrous and cancerous tumors that we meet with are but rarely observed. The cysts that are found in this region are developed in the cellular tissue external to the vagina, and, anatomically speaking, bear a very subordinate relation to the latter.

Fibroid productions almost invariably coexist with similar growths in the uterus; they may be developed in the external fibro-cellular layer of the vaginal parietes, and especially at their posterior surface; they then project with a larger or smaller segment, in the shape of round tumors, into the vaginal cavity. In other instances they are developed in the cellular tissue that is interposed between the vagina and the rectum, and, though in close relation to the vagina in point of origin, project chiefly into the rectum, and more or less obstruct its inferior portion. The latter circumstances are characteristic of the relation in which these morbid growths stand to the uterus and to the accumulations of cellular tissue which occur in these regions.

Carcinoma of the vagina is, in most cases, cancer of the uterus which has spread to the vagina; however it may exist, though the latter is in

a very undeveloped state, and even without it, in the shape of primary carcinoma of the vagina. It belongs to the fibrous or medullary variety, and, in proportion to its growth, induces thickening of the parietes, tuberculated condensation of the internal surface, and corresponding contraction of the passage; the vagina becomes adherent to the neighboring parts, in consequence of cancerous degeneration of the cellular tissue surrounding it and the rectum, and finally cancerous ulceration and excrescences are established. ' The greater part of the vagina generally becomes involved, and the lower portion is prolapsed; the disease extends to the rectum, the bladder, the urethra; by the pressure it exerts it causes retention of the urine and dilatation of the bladder, and, when it has reached the ulcerative stage, recto- and vesico-vaginal fistulæ result.

§ 6. *Anomalies of the Contents of the Vagina.*—Under this head we class, besides the anomalies of the mucous secretion in vaginal catarrh, the products of exudative and ulcerative processes, the contents of the bladder and the rectum, when introduced by fistulous communications, the products of the diseased mucous membrane of the uterus and the Fallopian tubes; blood that may be derived from various sources, and in various states of coagulation, discoloration, and decomposition. The presence of blood assumes particular importance when it is retained by a redundant hymen, or by congenital or acquired obturation; we include in this category pessaries and the adherent calculous deposits, various substances that have been introduced from without, and, lastly, the problematic cases of vaginal pregnancy.

THE INTERNAL SEXUAL ORGANS.

SECT. I.—ABNORMITIES OF THE UTERUS.

§ 1. *Defect and Excess of Formation.*—Complete absence of the uterus must be considered as extremely rare; in most cases in which the uterus was found deficient in the dead or living subject, rudiments of a uterine organ of different forms were discovered.[1]

The most common case of arrest, which is generally considered as absence of the uterus, is that in which the fold of the peritoneum, which is destined for the reception of the internal sexual organs, contains, on one or both sides, posteriorly to the bladder, one or two small, flattened, solid masses, or larger hollow bodies, with a cavity of the size of a pea or a lentil, which is lined with mucous membrane. They are to be viewed as rudiments of the uterine horns, and the Fallopian tubes bear an exact relation to their development. These may either be totally deficient, or terminate in the vicinity of the uterus in the peritoneum as blind ducts, or they may communicate with the uterus with or without an open passage.

[1] Oestr. Jahrb. xvii. 1.

This formation of the uterus, and especially the existence of two lateral, hollow, elongated and rounded uterine rudiments, each of which is connected with a corresponding Fallopian tube and ovary, constitutes what Mayer terms the uterus bipartitus. From each of the uterine rudiments a flattened, round cord of uterine tissue ascends within the fold of the peritoneum, and the two from each side coalesce. The place of the uterus is occupied by cellular tissue, in which a few uterine fibres, derived from the just-mentioned cord, may be traced; it presents the general outline of a uterus, and, reaching downwards, rests upon the arch of a short vaginal cul-de-sac. The external sexual organs and the mammary glands, as well as the general sexual character of the individual, attain a normal development.

If we pursue the progress of these uterine rudiments we find a development on one or both sides; representing in the former case, a uterine half, or a uterus unicornis; in the latter, a two-horned uterus, or uterus bicornis, varying in degree; this is what is falsely called the double uterus, uterus duplex. These, and the following uterine formations which depend upon fissure, offer considerable interest.

The one-horned uterus may be always demonstrated to be a uterine half, developed from a rudimentary uterine horn, or the unsymmetrical half of a uterus bicornis, either of the right or the left side. It is a cylindrical or fusiform body, that is curved towards the corresponding side, and from the superior portion of which a tube passes to the ovary. The following are the proofs of its resulting from an arrest of development; it presents:

Firstly. A vertical diameter, which generally resembles that of a normal uterus;

Secondly. A diminution of the transverse diameter;

Thirdly. A small (virginal) fundus, with a preponderating thickness of the long and spacious cervix (fœtal state);

Fourthly. The arch in which this uterus is deflected from the meridian is variously curved;

Fifthly. The cervix, as it descends, corresponds more and more to the axis of the body, and its vaginal portion entirely coincides with it. In the virginal uterus the latter is always small, and the vagina narrow;

Sixthly. In the os tincæ the palmæ plicatæ approach closer to the convex margin of the uterus;

Seventhly, The broad ligament on the side of the deficient uterine half is in some cases remarkably large; it at least presents sufficient room for the absent symmetrical half of the uterus.

The Fallopian tube of the defective side shows various relations; if there is no indication of a uterine horn it is almost always absent, and the broad ligament generally forms a slightly fringed prolongation at the point corresponding to the free end of the tube. Occasionally it is even absent when there is a rudimentary uterine horn, and it presents the relations described at p. 206. In rare cases we find a total absence of one half of the uterus, whilst the corresponding tube terminates blindly in the convex margin of the one-horned uterus above its cervix.

The ovary of the defective side is, with rare exceptions, present even when the Fallopian tube is wanting.

We are the more induced to extract the following remarkable case from the essay cited elsewhere (Vol. III.) as an instance of the transition from the uterus bipartitus to the uterus bicornis, as the case of pregnancy in a uterine rudiment (one-half of the uterus bipartitus), which we shall have occasion to quote at a future period, will thus be rendered more intelligible.

The internal sexual organs of a tailor's wife, æt. 34, who died in the lunatic asylum on the 24th of September, 1830, had always menstruated scantily, and bore no children, present the following relations. The uterus has a conical shape, is two inches and three lines in length, presents a curve to the left, has tolerably thick parietes, and is acuminated above; the fimbriated extremity of the Fallopian tube is agglutinated to its ovary. On the right side there is a very large ligamentum latum, within which, at a distance of two inches from the uterus just described, and on a level with its superior portion, there is a body of the size of a hazel-nut, consisting of uterine tissue, and presenting a cavity of the size of a lentil, into which a tube an inch and a half long, and of a sigmoid serpentine form, opens. Posteriorly this uterine rudiment sends off a carneous prolongation, representing the ovarian ligament, anteriorly it gives off a round ligament. On its inner side it is prolonged in the direction of its axis, i. e. obliquely downwards, as a solid band of uterine substance, which impinges upon the convex right margin of the left uterus one inch above its external orifice. Both ovaries are small and contracted, the cervix is small, the vagina narrow, and its arch infundibuliform.

If the two rudiments of the uterus bipartitus are developed uniformly, according to the type of the one-horned uterus, two uterine halves are formed, which unite at one point of their convexity, and thus give rise to the uterus bicornis. The degree of this abnormity varies, and depends chiefly upon the point at which the two halves coalesce. The nearer the latter approaches to the external orifice, the more obtuse will be the angle at which the junction takes place, and consequently the more extensive the fissure. The higher the point of union, the more acute will be the angle, and it may thus become so small that the two halves lie almost parallel to one another, and there is only a slight divergence of the two horns. In the latter case the uterus closely resembles the normal condition; there is always a shallow excavation of the fundus between the projecting horns; the uterine cavity is either simple or divided by a septum of varying length.

The part that unites the two uterine halves always represents the fundus uteri; the higher it is placed, the more this character becomes evident; and when it attains the same level as the uterine horns and surmounts them with its arch, the form of the two-horned uterus disappears. We consequently find, firstly, that the commissure in all cases occupies a horizontal position in the angle in which the two uterine halves meet.

Secondly. That the commissure is always developed in conformity with the fundamental type, viz. that it is a portion of uterine tissue presenting an arch posteriorly, or rather being obtuse-angled and thicker behind.

Thirdly. That when a septum exists it always proceeds from the commissure.

Fourthly. That, however low the commissure be placed, it exerts an evident influence upon the mutual position of the two uterine halves and the internal conformation of their cervices. This consists, in the first instance, in the slight convexity of the posterior, and the slight concavity of the anterior, surface of the uterus bicornis; and in the peculiar relation of the two uterine halves to one another, which is marked by a slight convergence and inclination anteriorly, thus affording the character of a normal uterus. The influence too that is exerted upon the palmæ plicatæ in the uterine halves is singular; the anterior one is placed internally next to the septum, the posterior one lies more externally, and on account of the greater thickness of the fundus uteri—corresponding to the normal character—more towards the posterior surface. The fact of the fundus being wedged in between the cervices in its original form, causes the palma plicata posterior to diverge still more; it induces a slight rotation of the uterine halves anteriorly, which is followed by the above-described form and position of the uterus bicornis.

The septum, which descends from the fundus uteri, may reach down to the os tincæ and divide it, or it does not reach so far, and then the orifice or the cervix is common to both halves, or, lastly, it may be nearly or totally absent, and we then find the cavity of the cervix and the uterus more or less uniform, in proportion as the fundus itself is more or less elevated. If the latter is much depressed and presents no septum, a single cervical channel conducts into two uterine halves that diverge considerably, sometimes so far as to assume a horizontal position.

In rare cases, the two uterine halves do not coalesce, owing to coexisting malformations, such as fissures of the abdominal and pelvic parietes, of internal organs, especially the bladder and the intestine; the uterus thus remains completely divided, and the two halves are separated by the rectum, the colon, the small intestine, or by a rudimentary portion of either, by the mesentery, or the bladder. In the majority of cases, the inferior section of both, or at least of one uterine half, is but very imperfectly developed, and this applies still more to the vagina and to the pudenda.

The lowest degree of uterine fissure is represented by the bilocular uterus. Here the projection of the uterine horns has entirely disappeared; the fundus uteri occupies a position level with the orifices of the Fallopian tubes, and its convexity projects above them. The uterine cavity is divided into two vertical partitions by a central septum; the uterine horns present a normal divergence and the normal length. Yet even here the division of the uterine cavity is perceptible externally; the body of the uterus presenting greater breadth, and generally a shallow fundus, in consequence of which the uterus appears lower, and its dimensions do not, in most cases, exceed those of the normal uterus; the division is also indicated by a shallow furrow running down the posterior surface of the organ.

The division of the uterine cavity by a vertical septum into two loculi extends in rare cases into the external orifice, but more generally is

united to the cavity of the uterus, or the septum does not even suffice to divide the uterine cavity: when this malformation approaches the normal character of the organ, we merely observe a ridge on the fundus and along the posterior wall of the uterus, representing a rudimentary septum. If the septum does not reach the external orifice, its lower free border is always thinner, pointed, and falciform. It probably always descends lower at the posterior than at the anterior surface of the uterus, and this becomes particularly apparent when it merely exists in a rudimentary state.

In the case of the uterus bicornis or bilocularis, the vagina is either single, or may in either be divided in all the forms and degrees described at p. 202. The most perfect fissure seen is that in which the septum of a uterus bicornis or bilocularis descends to the external orifice, divides the latter, and extends to the vagina; the septum may reach as far as the pudenda, and in the virginal state divide the hymen. In this case there is a separate vagina for each half of the uterus.

All these malformations of the uterus occur associated with various irregularities in other organs, as also in individuals that in other respects are well developed. In reference to conception, pregnancy, and parturition, connected with the uterus bicornis, bilocularis, and unicornis, we have to make the following remarks.

Firstly. Numerous well-authenticated observations prove that the anomalous conditions of the uterus which we have discussed, i. e. the uterus bicornis and bilocularis, with or without division of the vagina, and even the uterus unicornis, are capable of being impregnated. In the first we find repeated pregnancy occurring in either half, but there is a preponderance in favor of the right side. There are even cases on record of a twin pregnancy occurring in one, or of concurrent pregnancy in both halves; one fœtus has been found less developed and smaller, and in solitary cases perhaps—though this is to be received with certain doubts—superfœtation had taken place. In the Viennese Museum we have even an example of pregnancy in a rudimentary uterine horn, which terminated fatally in the third month by rupture and sanguineous effusion into the peritoneal cavity. The case was formerly taken for impregnation of the Fallopian tube, until a further examination convinced me to the contrary. It is highly instructive, and doubtless the only case of the kind on record. We shall, therefore, devote a little further attention to it.

The true uterus is a uterus unicornis of the left side with a cervix, in which cicatrices that have been left by former births are visible; the left Fallopian tube issues from its apex, which is turned to the left side. A tolerably thick, roundish, flattened, and hollow cord, consisting of uterine parenchyma, is inserted into the convex right margin of this uterus, and communicates by a millet-sized opening just above the internal os uteri with the cavity of the latter. This cord is above two inches in length, and is dilated externally into a sac of the size of a duck's egg, from the termination of which the right tube with its ovary, and from the lower surface a round ligament proceed. This sac, the rudimental right half of the uterus, contained a female fœtus of the third month enclosed in the normal membranes; it presented a transverse fissure, in the vicinity of the insertion of the umbilical cord, of almost two inches in length. All the membranes were ruptured. The left half of the uterus is twice

as large as it would be in an unimpregnated state, its walls thick, and its innner surface, as well as that of the channel of its parenchymatous process, invested by a deciduous membrane, and the cervix blocked up with a plug of coagulable lymph.

The preparation was taken from a maid-servant twenty-four years of age, who had died suddenly after attacks of pain and spasm in the abdomen on the 24th of March, 1824, and was examined by order of the sanitary board. The body was delicately built and rather emaciated; four pounds of blood, which had been effused in consequence of the rupture of the pregnant rudimentary uterus and the fœtal membranes, were found in the lower part of the abdomen.

The formation of which we are speaking, is the same as the transition form from the uterus bipartitus to the uterus bicornis described at p. 208, with the exception that in this case the parenchymatous cord that passes from the rudimentary to the developed half of the uterus is hollow, and contains a channel which establishes a communication between the two, whereas in the other case the cord is solid. By means of this channel impregnation of the rudimentary uterus was rendered possible; this pregnancy forms a species of transition from uterine to extra-uterine pregnancy, and particularly to pregnancy in the Fallopian tube.

Secondly. In reference to the course of pregnancy and of parturition in uterine formations that are capable of being impregnated, Meckel concludes, from a review of the cases that had been published in his time, that of the comparatively small number of cases of fissured uterus the majority died during or after birth; this ratio is increased by the consideration that in the great majority of these cases the malformations occurred in monstrosities, children, and virgins. Since Meckel, Carus has directed particular attention to the unfortunate issue of these cases. Numerous cases may now be opposed to the ancient and modern observations of the above description, but it appears that the unfavorable ratio pointed out by Meckel still holds good with regard to the uterus bicornis and bilocularis.

Various circumstances conspire to induce great distress or rupture of the womb, even during the early periods of pregnancy (Canestrini, Dionis), to give rise to abortion, flooding, difficult and slow parturition, with consequent exhaustion and predisposition in the uterus to puerperal disease. They become apparent on examining the fissured organ, and we find them to be the following.

a. The absence of the necessary dimensions in the uterine half that undertakes the functions of the entire organ during pregnancy, and the development of which is only provided for by one set of vessels. This applies with additional force to a rudimentary uterine half, as in the case just detailed; in reference to its termination in rupture also, it is allied to extra-uterine pregnancy, and especially to pregnancy in the Fallopian tubes.

b. The obstacle opposed to the uniform development of the impregnated uterine half by the unimpregnated half. It appears that the latter, after the formation of a more or less complete decidua, keeps pace in its development with the impregnated half up to a certain point only, and then remaining stationary, forms an impediment to the uniform growth of that half. This observation is particularly applicable to the bilocular womb,

with a complete septum, as the latter being common to both cavities, remains undeveloped on the side of the unimpregnated portion; it applies less to the true uterus bicornis, the two sides of which are independent of one another.

c. The nearer the uterine malformation approaches the uterus bicornis, the more the two halves of the organ diverge from the axis of the body and the pelvis. In the bilocular uterus, the uterine halves are tolerably parallel to the axis of the body; in the uterus bicornis they form an acute, or even almost a right angle with the latter. The impregnated half of the uterus certainly shows this deviation; but in the uterus bicornis it appears to diminish, whereas in the uterus bilocularis it seems to increase. The axis of the impregnated uterine half is therefore certain to meet with the vaginal axis in an obtuse angle; consequently, during the act of parturition, the direction of the uterine force and of the expulsion of the fœtus will cross the axis of the pelvis, and fall upon the pelvic parietes that lie opposite to the vertex of the pregnant half of the womb. The direction of the impregnated half and of its force, will also be influenced by the unimpregnated half, which during the act of parturition rests upon the pelvis, and especially on the linea innominata of the corresponding side.

d. The fundus uteri and its expulsive power is of particular importance in the act of parturition. The uterus bilocularis has only one half of this part of the organ, and in the uterus bicornis it is totally deficient.

e. Carus considers the impediment to the discharge of the superfluous amount of blood from the uterus to be the cause of the fatal issue which commonly follows birth in the case of fissured uterus. In the normal uterus the return of the blood accumulated in the pregnant womb is effected by means of two sets of vessels; whereas in the fissured uterus, each half of which is supplied by separate vessels, one-half of the venous channels only can carry off the blood. Consequently, although the single uterine horn becomes almost as much developed as the undivided uterus, an unfavorable relation is established, from one set of vessels only being charged with the entire quantity of blood that has to be returned. Besides the above arrests of development, we find, not so much in new-born infants as in the later periods of life, an imperfect development of the uterus occurring in reference to its size, its tissue, and especially to its vascular system; the organ remains small and retains the fœtal or infantine character.

Excess of development, except in the shape of precocity, does not occur; the cases on record of plurality of the uterus are to be viewed as cases of fissure.

§ 2. *Anomalies of Size.*—These consist in irregular enlargement or diminution.

The former either occurs as precocious development, depending upon a congenital vice or accompanying early puberty, or it is the result of morbid increase of size, depending chiefly upon hypertrophy or dilatation.

Hypertrophy either affects the entire uterus uniformly, so that its normal form and the relations of the different parts in point of size and capacity are preserved, or it affects one segment alone, and this partial hypertrophy is particularly remarkable in the cervix.

The hypertrophy varies in degree; it not unfrequently reaches such an extent, that the uterus attains the size of a goose's egg, or of an ordinary fist, and that its parietes present a thickness of from six to nine lines.

In hypertrophy of the cervix, the coexistent malformation is remarkable. The two labia of the os tincæ often enlarge uniformly, so as to form an annular tumor; they more frequently represent two cylindrical swellings, separated by lateral fissures or oblong tumors that are turned up outside; still more frequently we find the anterior lip to be the seat of hypertrophy, and it is often elongated so as to form a simple, cylindrical, or conical teat-like body, or if the cicatrices resulting from previous lacerations prevent the uniform enlargement, it assumes the appearance of an indented or lobulated appendix, and various other strange shapes.

Hypertrophy is caused by previous and repeated pregnancy, by idiopathic or consensual irritation of the uterus, the latter involving the frequent coincidence of hypertrophy of the uterus with diseases of the mammary glands, by prolapsus, and by tedious vaginal, and especially uterine catarrh. Morbid growths, and above all, fibrous tumors developed in the vicinity of the uterine mucous membrane, and projecting into the cavity of the uterus, are another common cause of hypertrophy; on account of the numerous peculiarities presented in these cases, we have hitherto excluded them from our investigation, and shall leave them to be discussed at a future period.

Among the cases of dilatation of the uterine cavity, we have first to notice the one in which it is complicated with hypertrophy caused by fibrous polypi, and which resembles pregnancy, and then those important cases in which the dilatation is the result of an accumulation and retention of the mucous secretion in blennorrhœa, and of tubercular pus in tuberculosis of the uterus. According to the seat of a stricture or of atresia at the internal, or at this and the external orifice of the womb, we find the uterus converted into a simple globular, or into an hourglass-shaped body; dilatation of the proper cavity of the uterus sometimes attains such a degree as to be capable of containing a hen's or even a goose's egg. We shall speak of this under the head of acquired anomalies of the shape, as well as under that of textural changes of the uterus.

Unusual smallness of the uterus occurs in the shape of arrested development, and is the more conspicuous if affecting individuals at or after the age of puberty. The entire uterus, but especially its neck and vaginal portion, is small, dense and hard in structure, and anæmic, its mucous membrane smooth and attenuated, the follicles and folds undeveloped, and the remainder of the sexual apparatus, and particularly the ovaries, in a corresponding state of imperfect development. The affection may also consist in an acquired diminution, reduction, or atrophy of the uterus.

Atrophy generally affects the entire uterus uniformly, though it sometimes predominates in the cervix.

Atrophy of the entire organ is presented in its most remarkable form as marasmus or senile atrophy; sometimes occurring very soon after the climacteric change, and especially in consequence of tedious catarrhs which have ceased with the cessation of the menstrual discharge; sometimes occurring even before this period from debility or exhaustion of the uterus, consequent upon a rapid succession of births, or upon blennor-

rhœa. This condition is generally combined with contraction of the uterine cavity (concentric atrophy), or with partial contractions, atresiæ of the cervix, thinning (atrophy) of the uterine mucous membrane, and accompanied either by increase of density and coriaceous toughness of the uterine tissues, or by another change of peculiar importance, great friability and softness.

A thinning of the uterine walls is also observed to occur in various degrees, as excentric atrophy in the above-named dilatations of the uterus.

Atrophy of the cervix is of great importance on account of its occurrence in young subjects at the age of puberty, and from its probable evil influence upon conception. It has not been as yet clearly demonstrated how this affection is caused. The cervix becomes smaller in consequence of the condensation of its tissues, and at the same time the arch of the vagina is considerably diminished.

Atrophy of the entire cervix is often induced by the tension and traction resulting from the consecutive malpositions of the uterus, which accompany enlargements of the ovaries and large fibrous tumors of the uterus; in the latter case it is not unfrequently associated with hypertrophy of the body of the uterus. It is recognized in the living subject by the elongation of the vagina, and the concurrent disappearance of the cervix, and the conical shape of the vaginal fornix. In rare cases, which we shall have occasion to investigate more closely at a future period, the affection attains such a degree as to induce solutions of continuity in the cervix.

Diminution of the uterine cavity presents the various degrees of stricture, atresia, and obliteration.

Strictures and atresiæ occur generally at one or both orifices of the cervix, but rarely at other points; from here they occasionally extend so as to give rise to a partial or entire obliteration of the uterine cavity. The causes of their origin, both in reference to the physiological and pathological conditions of the organ, have not as yet been fully explained. Our own observations lead us to adopt the view that, in old persons, it is caused by an excessive concentric diminution from marasmus (a tendency in the retrograde organ to complete obliteration); in younger individuals, by chronic, and especially by gonorrhœal catarrh of the uterus.

Contraction of the internal orifice is caused by concentric atrophy, by curvature of the uterus, or sometimes by a fine duplicature of the mucous membrane. Atresia of the passage is either induced by delicate tendinous deposits of epithelium, or by agglutination of the mucous surfaces; the external orifice becomes contracted by inflammatory swelling, hypertrophy, and cancerous degeneration of the cervix; it is closed up by the formation of a whitish layer of epithelium, or by agglutination of the mucous membrane; or, in rare instances, by parenchymatous adhesion subsequent upon injury, inflammation, and ulcerative loss of substance. The two orifices and the entire cervix may also be blocked or closed up by hypertrophied follicles, mucous polypi, cancerous growths, &c.

3. *Anomalies of Form.*—Besides those malformations of the uterus which we have alluded to as resulting from arrest of development, we

have here to mention congenital obliquity of the uterus. Although many doubt its existence, occasional opportunities occur of observing it in a greater or less degree of development. It presents several varieties; the simplest and original form is that in which two lateral halves of the organ are so changed in position that the upper margin does not occupy the horizontal position, and that consequently one horn and its Fallopian tube is placed higher than the other, and the cervix presents a corresponding degree of obliquity. The upper border slants to either side, and its axis forms an angle with the mesial line; a vertical line would divide it in such a manner that the greater part would belong to the elevated side. The inferior half of the uterus is generally bent, or forms an angle at the internal orifice, the higher portion being at the same time much thicker and more massive. The obliquity may confine itself to the body of the uterus, and the latter then forms an angle with the cervix, which either remains perpendicular, or, in rare cases, is even deflected in the opposite direction. A slight degree of this anomaly is presented in a preponderating development of either horn. In many of the last-named cases the uterus assumes the appearance of a retort.

Obliquity is probably of importance in reference to conception, pregnancy, and parturition. It must be distinguished from the mere slanting position of the uterus.

Among the acquired malformations we first notice the oblique position induced by traction exerted upon one side by fibroid tumors, or by an enlarged ovary which has risen into the abdomen. Then those malformations are to be mentioned which the uterus presents in consequence of traction exerted uniformly on both sides, of fibrinous tumors developed within its parietes, and those presented by the vagina in hypertrophy, from cicatrization after rupture or ulcerative loss of tissue; lastly, there are the malformations accompanying dilatation of the uterine cavity, and the development of a uni- or bi-locular capsule. If the cavity of the uterus alone is the seat of an accumulation of mucus, owing to stricture or obstruction of the internal orifice, the former dilates into a globe, which appears seated upon the cervix as upon a stalk; if a similar accumulation takes place in the channel of the cervix from stricture or atresia of the external orifice, the cervix is converted into an ellipsoid capsule, and we then have two cavities, one above the other, separated by an isthmus, and resembling an hourglass. Mayer has termed this malformation of the uterus the uterus bicameratus vetularum.

§ 4. *Deviations of Position.*—As a congenital anomaly of this variety, we have to mention the oblique position of the womb, brought on by shortness of one of the broad ligaments, which it also retains in the impregnated state. Among the acquired deviations of position, we have first to mention anteversion, retroversion, and the less frequent and less important lateral deviations of the uterus. Retroversion is the most frequent, and this may even affect the pregnant uterus.

A condition to which hitherto little attention has been paid, consisting in an angular deflection of the fundus from the cervix uteri, must be carefully distinguished from the two former irregularities. This deflection almost always takes place forwards (Walshe's anteflexion), and very rarely

backwards (retroflexion); the latter never considerable, whereas the former not unfrequently attains such an extent that an angle of 90° and less results. The fundus uteri, in this case, occupies a horizontal position, or may even direct its posterior surface forwards; and occupies the cul-de-sac placed between the uterus and the bladder. This deformity would appear to be an excessive increase of the shallow anterior curvature developed at the period of puberty, and a separation and division of the uterine cavity from the channel of the cervix, consequent upon the preponderating development of the body of the uterus. It is of importance, as it induces similar symptoms as anteversion and retroversion, and also as it probably interferes with conception in the same manner as the congenital obliquities that are complicated with similar lateral deflections, viz. from contraction of the internal orifice.

We have here also to mention prolapsus of the womb, which, as Froriep has satisfactorily demonstrated, may occur spontaneously in consequence of traction exerted upon the womb by the vagina, in the shape of hernia vaginalis posterior. The uterus appears extended; in consequence of the dilatation of the venous plexuses, and the impediment offered to the circulation by pressure, it becomes the seat of hyperæmia; there is increase of size and substance (hypertrophy); and the cervix, at the same time, from being exposed to atmospheric and other influences, is attacked by active congestion, increased secretion, exuberance of epithelium, inflammation, &c. Spontaneous prolapsus occurs in the unimpregnated uterus, and presents various degrees; so-called accidental prolapsus is developed rapidly, it may be brought on immediately or soon after parturition by direct exciting causes, and be complicated with partial, or in rare cases with complete, eversion of the uterus.

Lastly, we find the position of the uterus variously affected by enlargement or dilatation of neighboring organs, by pelvic tumors, malformations of the pelvis, &c.

§ 5. *Deviations of Consistency.*—We shall subsequently advert to numerous deviations in the consistency of the uterine parenchyma, and especially to a diminution of consistency, resulting from various morbid processes; but an increase or diminution in the consistency occurs even without apparent disease of the tissue.

Diminished consistency is not only presented as a relaxation of the uterus accompanied by marasmus, consequent upon the exhaustion induced by parturition, or arising from paralysis of the uterine fibre in puerperal diseases, but it also occurs in a distinct form as pulpiness (marciditas), slight friability or fragility. It very frequently affects the decrepit uterus, involves chiefly the fundus, and appears generally to result from exhausting uterine discharges. The tissue of the affected uterus is of a pale or yellowish red, or sometimes ashy color, it is torn by the slightest effort, its vessels are thickened, rigid, and sometimes ossified. This condition predisposes more particularly to apoplexy of the uterus in the advanced periods of life, and to the consequent conversion of the uterus into a sanguinolent, dark-red, and subsequently, rusty, lee-colored pulp.

This condition is of much greater importance when following parturi-

tion and puerperal morbid processes that have been complicated with phlebitis; we shall have occasion to speak more fully of this *tabes uteri post puerperium* in the sequel.

The uterus presents increased consistency as a consequence of lasting hyperæmia, of hypertrophy or even of atrophy; the entire organ, or certain portions only, as e. g. the cervix being affected. There are various degrees, from coriaceous condensation and toughness to fibroid or cartilaginous induration.

§ 6. *Solutions of Continuity.*—Under this head we include the solitary cases observed by old writers, of rupture of the pregnant womb about the middle of pregnancy, caused by a deficiency in the substance of the uterus bicornis; the more frequent rupture of the uterus at its superior portion, in consequence of excessive labor-pains, caused by insuperable obstacles to birth on the part of the mother or the child, and accompanied by hemorrhage and escape of the contents into the cavity of the abdomen; and the still more common rupture of the uterus at its lower segment during parturition, in consequence of various difficulties.

The latter generally extends from the cervix to the vagina; it may also affect the parietes of neighboring hollow viscera, especially of the bladder; the blood may be effused into the pelvic adipose and cellular tissue, in the vicinity of the bladder and the rectum, and between the broad ligaments; it may pass downwards into the labia, or upwards under the peritoneum into the iliac and lumbar region; or the effusion may be accompanied by rupture of the peritoneum or the bladder, and take place into their cavities. These ruptures affect the entire thickness of the uterine or vaginal parietes, or are limited to an internal layer, or they are lacerations of the vaginal portion of the uterus. They generally have a vertical direction, transverse lacerations being very rare.

In cases of difficult labor the uterus may be subjected to contusions of more or less intensity, which sometimes involve the entire thickness of the organ. The parts adjoining the promontory, or the symphysis pubis and the horizontal rami of the pubes are most liable to suffer. The contusions may affect a circular spot and have a various extent, or they may be chiefly in a transverse direction.

In rare cases the uterus suffers a severe contusion immediately above the vaginal segment, and throughout its circumference, amounting even to laceration; thus the vaginal segment of the uterus may at once, or by a subsequent process of suppuration, become detached, and in the case of eversion of the uterus after parturition, the separation of the entire uterus from the vagina has been observed (Cook).

Finally, we have to allude to ulcerative affections of the uterus caused by or resulting from malignant puerperal disease, and in various other ways.

§ 7. *Diseases of Tissue.*
1. *Hyperæmia—Apoplexy of the Uterus—Anæmia.*—Hyperæmia of the uterus, and especially of its mucous membrane, with effusion of blood in various states of coagulation and discoloration, is often observed in the dead subject as menstrual congestion and hemorrhage. It also occurs

in combination with tumefaction (congestive intumescence) of the uterus and its appendages, with relaxation of its parenchyma and the mucous membrane, dark color, copious sanguineous contents, and hemorrhage into the uterine cavity, representing active or passive congestion or mechanical stasis, consequent upon excessive or anomalous menstrual discharge, or other injurious influences.

Advanced degrees of hyperæmia give rise to uterine apoplexy, i. e. to effusion of blood into the uterine parenchyma, with or without concurrent hemorrhage into the cavity of the organ. It is observed in two distinct forms.

One occurs at the period of decrepitude, and is chiefly caused by the marcidity of the uterine tissue above alluded to, and by the rigidity of its vessels ; its main seat is the fundus uteri, to which it may be entirely limited, or at which, if more extensively diffused, it has taken its origin, and is most prominently developed. The fragile and softened uterine tissue presents a dark red or black discoloration, extending to a greater or less distance from within outwards ; the accumulation of blood may be so considerable as to destroy all traces of structure ; it oozes from the cut or broken surfaces, in greater or smaller quantities, according as it is more or less coagulated. The mucous membrane presents a similar condition, and the uterine cavity very often contains more or less slightly coagulated or fluid blood. The posterior wall of the uterus is but rarely affected, and if so, but to a slight extent.

This form of apoplexy undoubtedly constitutes many of the metrorrhagic cases that occur in advanced age ; the lower degrees may be cured, the tissues subsequently presenting a loose, retiform, contused, and porous appearance, of a rusty or yellowish color.

The second form results from tedious and slow labors ; it occupies the lower segment and the cervix of the uterus. The affected portion appears dark rek, and full of blood ; the part is dilated, relaxed, pendulous and paralyzed, and there may be contusion and laceration also.

Anæmia accompanies an arrest of development, marasmus, induration of the uterus, and general anæmia.

2. *Inflammation.*—Although we shall, as much as possible, distinguish between the mucous membrane and the uterus itself in examining this subject, we must confess that, as may be expected from the close anatomical connection of the two, the diseases which we shall have to consider, very readily pass from the one to the other. Yet we must also affirm that generally the lining membrane of the uterus is affected primarily, and that this is scarcely ever the case with the uterine tissue, as far as can be demonstrated by the pathological anatomist, with the exception of the reaction following traumatic influences, especially of the vaginal portion. We shall not at this place devote any attention to peritoneal inflammation, but discuss the inflammatory affections of the unimpregnated uterus, and the participation of the uterine parenchyma in them. The uterine inflammations occurring after childbirth, with their sequelæ, we shall consider in a separate appendix on puerperal diseases of the uterus.

a. *Catarrhal inflammation* (*endometritis catarrhalis*).—This is an acute affection ; it occurs in combination with inflammation of the adja-

cent uterine tissues, extending to a greater or less depth and of various intensity, and even complicated with peritonitis; it is frequently met with in the sick-room, but rarely in the dead-house: it is here only occasionally observed in a protracted blennorrhoic stage.

The uterine mucous membrane is much more commonly discovered in a state of chronic catarrh and inveterate blennorrhœa, which is either the residue of acute catarrh, or the result of a similar affection of the vagina; it may occur as a sequela of parturition, or as a complication of those morbid growths that bear a near relation to the uterine mucous membrane. The mucous membrane offers a pallid appearance, or there is evidence of previous stasis and inflammation, and it then presents, with the adjoining uterine tissue, a brownish-red or slaty color; the membrane is tumefied, relaxed, plicated, and secretes a grayish-white viscid mucus, which during temporary exacerbations, or an enduring state of more intense inflammation, appears streaked with blood, creamy, yellow, and puriform.

Here, too, we find hypertrophy of the mucous membrane resulting from chronic catarrh, in the shape of mucous or cellular polypi. They consist of club-headed elongations of the mucous membrane, in which we find a group of closed follicles, or a lobulated tissue containing a gelatinous mucus, which is discharged from time to time in consequence of a dehiscence of the follicles. These excrescences occur chiefly at the fundus uteri, in the neighborhood of the insertions of the Fallopian tubes and in the channel of the cervix—a point at which, in the normal condition, large follicles (ovuli Nabothi) are found, which occasionally undergo considerable enlargement.

We find that the uterine parenchyma becomes more or less hypertrophied during catarrh, in the same manner as other muscular layers which are subjacent to mucous membranes.

Inveterate uterine catarrhs not unfrequently give rise to the above-mentioned strictures and atresiæ; and if the blennorrhœa persists, the dilatations of the uterine and cervical cavities previously discussed, result. During the progress of dilatation occurring under these circumstances, the same changes that we have already repeatedly met with under similar circumstances, in dropsy of mucous cavities and canals, are sometimes found to occur in the uterus. As a dilatation from the accumulated secretion increases, the uterine mucous membrane is converted into a thin serous membrane, which secretes a colorless, serous, albuminous fluid, resembling synovia. The uterus appears in the shape of a round, slightly-thickened, hydropic capsule, of the size of a hen's or duck's egg or a fist. This condition is the only one that really deserves the name of hydrometra, of which several remarkable instances are related, especially by older writers. The contained fluid may always, or for a long time, remain such as above described; but it generally undergoes some alterations from the admixture of various products of slight inflammatory attacks, and especially of hemorrhagic exudations of the uterine lining, which give it a chocolate-colored, rusty, or black tinge.

Occasionally temporary discharges of these fluids occur by the vagina during life, after which fresh accumulations take place. They are to be distinguished from similar discharges from the hydropic Fallopian tube.

Uterine catarrh generally suffices to produce sterility; but it often ex-

tends to the Fallopian tubes, and there also gives rise to changes that are of extreme importance in this respect.

b. Exudative processes (endometritis exudativa).—Croupy or plastic fibrinous exudation, whether or not accompanied by a similar process in the vaginal or Fallopian mucous membrane, very rarely occurs on the inner surface of the uterus, except after confinement. It is, at all times, rather a secondary than a primary process. Exudative affections of the uterus and their varieties, occurring after parturition in the shape of puerperal diseases, are all the more frequent and the more numerous.

3. *Ulcerative processes.*—In treating of catarrh of the vagina, we have alluded to excoriation, superficial and follicular ulceration of the vaginal portion of the uterus. The specific character of the catarrh and the follicular ulceration, as well as neglect of proper attention and treatment, cause the resulting ulcers to present a more or less remarkable appearance in reference to the shape of their edges, the reaction set up, the product and the change of texture, as well as in regard to the consequent fusion of the diseased tissue, and to the concurrent tendency of disorganization beyond the ulcer. It is stated that, in reference to the first of these considerations, we may distinguish the simple (catarrhal), the herpetic, scabious, and scrofulous ulcer of the cervix; as regards the local process, there may be a fungous, lardaceous, or callous ulcer, &c. We also find primary and secondary syphilitic ulcers, cancerous ulcers that have resulted from the fusion of cancerous morbid growths, the so-called phagedenic ulcer of the os tincæ (Clarke's corroding ulcer). The latter may be compared to the phagedenic (cancerous) sore of the skin; without having a morbid growth for its base, it gradually destroys the cervix, and even the greater part of the uterus, and may extend to the rectum and the bladder. It is an irregular, sinuous, jagged ulcer, the tissues at the margin and the base of which are thickened or hypertrophied, in consequence of a sluggish inflammatory process; the base presents a greenish and brownish-green discoloration, with a slight glutinous and purulent, or a more copious watery, secretion: there are no granulations, but we find a gelatinous exudation, and according to the state of the immediate reaction, the tissues are converted into the above-mentioned products of the ulcerating surface.

Lastly, we find the uterus liable at different parts, and in a varying extent, to acute or chronic ulcerative disorganization, as a consequence of puerperal affections; this subject will be examined in the appendix.

4. *Morbid growths. a. Cysts.*—Cysts are very rarely formed in the uterus; we have not met with a single example in Vienna, and I myself have only inspected one case of uterine acephalocysts. It is necessary to distinguish the very much hypertrophied follicles that may occur in the uterine cervix, from newly-formed cysts.

b. Fibroid tumors.—Anomalous fibrous tissue is the most frequent of all new formations occurring in the uterus, in the shape of fibroid or fibrous tumors (tumor fibrosus, desmoides, formerly called sarcoma; when ossified, osteosteatoma of the uterus; scirrhus; W. Hunter's carneous tubercles, &c.) These fibroid growths of the uterus not only present all the essential characters peculiar to them elsewhere in a remarkable degree, but they also offer numerous important and accidental modifica-

tions, some of which exert a considerable influence upon the uterus; it therefore becomes necessary to devote a more extended consideration to them, in addition to the general outline which we have already given. The uterus, as well as the adjoining tissues, are particularly liable to be the seat of fibroid growths. They not only present all the varieties and degrees as regards size and volume, shape, number, and metamorphosis, in so characteristic a form, that we have thought it right to take them as the specimen and groundwork of general disquisitions on the subject, but they also offer the most various modifications in reference to their seat, and consequent reflex influence upon the womb.

We also find that the changes in position of the uterus, the deviations of its shape, and of the direction and form of its cavity, of its size in reference to the coexistent hypertrophy and atrophy of the organ, and the relations of the uterine mucous membrane, &c., are very remarkable.

The three varieties distinguishable in the fibroid tumor, according to its internal structure, are all found in the uterus. The variety in which a concentric disposition of the fibres is displayed, is here also distinguished by its density, hardness, poverty of vessels, smallness, and spherical shape.

The second variety, in which the fibres appear irregularly disposed, and issue from numerous centres or nuclei, present a rounded form, and an uneven, nodulated surface, which indicates the aggregation of the fibrous centres in reference to density and consistency, vascularity and volume, they offer the extensive modifications already spoken of; they may, on the one hand, be very dense and hard, and unvascular; on the other, in consequence of an accumulation of cellular tissue in the interstices of the fibrous layers, they may be more or less vascular and succulent, or soft and elastic, soft and doughy, flabby, &c., sometimes resembling a soft mammary gland, sometimes a coarse-grained salivary gland. Those fibroid tumors, the interstices of which are dilated into cells or cavities, containing a serous fluid from excessive exhalation of the intervening cellular tissue, are of extreme importance. They present fluctuation, and may, on account of the deceptive appearances accompanying fibroid tumors, be easily mistaken for ovarian dropsy, hydrometra, acephalocyst of the uterus, or pregnancy.

The fibrous polypus of the uterus, the third variety of fibroid tumors, takes it origin by a single or divided trunk in the interstitial cellular tissue of the uterine parenchyma; the former expands into striated fasciculi, which are bound together by softer vascular and cellular interstitial substance, and the entire mass presents a distinctly lobulated structure, which is more or less visible externally. The polypus grows into the cavity of the uterus, with which it is in the closest anatomical connection, and upon the functions of which it exerts a considerable influence. It enlarges chiefly in one direction, and has a cylindrical, fusiform, clubbed, pyriform shape, and is more or less flattened; it is provided with numerous and very large vessels, is apt to swell, and in consequence of excessive congestion and rupture of the vessels, we often meet with extravasation within its tissues.

The anatomical relation of fibroid tumors to the uterine parenchyma is very intimate in the third variety, less so in the second, and least of all

in the first, in which the tumors adhere to the uterine parietes by a thin layer of whitish or reddish, more or less vascular, cellular tissue, so that they may be detached without difficulty.

The form of the fibroid tumors of the first and second variety, we have already described as being generally round ; in the second variety some alterations may occur, though the globular form still predominates. The peculiarities of shape of the fibrous polypus, or third variety have already been stated. The greatest variety occurs in reference to size. Fibroid tumors are found from the size of a hemp-seed to that of a man's head.

The fibroid tumors belonging to the second variety attain the largest size, especially when of loose texture, and rich in interstitial cellular tissue ; the fibrous polypi also reach a considerable magnitude, but the fibroid tumors of the first variety are the smallest. They are all generally developed slowly, though the second and third variety are occasionally developed with extraordinary rapidity ; they are also liable to a temporary increase of size or tumefaction proportionate to their vascularity.

As to their number, we sometimes only find a single, sometimes several or many fibroid tumors in the same uterus. We then observe tumors of the most different sizes coexisting. This applies chiefly to the first two varieties ; the fibrous polypus is often solitary, but it also occurs in company with the others.

The uterine parietes are the seat of the fibroid tumors, but not only do they occur much more frequently in the body than in the cervix, but in the former they chiefly affect the upper portion or fundus. They very rarely occur at the inner orifice, and if possible, still less frequently in the vaginal portion. This is the case with all fibroid tumors, a fact that forms an interesting contradistinction to the relation which cancerous disease bears to the inferior segment of the uterus. Fibrous polypus, more especially, is apt to commence at the fundus, and at the orifices of the Fallopian tubes. The fibroid tumor is inserted into, and takes its origin from, the middle layers of the uterine substance, or it appears to be more connected with the external layer, or even to lie under the peritoneum, or again, it lies nearer the inner surface, or immediately under the mucous membrane. The first two varieties are developed in the most various layers, though generally in the external ones ; the third forms upon the internal layer exclusively. The former also very frequently present other curious relations, whether they have been developed in the vicinity of the peritoneum, or of the mucous membrane of the uterus. In the first instance the tumor, as it enlarges, gradually becomes detached from the uterus, dragging the peritoneum after it, and thus at last becomes pediculated or pendulous, by a peritoneal cord of various length. In the second instance it pushes the mucous membrane before it, as it enlarges, and at last hangs into the uterus by a mucous pedicle, thus resembling the true fibrous polypus, from which it may be distinguished by its relation to the uterine parenchyma, and by its internal structure.

We must here advert to a circumstance that is not of very rare occurrence, viz. we sometimes find a fibroid tumor in the pelvic cavity, and generally in Douglas's space, without any further connection with the uterus, except by means of cellular cords, or laminæ of new formation

(false membranes), which pass from the tumor to the uterus and its appendages, to the pelvic walls, the rectum, &c. The question presents itself, which is the original point of development of such fibroid tumors. They are generally tumors which have originally been developed under the uterine peritoneum, and, after having become entangled in a network of pseudo-membranous formations, resulting from the peritonitis they have excited, are gradually detached from the uterus. Occasionally, however, they may have been developed within the false membranes themselves, which is the more probable, if we consider that the new tissue as it proceeds from the uterine peritoneum, participates in the character of the subserous uterine cellular tissue. Hence it is extremely likely that we really see very small fibroid tumors occasionally developed in this new tissue.

To these fibroid tumors, the loose fibrous concretions which are sometimes found in the pelvic cavity are allied ; they must be considered as fibroid tumors of the uterus, which have become detached in consequence of atrophy of the peduncle.

Metamorphoses and diseases of the uterine fibroid tumors. Spontaneous cure.—We have already spoken of ossification, congestion, inflammation, suppuration, and solution of fibroid tumors generally ; and those remarks apply with the more force to uterine fibroid tumors, as we assumed the latter as the foundation upon which we based our observations. Ossification occurs very frequently, congestion less so, and inflammation and its terminations rarely. A spontaneous cure, under which head we must also class ossification, on account of the destruction of vitality in the tumor, occurs in a few rare cases, by a detachment of the fibroid tumor as it projects into the uterus, or is suspended in it by a mucous pedicle. It is effected in the following manner : the mucous membrane of the uterus covering the apex of the tumor is in a condition of permanent irritation and congestion ; this is at last converted into inflammation, and terminates in suppuration and gangrene. The tumor is thus partially exposed towards the uterine cavity, and the destructive process gradually involving its entire cellular investment, it becomes detached, and passes through the opening in the uterine mucous membrane into the uterine cavity. Ancient and modern cases are on record, in which fibroid tumors of various sizes and ossified tumors were thus discharged. The powers of nature rarely suffice if the tumors are of considerable size, as the extensive suppuration necessary for that purpose is likely to prove fatal, both by exhaustion and by the extension of inflammation to neighboring organs. It would appear that the fibrous polypus is occasionally, though very rarely, discharged in a similar manner, in consequence of suppuration occurring at its roots and in the surrounding tissues.

The changes in the uterus, consequent upon the presence of one or of several large fibroid tumors, are numerous and important, by reason of the diagnostic characters they afford.

In the first instance, the volume of the uterus increases in proportion to the number and size of the tumors ; the fibrous polypus causes an enlargement of the uterine cavity, corresponding to the size of the polypus. The increase in the substance, the hypertrophy of the uterus, which the fibroid growths generally induce, and, on the other hand, the atrophy of

the organ, are of greater interest. The hypertrophy appears as a development of the uterine tissue, resembling that occurring in pregnancy; it varies in degree. In reference to the latter subject, the question presents itself by what means the different degrees of hypertrophy are determined, and on account of the occasional passive condition and the occasional atrophy of the uterus, it is necessary still further to generalize, and to ask how it happens that under some circumstances the uterus becomes hypertrophied, in others remains unchanged, and in others again becomes atrophic? In answer, we offer the following remarks:

a. The nearer the fibroid growths approach to the uterine mucous membrane, and project into the cavity of the uterus, and thus maintain the mucous membrane in a state of irritation and inflammation, the more palpable is the hypertrophy of the uterus. It is most fully developed, so as to resemble pregnancy, in the case of the fibrous polypus.

β. Hypertrophy of the uterus appears to be encouraged by a vascular state of the tumor, by the latter being less dense and capable of rapid growth.

γ. As also by the development of the tumor, during or shortly after the period of conceptivity.

δ. The size of the tumor exerts no direct influence upon the origin of hypertrophy or atrophy.

ε. Atrophy undoubtedly results very rarely from fibroid tumors, nor must we forget that they are not unfrequently developed in the uterus during the period of decrepitude, and that they increase very slowly on account of the universal state of marasmus. In this case the atrophy of the uterus is entirely independent of and antecedent to the fibroid tumors.

The atrophy of the cervix accompanying large fibroid growths is, as we shall have occasion to explain more fully, the result of mechanical traction.

An important change takes place in the position of the uterus, which may be discovered by external examination. Not only does a large fibroid tumor that occupies the external layer of the uterine tissue, push the organ to the opposite side of the pelvis, but we also notice a remarkable ascent of the organ. The more numerous and the larger the tumors are, and the more they consequently rise out of the pelvis, as it interferes with their growth, the more they drag the uterus after them; its vertical position being also changed in proportion as the fibroid tumors preponderate on one side or the other. This traction necessarily causes an elevation and elongation of the cervix.

The external surface of the uterus is, as may be easily understood, variously disfigured by the projecting tumors. In the same manner the cavity of the uterus, in addition to a corresponding elongation, undergoes various alterations in form and direction, proportionate to the number and size of the tumors which project internally. In reference to the displacement, we sometimes find the entire cavity forced out of the mesial line, at others it presents more or less angular deflections. The most important disfiguration is effected by the upward traction exerted by numerous and large fibroids. The uterus, and particularly the cervix, is elongated to a degree proportioned to the degree of traction, it be-

comes thinner, and the attenuation may, in rare cases, even cause a gradual solution of continuity, one portion remaining attached to the vagina, another following the upward direction of the uterus, and the connection being maintained by a mere band of cellulo-fibrous tissue. The channel of the cervix at the same time contracts, and may even become entirely obliterated. The vaginal portion gradually disappears, the vagina itself becomes smooth and narrower in consequence of the elongation, and its arch is converted into a funnel, the apex of which terminates in the os uteri.

If one or more fibroid growths occupy a lateral portion of the uterine parietes, and especially if they be seated in the vicinity of the Fallopian tubes, the external form of the uterus may be rendered oblique ; if under these circumstances the tumors enlarge, and consequently exert lateral traction, this may be recognized by the elevation of the corresponding side of the os tincæ, and the increased distension of the vagina.

Fibrous polypus gives rise to a dilatation of the uterine cavity, and of the cervix, corresponding to the size of the morbid growth ; if the enlargement proceeds to a greater extent, the external orifice becomes dilated, and the tumor projects through it into the vagina. Large and heavy morbid masses of this description frequently cause a slight descent of that portion of the uterus into which they are inserted, by the traction they exert, and sometimes even induce complete inversion of the womb.

The mucous membrane of the uterus is the more liable to catarrh and blennorrhœa, the nearer the fibroid tumor approaches to it ; sometimes it becomes hyperæmic, and blood is effused upon it. This is particularly the case with the fibrous polypus, which is not only accompanied by the ordinary hemorrhage from the capillaries of the mucous membrane, but also from larger vessels of the uterus, or sinuses of the morbid growth that have given way to excessive traction.

Fibroid tumors of the uterus scarcely ever occur before the twentieth year ; a fact which is established by the numerous observations made by ourselves and other anatomists. They are even unusual up to the thirtieth, and present themselves most frequently shortly after the fortieth year. Without entering into an analysis of the almost innumerable cases that we have ourselves met with, we may mention the results of Bayle's calculations as to the frequency of their occurrence ; he states that of one hundred females that die after the thirty-fifth year of life, twenty at least are affected with fibroid tumors.

They are found in complication with the most various morbid growths of the uterus and its appendages ; but especially with cancer of the cervix, with the corroding ulcer of the os tincæ, with ovarian dropsy, &c., still on the whole the complication with cancer is not frequent.

The powers of conception are commonly not impaired by the presence of fibroid tumors, and if these are small, and do not occupy an unusual position, they have not necessarily an injurious influence upon pregnancy and parturition, though they frequently cause abortion and hemorrhage after birth. Parturition may be very much impeded if they occupy the cervix uteri. It is important to know that these tumors become more vascular, succulent, and softened during pregnancy, and assume a bluish-

red color, so that their original appearance is entirely changed. As the uterus returns to its original shape, the morbid growth also resumes its ordinary characters. Pregnancy is even said to give rise to hemorrhage and inflammation in the tissue of the fibroid tumor.

An unusual though very important occurrence, brought on by the excessive expansion and traction exerted by large fibroid tumors, is the laceration of the vessels, and especially of the veins. We have once observed the rupture of a vesical vein (with that of the mucous membrane) followed by hemorrhage into the bladder, and in another case the rupture of the subperitoneal vein of a fibroid tumor, with hemorrhage into the abdominal cavity, as described by other writers.

Ligature of the fibrous polypus is sometimes followed by uterine phlebitis.

5. *Osteoid growths.*—We have not met with osseous formations in the uterus, except in the shape of ossification of the fibroid tumors.

6. *Tubercle.*—Tubercle occurs primarily as tubercle of the uterine mucous membrane; the uterine parenchyma is like the submucous muscular layers, only attacked secondarily by tubercle.

It generally occurs in the uterine mucous membrane in the shape of an infiltrated mass, which fuses into and attacks the uterine parenchyma to a greater or less extent. The mucous membrane appears converted into a fissured, cheesy, purulent mass of tubercle. The cavity of the uterus contains tubercular pus, which may be retained in consequence of closure of the orifice, and accumulate so as to cause a globular distension of the organ. The disease is very rarely observed in its early stage, in the shape of scattered or grouped gray miliary tubercle of the mucous membrane and the adjoining submucous tissue.

Uterine tubercle is formed during childhood, in the period of puberty, and during the prime and even, though rarely, during the decline of life. It is most frequently complicated with tubercle of the Fallopian mucous membrane, and with the latter may constitute the primary tubercular affection. It is also found complicated with abdominal tubercle, and especially of the abdominal lymphatic glands, and of the peritoneum; and may serve as a point of discharge for the latter. A translation of the tubercular disease to the urinary passages is very rarely observed.

It is curious that the tubercular deposit stops short at the cervix, and very rarely passes even beyond the internal orifice of the womb; the vaginal portion is never affected with tubercular disease. This is extremely remarkable on account of the marked contrast offered by carcinoma, both in reference to its primary and secondary development.

7. *Carcinoma.*—Next in frequency to fibroid growths is the occurrence of cancer. It always attacks the cervix in the first instance, and especially that portion which projects into the vagina; the primary occurrence of carcinoma at the fundus uteri is so extremely rare, that the above observation may be considered as an absolute rule. It is contrasted in this respect with fibroid and tubercular disease of the uterus, and it presents a similar contrast in reference to its extension and ulcerative destruction.

According to our observations, fibrous cancer very rarely affects the uterus; the most common form is the medullary, either by itself or complicated with the former.

Opportunities very rarely present themselves of investigating the early stages of cancer in the dead subject; according to a few observations, fibrous carcinoma, when closely examined, appears to consist of dense whitish, retiform fibres, differing from the normal texture of the vaginal portion of the uterus in which they are found, and in their very minute meshes a pale reddish-yellow or grayish translucent substance is deposited. This morbid growth is inserted into the uterine tissue without well-marked boundaries; it occupies a various extent, and from accumulating at certain points, gives rise to the irregular nodulated character and the well-known induration which accompanies the enlargement of the cervix.

Medullary cancer in the first instance appears as an infiltration of a white lardaceo-cartilaginous or lax encephaloid matter, in which the uterine fibre disappears; as the deposit increases the vaginal portion assumes an uneven nodulated character, and appears hard and elastic to the touch. Cancer of the uterus very rarely presents itself in the shape of isolated globular growths.

As the cancerous degeneration proceeds, and especially on the commencement of the stage of metamorphosis, with its consequent new formations, particularly if they belong to the medullary variety, the lower segment of the uterus undergoes a very considerable and rapid enlargement. At last we find a callous, loose, spongy ulcer developed in the usual manner, which discharges a very fetid, greenish-brown, sanious and sanguineous fluid, and as it extends, generally causes a progressive infiltration of cancerous matter. The tumefaction of the cervix and the fungoid excrescences not unfrequently close up the orifice, and the consequent enlargement of the womb will be the larger, the more copious the secretion of the mucus.

Cancerous degeneration of the uterus is generally confined, in a very remarkable and distinct manner, to the vaginal portion; still there are frequent exceptions to this rule, as the disorganization is sometimes found to extend with great rapidity to the body, and even to the fundus of the uterus; this is particularly the case if the os tincæ has already been attacked by ulceration. The disease may spread downwards and involve the vagina, thus establishing vaginal cancer. It may extend in other directions, and thus give rise to cancerous degeneration of the rectum, the bladder, the pelvic, cellular, and adipose tissue, and the periosteum; the uterus thus becomes fixed in the pelvis, and at last we find the peritoneum attacked, cancerous growths being formed upon it and its tissue, or perforating it, especially in the shape of medullary masses.

Cancerous ulceration spreads in the same direction; in rare cases we find the greater part of the uterus, and even its fundus, destroyed. The destructive process, when attacking the vagina, sometimes predominates on the anterior, sometimes on the posterior surface; sometimes it attacks both equally, and may extend downwards almost to the external orifice. It also involves the degenerated parietes of the rectum and of the bladder, and generally produces extensive communications between their cavities and the original cancerous sinus (ulcerous cloacæ). It finally extends in the shape of sinuous passages, through the remainder of the

cancerous mass that fills the pelvic cavity, to the pelvic bones. In this manner a large cavity with fungoid parietes is at last established, which occupies the greater part of the uterus and the vagina, and opens into the cavities of the rectum and the bladder; above it is closed in by the fundus uteri and the adherent rectum and cervix vaginæ, as also by the cæcum and small intestine, which are agglutinated to these parts, and at last it penetrates into the cavity of the peritoneum or the intestines. The contents of the cavity are cancerous ichor mixed up with fæcal matter, urine, and portions of gangrenous tissue.

The temporary and tumultuous periods of development presented by the peritoneal inflammations of the pelvic and hypogastric regions, which accompany and characterize the metamorphic and ulcerative stages, and which not unfrequently extend from the original layer over the entire peritoneum, are important occurrences in the progress of cancerous disease.

Uterine cancer is, in most cases, a primary disease, and generally remains for a long time, if not throughout the sole carcinomatous affection of the organism. However, it is sometimes developed concurrently with or consecutively to mammary and ovarian cancer; or it is accompanied by degenerations of the adjoining tissues above mentioned, and of the lymphatic glands, which must be explained upon the theory of propagation by contact; or again, it is associated with cancer of the peritoneum, of the liver, the stomach, and the breasts, with cancer of the bones, with mollities ossium, ovarian cancer, and universal cancerous deposit, as a consequence of the resulting cancerous dyscrasia.

Uterine cancer most frequently occurs between the fortieth and fiftieth year; still there are many cases on record in which it appeared between the thirtieth and fortieth year, and even earlier.

The cases of spontaneous recovery from uterine cancer are of extreme rarity, but they do occur; the carcinoma and the cancerous ulceration are then limited to the cervix, the internal orifice forming the boundary; the loss of substance heals with a funnel-shaped cicatrix.

We append to the above remarks on uterine cancer a brief account of the so-called—

8. *Cauliflower excrescence of the os uteri*, which we are inclined to consider as of a cancerous nature. It is of very rare occurrence, and we have only once observed it in the living subject, in a form similar to that described and delineated by Clarke. It presented the appearance of a confervoid growth, consisting of lenticular, pale red, transparent, and tolerably hard corpuscles, strung together like the beads of a rosary, projecting on the orifice of the uterus into the vagina, and bleeding on the slightest touch. It was developed and grew from an evidently cancerous base of the medullary variety.

Clarke states, that it also occurs without this complication, and that it is curable; the unfrequency of its occurrence and the circumstance that after death it collapses, and merely appears like a slight accumulation of delicate cellular tissue, render it difficult to decide the question as to its cancerous nature; this, however, is the view we are inclined to adopt.

The chief and very dangerous symptom which the affection presents

are frequent exhausting hemorrhages, which are brought on by the most trivial causes. It is said to occur at any period of life after the twentieth year, but very rarely before that.

SECT. II.—DISEASES OF THE UTERUS AFTER PARTURITION.

Under this head we include diseases to which the uterus is liable in consequence of the puerperal state, which are essentially (in reference to causation) connected with the latter, and especially with the concurrent detachment of the membranes and the placenta from the inner surface of the uterus, and which, for that reason, must be termed puerperal affections. We pass over the subjects which have already been discussed, and enter at once upon the consideration of these diseases in the following (natural) sequence.

§ 1. *On defective and irregular Contraction and Involution of the Uterus after Childbirth.*—We occasionally find that the uterus presents a condition of universal flabbiness or collapse of its parietes, accompanied by a trifling reduction of size, which must be considered as paralysis from exhaustion, and which results from tedious or instrumental labor, or from parturition, the first stages of which had been much accelerated. In other cases, and they are of frequent occurrence, we find the fundus and the neighboring parts of the corpus uteri to be the seat of excessive contraction and energy, whilst the inferior segment is in a contrasting state of atony and collapse; there are other cases again in which excessive contraction prevails at the middle of the uterus forming a zone round it, or at smaller and less defined portions. These occurrences may be brought about by the most various impediments to parturition, by pressure, contusion of the uterus, apoplexy of the womb (vide page 217), by original irregular innervation of the uterus, &c. As may be supposed, they give rise in the first instance to hemorrhage, and in consequence of this and of the general debility, they impede the further involution of the uterus, and thus protract the disposition to puerperal affections. We must here mention a very singular circumstance, which may, on account of the consequent danger, become important, and may even be misunderstood in post-mortem examinations; it is paralysis of the placental portion of the uterus, occurring at the same time that the surrounding parts go through the ordinary processes of reduction. It induces a very peculiar appearance. The part which gave attachment to the placenta is forced into the cavity of the uterus by the contraction of the surrounding tissue, so as to project in the shape of a conical tumor, and a slight indentation is noticed at the corresponding point of the external uterine surface. The close resemblance of the paralyzed segment of the uterus to a fibrous polypus, may easily induce a mistake in the diagnosis, and nothing but a minute examination of the tissue can solve the question. The affection always causes hemorrhage, which lasts for several weeks after childbirth, and proves fatal by the consequent exhaustion. We have met with it twice, once after abortion, and once after parturition at the full period.[1]

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[1] Dr. Betschler, during his visit to Vienna in 1840, communicated a similar case to me as having occurred at Breslau; and there can be little doubt that Dr. Burkhardt (vide Berliner Centralzeitung, x, 19) speaks of this condition, under the title of acute fungus hæmatodes uteri, as of a new and hitherto unknown cause of flooding after childbirth.

Lastly, we observe that the contraction and involution of the uterus is more or less permanently impaired by all the different puerperal inflammatory processes.

§ 2. *Puerperal Inflammations.*—Puerperal inflammations generally, are in most cases of a very complicated nature, and it is of extreme scientific and practical importance that we should obtain a comprehensive sketch of their anatomical bearings, as well as an analysis and correct interpretation of the constituent phenomena. If we consider puerperal inflammation of the uterus by itself, we find that it always appears in the shape of an exudative process, affecting the raw exposed surface of the uterus to which the placenta had been attached; in reference to its original seat, it must therefore always be considered as endometritis. We shall first have to examine into the characters of this affection, and then proceed to investigate other important puerperal diseases; after which, we shall give a summary and an analysis of changes taking place in organs and tissues that do not belong to the original seat of disease, and conclude with a consideration of the issues and consequences of primary and secondary puerperal affections.

1. *Puerperal endometritis.*—This affection, as has already been observed, is invariably an exudative process; but it offers the greatest variety, both in reference to the plasticity of its product and to the condition of the diseased tissue, either in individual cases or in entire epidemics. The series is almost endless, but we may consider genuine uterine croup on the one hand, and the so-called genuine putrescence of the uterus on the other, as its extremes; the very fact of this great multiplicity of forms obliges us to limit our descriptions to the most prominent ones.

In certain cases we find the internal surface of the uterus lined by a yellowish or greenish dense exudation, of greater or less thickness and extent, either in small patches or investing the entire uterus, and either firmly or loosely agglutinated, and occasionally partially or entirely detached from the subjacent tissue, so as to appear corrugated or plicated. The uterine mucous membrane under the lymphatic coating is found reddened, tumefied, and slightly softened; the free parts are discolored, and invested with a dirty reddish or brownish secretion, and with remnants of the deciduous membrane. The exudation generally interpenetrates largely the exposed raw tissue of the placental portion of the uterus, and causes it to assume a peculiar ulcerated appearance. This is uterine croup.

In other cases the exuded matter is a gelatinous, purulent, dirty yellow, loose and easily detached layer, beneath which the internal stratum of uterine tissue appears spongy, infiltrated, soft, and may be easily detached in the shape of a dirty yellowish-red, or partly greenish and brownish pulp. The internal surface of the uterus presents, in addition to the lymphatic exudation, a glutinous secretion of a similar tinge.

Again, the internal surface of the uterus may not present a trace of coagulable lymph, but be invested by a purulent sanious and very discolored exudation, beneath which we find the uterine mucous membrane infiltrated, in more or less extensive or circumscribed patches, with a

similar product; and it may either be easily removed in the shape of a thin and much-discolored pulp, or it has already become detached, and is mixed up with the contents of the uterus in the shape of friable discolored flocculi. In the place of the destroyed tissues, we occasionally discover the products of a reactive process, in the shape of a more or less consistent sanio-purulent secondary exudation.

Again, the internal layer of uterine tissue may be covered with a thin opaque or more dense, pale green or brownish, or dark chocolate or coffee-colored product, beneath which it is converted, to a greater or less depth, into a loose, infiltrated, fetid pulp, of a similar tint. This condition, which differs from ordinary sphacelus, has been termed putrescence of the uterus.

All these characters point to an exudative process, the peculiar nature of which is fixed by the form of its product, and the condition of the substratum, and especially by the state of fusion of the latter. There are numerous states of transition between the forms described, and they not unfrequently become complicated with one another in such a manner that a process of a malignant nature follows one that is accompanied by a secretion of plastic lymph. As primary exudative processes, they are, if possible, to be distinguished from similar secondary processes which may occur in the course of the disease in consequence of a secondary affection of the blood, resulting from inflammation of the veins or lymphatic vessels.

As supplementary to the above, we have to examine those anomalies presented by the uterus, which are either direct reflexes of the processes in question, or which occur as accidental complications.

To the former appertain paralysis of the uterine fibres and impeded involution of the uterus in various degrees. According as the puerperal affection attacks the uterus, sooner or later after parturition or with more or less intensity, the womb is found of greater or less size, more or less relaxed, collapsed, softened; and certain portions that contain a large amount of cellular tissue, such as the lateral edges and the cervix, are infiltrated with a pale yellow, sero-gelatinous, or sero-purulent fluid. The external surface of the fundus and body of the uterus not unfrequently exhibit numerous shallow depressions, that are caused by the pressure of adjoining tympanitic coils of intestine.

Among the accidental complications we reckon sanguineous engorgement (apoplexy) of the neck of the uterus, the superficial or profound lacerations and contusions which occur at this point, and in the vaginal segment; the lacerations being invested with exudation of a more or less plastic character, whereas the contused parts not unfrequently appear in a state of gangrenous solution. We have to mention the sloughs of greater or less dimensions, which occur chiefly at the neck and vaginal portion of the uterus, and also in the vagina and the external genitals, in company with malignant exudative processes. These processes lead to ulceration and gangrenous fusion of the tissues, very often inducing extensive loss of substance in the external sexual organs and the neighboring parts; they render the prognosis of the individual case very unfavorable, both on account of the character of the original affection, as well as of the consecutive destruction which they entail.

Notwithstanding its close relation to the processes of exudation and fusion, which we have hitherto investigated, we think it necessary, on account of the novelty and scientific interest attached to the question, to devote a separate consideration to the dysenteric process occurring in the uterus after childbirth, or puerperal uterine dysentery.

The appearance presented by the inner surface of the uterus varies according to the intensity of the disease. In one case it is uneven, nodulated, and invested by a dirty reddish, or brownish fetid secretion; the projecting parts of the mucous membrane are covered with a grayish-yellow or firm greenish exudation, which here and there presents a fur-furaceous exfoliation, and the subjacent mucous membrane itself is gene-rally converted into a yellow slough; the entire surface may thus in the advanced degrees present an appearance exactly resembling the impeti-ginous condition of the intestine in dysentery. The tissues of the uterus are infiltrated throughout with serum, and, as in the intestine, we find the projections to be more particularly owing to an accumulation of the serous fluid at certain points. In another and more advanced degree, which always runs a very rapid course, the internal layer of the uterus is found degenerated into a brownish-black, friable, loose or detached mass; the uterine cavity contains a fetid matter resembling coffee-grounds; the uterine tissue is flabby, pale, discolored, and more or less infiltrated with the sanious matter. The process may thus be said to represent essentially, what we must call, if consistent in our terminology, dysenteric putrescency of the uterus.

The uterus in this case is always very large, or, in other words, its in-volution is eminently retarded.

It is an additional evidence of the nature of this affection that it is often seen combined with true dysentery, or with the dysenteric process on the mucous membrane of the colon. The puerperal diseases occurring during the prevalence of a dysenteric epidemic therefore deserve a more careful examination and appreciation in reference to this point, both at the bedside and in the dead-room.

These processes are scarcely ever isolated, but are almost invariably complicated with others. The degree of connection existing between them and the complications, and between the complications themselves, differs very much; we shall consider these points more fully, as we are about to examine the more important of these processes separately.

2. *Inflammation of the veins and lymphatics of the uterus.*—Both, but especially phlebitis, are important puerperal diseases.

Uterine phlebitis is generally a primary affection, originating in the open mouths of the veins at the insertion of the placenta, and caused as well by their laceration as by contact with the external atmosphere, with the traumatic secretion of the part, and with the product of exudation on the internal surface of the uterus. It is either confined to a small portion of the veins, or it spreads over the greater part of the veins of the uterus belonging to the spermatic or uterine system of vessels. In the latter case, a secondary inflammation of the trunk of the spermatic vein, brought on by coagulation of the blood, may on the one hand extend through the vena cava to the right auricle, or on the other along the iliac and the

crural veins, to the cutaneous veins of the lower extremity ; in this case the symptoms of phlegmasia alba dolens are induced.

The resulting products differ very much. There is no doubt that coagulable lymph is frequently secreted, which causes the venous parietes to become agglutinated to one another, or to a contracting plug of coagulum ; but in most cases pus is formed, which is variously discolored, presents a dirty greenish, or brownish, or chocolate-colored hue, with a fetid odor, varies in density, and is more or less sanious (septic phlebitis). In consequence of exacerbations, the same portions, or, if the disease extends, consecutive sections of the uterine venous system, may present various exudations at the same time or in succession.

Metrophlebitis undoubtedly sometimes occurs as the sole and primary disease, but in the vast majority of cases it is complicated with exudative processes on the internal surface of the uterus. This combination commonly takes place from the commencement, or the phlebitis supervenes upon and is induced by the exudative process ; or, lastly, phlebitis may exist for a short period in an isolated form as the primary disease, and give rise to a single or to repeated exudative processes.

We thus find that the combined processes are closely related to one another, in reference to their essential characters and the nature of their product ; this and other points will become more apparent from the description of the chief anatomical symptoms which we are about to give.

If incisions be made in various directions from the point of insertion of the placenta, to the lateral parietes of the uterus and the adjoining broad ligaments, a large number of veins become apparent, which are dilated and varicose, and filled with yellow or greenish-yellow viscid pus, or even with chocolate-colored sanies. Their orifices at the placental portion of the uterus, are either closed up by loose pale coagula, or they are covered over with an exudation which attaches itself to the spongy tissue of the raw surface, or, lastly, they are exposed so that their contents exude on the application of a slight pressure. The coats of the veins are relaxed and pale, the lining membrane is opaque, and discolored by the contents of the vessels, and after a protracted duration of the disease, it appears tumefied, thickened, partially gangrenous and ichorous. The tissue surrounding the veins, and especially the cellular tissue at the lateral portions of the uterus, is infiltrated with a yellow gelatinous or purulent matter, which is much discolored if the contents of the veins are ichorous ; the tissue is relaxed, soft, friable, and lacerable. At different points there are abscesses of greater or less dimensions, which not unfrequently burst internally, and discharge their contents into the uterus.

The internal surface of the uterus presents purulent and ichorous exudations, the products of primary or secondary processes, or of both. The tissues throughout are in a state of disorganization or putrescence, becoming dissolved in a manner analogous to the exuded product, and being attacked from the various foci of destruction within the parietes of the uterus themselves. The discoloration advances as far as the peritoneum, and the affection may, therefore, be recognized by the external appearance, as well as by the general habit of the organ. The fusion occasionally predominates at one portion of the placental segment of the

uterus, involves the entire thickness of the parietes, and causes the portion to be detached, and to pass into the uterine cavity in the shape of a pulpy, discolored, semifluid plug.

Uterine phlebitis often runs a rapid course, with intense typhoid symptoms, proving fatal by uterine paralysis ; or it proceeds more slowly under circumstances preventing a general infection of the blood, even when the product is of a putrid character, and then proves fatal by the secondary destruction set up.

Inflammation of the uterine lymphatics is, on the whole, less frequent than phlebitis, and is generally complicated with the latter. When it occurs, the lymphatics, and particularly those of the lateral and posterior portions of the uterus, of the ovary, and the Fallopian tubes, become dilated and varicose, their coats pale and opaque, the lining membrane dull and furred, and they contain a yellow, yellowish-green, purulent fluid. By these characters they may be traced into the neighboring hypogastric and lumbar plexuses, and into the associated glands, of the lymphatic system.

Inflammation of the veins and lymphatics of the uterus is generally the source of secondary occurrences, the so-called metastases, or lobular foci of inflammation (lobuläre Entzündungsheerde), in the most various tissues and organs, as well as of exudative processes occurring in serous and mucous membranes during the later stages of puerperal disease.

3. *Inflammation of the peritoneum (peritonitis puerperalis), viewed in connection with puerperal inflammations of other serous membranes.*— Peritonitis is known as a very common puerperal disease ; in rare cases it actually constitutes the original (primary) puerperal exudative process, and as such remains isolated. It more frequently simulates this form, inasmuch as the processes with which it was originally complicated have become retrograde or imperceptible, or have actually ceased after the discharge of their products has been effected. We most frequently find it complicated with the puerperal affections already examined—viz. with the exudative processes occurring on the internal surface of the uterus, with metrophlebitis and inflammation of the uterine lymphatics. The pathogenetic relations of puerperal peritonitis, and especially its relations to the last-mentioned puerperal processes, have been much discussed, but the subject has not as yet been adequately elucidated.

We commence with a statement of the anatomical signs presented by puerperal peritonitis, in reference to its extent and terminations, the quantity and quality of the effusion, and the coexistent degree of reddening and vascular development.

Puerperal peritonitis is not unfrequently limited to the peritoneal covering of the uterus and its appendages, when it presents more or less redness, with more or less distinct congestion and a thin partial lymphatic exudation, or a more dense and extensive layer of a viscid and consistent or loose and fluid secretion.

We not only find the peritoneal covering of the internal sexual organs attacked in this way, but also the peritoneum of the entire hypogastric abdominal region. The disease may even spread over the whole parietal and intestinal peritoneal laminæ ; the symptoms, however, at the same time predominating on the peritoneum of the internal sexual and adjoining organs.

The entire peritoneum is often uniformly involved in the disease, not only without any predominance of the symptoms in the sexual organs, but sometimes even with an apparent subordination of these symptoms.

The products of these processes vary very much; they may be firm, yellowish-gray concretions, loose, yellowish, membranous, grumous, gelatinous, or fibrinous coagula, which glue the intestines to one another, or to the parietes of the abdomen, or they may be yellow and greenish-yellow, thin, sero-purulent or thick purulent, dirty green and brownish, red, hemorrhagic, thin, opaque, sanious effusions, the result of septic peritonitis. The product is sometimes very limited in amount, and may merely present a thin covering of the internal sexual organs, or a few membranous or fibrinous flocculi of coagulable lymph, scattered through the abdominal cavity; but in the case of universal peritonitis it is generally extremely copious, whatever the particular variety of the product.

The vascular development and redness is, especially in the last-named cases, very slight, and bears a marked disproportion to the quantity of the exudation.

This fact in itself, and more particularly when examined in connection with combined processes occurring in the uterus, and numerous analogies and observations made at the bedside, justify the views we are about to propound, relative to the genesis of puerperal peritonitis and its connection with puerperal processes in the uterus.

Puerperal peritonitis not unfrequently arises by mere contiguity of tissue, from an exudative process affecting the internal surface of the uterus, or from metrophlebitis. It may remain confined to the internal sexual organs, or become generally diffused, and this occurs the more frequently the more the following circumstance prevails.

The disease is often, and even generally, the result of a primary condition of the blood of the female which predisposes to exudative processes, and is totally distinct from the physiological tendencies of the blood during pregnancy. This proclivity is evidenced by exudative processes on the mucous membrane of the uterus, the intestine, and various serous membranes, by exanthematic processes on the superficial integuments, by a revival of tubercular disease, &c.; and both epidemic and endemic influences and individual causes give it a peculiar character which becomes apparent in the product.

Under such conditions peritonitis will be the more liable to arise, the more the peritoneum has suffered by the revolutions in its local relations during parturition, by the excitement of the large organ, the uterus, which it invests, and by the concurrent disturbances in the circulation; the more exudative processes, or metrophlebitis, and various reactions consequent upon uterine lesions occur in the vicinity of the peritoneum, and especially in the uterine mucous membrane; the more the peritoneum has been previously affected by the contiguity of tissue to the internal sexual organs.

In this case peritonitis is a primary disease, and is either the first and not unfrequently the only puerperal affection, or it occurs, as is more frequently the case, concurrently with an exudative process of the uterine mucous membrane, or soon becomes associated with the latter; it invariably takes its origin in the above-mentioned predisposition existing in the blood.

Like other exudative processes that occur simultaneously or consecutively, we also find that peritonitis is often the result of a secondary disorganization of the blood, caused by the absorption of the products of exudation upon the external surface of the uterus, or by the direct admixture of the products of metrophlebitis with the blood. In this case it presents the characters of a secondary inflammation, and is commonly complicated with exudative processes on other serous, synovial, and mucous membranes, and with capillary phlebitis in the most different organs and tissues, the so-called lobular infarctions (Lobular-Infarcte) and abscesses.

The products of the peritoneal inflammation in either case correspond in character with those of the exudative processes affecting the internal surface of the uterus and of metrophlebitis, whether they occur simultaneously, or whether they precede the former.

Puerperal peritonitis is developed with more or less rapidity, and in the majority of cases proves fatal by inducing abdominal paralysis; or it leaves various morbid sequelæ. Those exudative processes are remarkable which result from a very rapid disorganization of the blood, and prove fatal within a few hours, or within two to three days, and are accompanied by paralysis and collapse, affecting the uterus immediately after parturition, and by a sanguineous ill-looking effusion.

Puerperal peritonitis, as may be gathered from the above, is almost always remarkable for its very exudative, or croupy, character.

4. *Puerperal inflammation of the ovaries and Fallopian tubes.*—We shall examine these affections when we speak of the diseases of the respective organs. The first is always complicated with one of the processes that have been just discussed, and probably always with an exudative process on the inner surface of the uterus; the affection of the Fallopian tubes is invariably the result of an extension of the uterine exudative process.

5. *Phlegmasia alba dolens (sparganosis).*—Various theories have been formed in reference to this disease of the puerperal state, from its symptoms in the living subject; and very different views have been even propagated with regard to its anatomical relations. The ancient and modern dicta that were based upon anatomical investigations may almost all be viewed as the result of preconceived notions, and of examinations, undertaken with a view to establish favorite theories, or conducted without the necessary distinction between essential and accidental circumstances being observed. It is only of late that the subject has been examined in the dead body with an unprejudiced and discriminating judgment, and that an anatomical basis has been obtained, which, though it may not be applicable to all conditions that are included under the head of phlegmasia alba dolens, and though it may not always have been properly interpreted, still appears to afford sufficient security.

Two lesions seem to be essentially connected with this affection. It either depends upon an inflammation of the veins of the inferior extremity, and especially of the crural vein, or upon an inflammation of the cellular tissue, which gives rise to the most various products. The latter form is particularly likely to cause the characteristic symptoms which a so-called sero-lymphatic or sero-purulent product, i. e. fibrinous or purulent exudation diluted by a large amount of serum, induces. It is characterized

by very slight reddening and vascularity, and must be considered as an exudative process. In this shape it often extends to the crural fascia, the neurilemma, the lymphatic vessels, and is sometimes complicated with exudations in the synovial capsules of the knee and the hip-joint. As we have already observed, it gives rise to the most various products, and terminates accordingly in tedious œdema, in sclerosis, suppurative fusion, and gangrenous destruction of the cellular tissue. It proceeds from a primary or secondary dyscrasia of the female, and is in either case generally combined with various other puerperal processes. This form of phlegmasia alba may, like the one that originates in phlebitis, occur, if similar causes prevail, independently of the puerperal state, in unmarried women and men ; and we find this to be particularly the case as a result of exanthematic and typhous processes, of the most various exudative processes, of cholera, dysentery, inflammation of the lining membrane of the vessels, of endocarditis, &c. The disease may attack the upper extremities and even the trunk, though in the puerperal state it generally affects the lower extremities. It occasionally proves fatal by its sequelæ, but more frequently by the associated puerperal processes.

Crural phlebitis generally passes from the uterine to the internal iliac vein, and either attacks the deep-seated or superficial veins, or both. An inflammation of the lymphatic vessels is often superadded.

Summary of the Anomalies in other Organs, accompanying the above-described processes.

Besides the changes which occur in the original seats of the puerperal processes hitherto examined, there are so many, important, and various anomalies in other organs and tissues, that it is not sufficient merely to give a supplementary account of the anatomical results, but that as copious an explanation of them as possible, becomes necessary. We shall, in the first instance, describe and account for the general appearance of the body, and the individual organs, and then arrange the separate morbid processes as much as possible in groups, according to their mutual resemblance.

The dead subject presents a remarkable disfiguration of the countenance, tumefaction and discoloration of the external genitals, excoriation, ulcerative destruction of various characters, with or without laceration of the perineum, various vaginal discharges, tympanitic distension of the abdomen, a livid erythema of the common integument at different parts of the body, white and often large coalescing miliary vesicles on the thorax and abdomen. Yellow, greenish, bilious, feculent, chocolate-colored fluids escape from the mouth.

The abdomen presents, in most cases, even if the peritoneal inflammation has been slight or entirely absent, a tympanitic distension of the intestines ; this symptom is most developed in universal peritonitis ; the entire intestine is then so much distended by gases, that it causes impressions upon the uterus, and forces the epigastric contents of the abdomen into the cavity of the diaphragm, and with the latter into the thorax as far as the fourth and third ribs. The firmer the exuded (plastic)

matter, the more firmly the intestinal coils and the other abdominal organs are agglutinated to one another and to neighboring organs. The coagulable lymph is chiefly contained in the lower segment of the abdominal and pelvic cavity, but also in the lateral parts, of the abdomen, between the mesenteries and in the vicinity of the large epigastric viscera, within spaces that have become more or less circumscribed by the adhesions. It not unfrequently causes, especially on the surface of the liver, shallow depressions, and gives to the superficial layer of this organ, if of a purulent and sanious character, a greenish, and to the spleen a blackish, tinge. The reddening and vascularity of the peritoneum are generally inconsiderable; but most evident at those parts, which are free from pressure, and take the form of narrower or broader striæ. The membranes of the intestinal canal are all tumefied, the interstitial cellular tissue infiltrated, the layers easily distinguishable and lacerable. The intestine generally contains, in addition to a large quantity of gas, a yellow, serous, feculent fluid, which mounts up to the duodenum and stomach. This fluid is in part the product of an exudative process that occurs in the greater part of the intestinal mucous membrane, and which we shall have occasion subsequently to examine more closely. The duodenum and the stomach may also be found to contain a copious amount of yellowish-green or intensly green biliary fluid.

We have here to advert briefly to two symptoms that occur during the course of puerperal peritonitis, and which not unfrequently coexist—they are, vomiting of the biliary matters contained in the duodenum and the stomach, and of the sero-feculent matters that rise from the intestine, and diarrhœa. The former is to be explained by the paralysis of the muscular coat of the intestine, caused by the peritoneal exudative process, and the fixation of the intestine by plastic exudations; it commences at the duodenum and the stomach, the peritoneal covering of which generally remains unattached. The latter is caused by the exudative process, and the consequent irritation of the intestinal muscular coat, which forms a counterpoise to, and even counteracts, the paralysis at some points; it is the more frequent and the more considerable the less marked the paralyzing influence of the peritoneal affection is.

Almost all organs appear in a state of relaxation, which is proportioned to the primary or secondary dyscrasia of the blood, and to the extent in which the blood has become deprived of its fibrine by the fibrinous exudations caused by inflammations of the peritoneum, the pleura, &c. It is owing to a moistening or imbibition of the tissues with the attenuated serum of the blood, which easily exudes through the vascular coats, and is for the same reason coupled with pallor or discoloration, owing to the coloring matter which adheres to the serum. In the abdomen we find that the kidneys and the liver are chiefly distinguished by the softening, pallor, or pale red discoloration, œdema and imbibition, relaxation and friability of their tissues. In the thoracic cavity, the lungs are chiefly affected by these and similar deviations; the muscular portion of the heart, too, is, like the other muscles, and especially those that are involved in the peritoneal inflammatory process, soft, pale, moist, and lacerable. All the serous membranes and the lining membrane of the vessels are infiltrated with serosity, and are more or less reddened, and

the serous cavities contain various quantities of a transuded, pale or dark-red serum. The brain alone, as in numerous other allied processes, e. g. in typhus, forms an exception, inasmuch as it appears denser and harder, drier and paler, than usual.

The spleen is very frequently, though not always, tumefied; it is so particularly in secondary disease of the blood, whether or not accompanied by the secondary processes (deposits), that we shall subsequently have to notice.

The lungs are reduced in size, and denser, in consequence of the upward pressure exerted by the contents of the abdomen; their inferior lobes are of a dark purple color, and in a condition of passive hyperæmia.

We now proceed to enumerate the separate morbid processes in the different organs, and to point out their relations to the original puerperal disease.

Our first attention is due to the exudative processes on the various mucous and serous membranes. That affecting the intestinal mucous membrane is of particular importance. The entire tract is generally involved; it is but slightly reddened, and commonly exhibits a thin, watery, serous, or viscid gelatinous, or gelatino-purulent or genuine purulent product; the tissue fuses, and the submucous cellular tissue is more or less infiltrated. In this manner the diarrhœas of the puerperal state are established. The exudation is rarely of a firm, fibrinous, or croupy nature, but most commonly its serous character predominates, and this is the more the case the larger or more fibrinous the product, resulting from the coexistent attack of peritonitis. In certain cases the process that takes place on the mucous membrane of the colon assumes a dysenteric type, and as in the above-named forms, corresponds to the exudation upon the internal surface of the uterus or to the product of metrophlebitis. Similar processes, though generally accompanied with a coagulable product, are occasionally discovered upon the mucous membrane of the stomach, the œsophagus, and the bladder, and in the lungs in the shape of (partial) aphthous pneumonia; this is chiefly the case when the blood has not been exhausted of its fibrine.

Among the exudative processes that take place on serous membranes, the most frequent, after that occurring on the peritoneum, is pleuritis, which is often coexistent with peritonitis; pericarditis is of less frequent occurrence. We also meet with exudations in the synovial bursæ, and especially in that of the knee-joint, the sterno-clavicular and humoral articulations, and, lastly, in the capsule of the humor aqueus. The exudations are generally very copious, fibrinous, and purulent. A thin soft exudation is often found upon the dura mater, accompanied by a slight reddening of the latter.

All these processes may be variously combined, and they are dependent upon the primary or secondary disorganization of the blood, and especially upon that caused by the absorption of pus in metrophlebitis.

Next in order come the processes dependent upon secondary phlebitis of the larger veins, and of the capillary venous systems of various organs and tissues.

The former are generally developed in the vicinity of the original morbid affection, as in the plexus pampiniformis, the trunk of the internal

spermatic vein, the internal iliac and crural veins; though they frequently, too, are generated at a distance, as in the cerebral sinuses and the pulmonary artery. These give rise to the so-called metastases or lobular abscesses, which we shall now proceed to examine.

We often find larger or smaller circumscribed spots in the most various organs and tissues; the dark-red points of congestion, or small accumulations of pus or sanies, which we have repeatedly adverted to. They are remarkably frequent and numerous in the organs of sanguification, especially in the lungs and the spleen; they are next seen in the kidneys, and more rarely in the liver; they are occasionally met with in the brain, in the thyroid and parotid glands; in all muscles, particularly in the heart; in fibrous tissues, as in the dura mater and the periosteum. Again, they are very common in the mucous tissue, especially of the bladder and the intestines; they occur throughout the cellular tissue, but they seem to predominate in the cellular tissue of the extremities, of the mediastina, of the neck, the iliac muscles, and the intestines and stomach.

We have already demonstrated that these processes are either genuine exudative processes, or that they consist in a coagulation of the blood within the capillaries (capillary phlebitis). In the latter case the coagulum fuses in a manner corresponding to the disease of the blood, and to the deleterious matter absorbed into the blood, and forms a purulent sanious fluid or gangrenous pulp (metastasis puerperalis septica).

They may probably be invariably considered as the result of a secondary infection of the blood, of a poisoning of the blood by the introduction of some product from the original nidus of disease, and particularly of venous pus and sanies in metrophlebitis. They consequently always give rise to purulent and sanious products, and terminate fatally as capillary phlebitis. They enter into various combinations with one another, and with the exudative processes occurring upon serous and mucous membranes. Owing to their position at the surface of the organs, we always find that pleurisy supervenes upon their occurrence in the lungs, and peritonitis upon their deposition in the spleen.

A black softening of the mucous membrane of the fundus ventriculi, or of the œsophagus, or of both at the same time, which is indicated during life by the vomiting of black coffee-grounds-like matter, is of frequent occurrence. It not rarely reaches that degree of intensity, that the fundus of the stomach, and sometimes the diaphragm also, and the œsophagus, with the adjoining cellular tissue and mediastinum, are ruptured, and the fluid that would have been evacuated by the mouth is effused into the abdominal or thoracic (especially the left) cavities.

After difficult labor, the cartilages of the pelvic synchondroses are liable to inflammation, in consequence of the traction exerted upon them, and if the blood has assumed a septic constitution, the inflammation may terminate in gangrenous fusion of the cartilage, the latter being converted into a dirty brown and very much discolored fluid, contained within the investing ligamentous tissue.

The blood contained in the cavities and larger vessels presents various and more or less evident changes. Its fibrine may be converted into consistent, viscid, greenish-white, or yellowish coagula; or after previous extensive discharges of fibrine it may be attenuated, watery, exuding

through the coats of the vessels and the adjoining tissues, and presenting but few and trifling, gelatinous, soft coagula. Again, after previous purulent or sanious absorption, it is of a dirty brown-red or chocolate color, viscid, glutinous, depositing dirty white, opaque, fibrinous concretions, which in the heart form numerous ramifications, or presenting dark-red coagula, which are paler at the surface, and fusible. Lastly, if the disease has run a rapid course, the blood is much reduced in quantity, and even without defibrination having taken place, it is attenuated and discolored, and transudes all the tissues. The fibrine is sometimes found deposited on the valves of the heart in the shape of vegetations, without the demonstrable occurrence of previous pericarditis. The severe jaundice affecting women during the puerperal state is always dependent upon pyæmia, and never upon an appreciable derangement of the liver.

The formation of bone occasionally noticed on the external and internal table of the skull after parturition is, as we have already observed, in no connection whatever with the puerperal process.

6. *Termination and consequence of the puerperal processes.*—We confine ourselves at present to an account of those terminations and consequences of the fundamental puerperal processes, which are not to be inferred from the previous remarks.

Puerperal peritonitis generally terminates in the same manner as ordinary peritonitis; we notice as particularly important the unfavorable terminations in suppuration—phthisis—of the peritoneum and the adjoining tissues (ulcerative perforations of the diaphragm, the abdominal parietes, the intestines, the bladder, the vagina, &c.), and in peritoneal tuberculosis. The exudations upon the internal sexual organs may become converted into cellular tissue, and by fixing the tubes in an unfavorable position, even without occlusion of the fimbriated extremity, cause sterility.

The exudative processes occurring on the internal surface of the uterus, as well as the exudation in the uterine parenchyma accompanying the former and metrophlebitis, not unfrequently degenerate into suppuration of the uterus, and the consequent purulent and sanious abscesses, extending chiefly from the point of insertion of the placenta in various directions, may discharge themselves into the peritoneal cavity. The affection generally runs its course as acute uterine phthisis.

A very remarkable and important result of the exudative processes on the internal surface of the uterus is tabes of the uterus, which is manifested by extreme brittleness and friability of the uterine fibre. The uterus very rarely attains such a degree of involution as to resume the size of the unimpregnated organ; it generally remains considerably enlarged, of the size of a duck's egg or a man's fist; its tissue at the same time is porous, of a pale red, and at some parts of a slate color; the insertion of the placenta continues visible, by the relaxation of the tissue and the irregularity of the inner surface, or the mucous membrane is at this place invested by a yellow or yellowish-white ashy substance, the remains of the exudation, and generally presents a retiform appearance.

Metrophlebitis, by the suppuration of the coats of the veins, gives rise to the formation of abscesses in the uterine parenchyma, which not unfrequently anastomose at various points, and thus form branched sinuses. The disease is very persistent if the uterus passes into a state of maras-

mus, and if it maintains dirty brown hemorrhagic and fetid exudations on the internal surface of the uterus.

SECT. III.—ABNORMITIES OF THE FALLOPIAN TUBES.

§ 1. *Defect.*—The tube may be absent on either side if there is a corresponding defect of one-half of the uterus, but this certainly is not always the case, inasmuch as it is not only often present when there is not even a trace of a uterine rudiment, but as it may exist in the shape of a solitary coiled tubercle even when the ovary is wanting.

In many cases the Fallopian tube may be imperfectly developed, its coat thin, its parenchyma impoverished, and its passage narrowed; or the uterus being normal, it may merely appear as an excrescence of the former, terminating blindly above the uterine horn, or it may be inserted either at its normal place, or elsewhere, without presenting an open channel.

When a Fallopian tube is absent, the peritoneum occasionally presents a fringed process, in imitation of the morsus diaboli.

§ 2. *Anomalies of Calibre.*—These consist in dilatation or contraction of the Fallopian tube; in the latter case obliteration may result.

The former is very commonly the consequence of a catarrh of the tube owing to retention of the mucous secretion from contraction, obliteration or obturation of the orifices; it may degenerate into dropsy of the tube, an affection of which we shall say more at a future period.

The latter consists—independent of the natural contraction of the tube in the decline of life—chiefly in a diminution of the passages by tumefaction of the mucous membrane, or in obstruction of the same by mucus. The contraction may pass into complete closure or obliteration of the tubes; it chiefly affects the uterine orifice in consequence of catarrh; the fimbriated extremity is often closed up by cellular formations, or organized peritoneal exudation (atresia tubæ). The imperforate condition of the Fallopian tubes is of importance in regard to sterility.

§ 3. *Anomalies of Position and Direction.*—Under this head we reckon the very unusual congenital irregularities in the entrance of the tube into the uterus, whether communicating with the cavity of the latter, or terminating in its tissue blindly.

Among the acquired abnormities the deflections and curvatures of the tubes become the more important, the more the unattached end of the tube is turned away from the ovary and fixed in its abnormal position by the products of peritoneal inflammation. It is found variously agglutinated to the neighboring tissues, and is particularly apt to become reverted upon and fixed to the posterior surface of the broad ligament, the ovary, and the uterus.

In consequence of chronic catarrh, or tubercular disease of its mucous membrane, accompanied by hypertrophy or thickening of its parietes, the Fallopian tube is apt to assume a serpentine tortuous course. Or if the ovary enlarges, it may be extended to an unusual length, and its

coats thinned; and if it happens to wind round the former, it is much stretched.

The tube has, like the ovary, occasionally been found in the abdominal ring, within an inguinal hernia.

§ 4. *Diseases of the Tissues.*

1. *Hyperæmia, hemorrhage.*—Hyperæmia of the Fallopian tube is almost always a symptom of general congestion of the sexual organs, and especially of the uterus. In rare cases, however, the hyperæmia of the tube predominates, and may lead to hemorrhage of the tube, in which case a larger or smaller quantity of blood is effused into the cavity of the peritoneum.

We have twice had occasion to observe the occurrence of such hemorrhage in the course of abdominal typhus; the left tube was distended, its mucous membrane of a purple tint, and congested. We have once seen it in the body of a female who was attacked, three days previous to her confinement, with pleuritis and hepatitis, and in the fourth instance it was associated with retroversion of the uterus. Barlow has met with this condition in purpura, in consequence of or connected with abortion; and Brodie has observed it in a case of retention of menses in the uterus, owing to occlusion.

2. *Inflammation. a. Catarrhal inflammation.*—Chronic catarrh, or blennorrhœa of the Fallopian tube, is a very common disease; it is frequently a residue of a puerperal affection of the mucous membrane of the tube; or the catarrh may have extended from the vagina and uterus to this point, and is coexistent with vaginal and uterine catarrh, or persists after the cessation of the latter.

At the same time the tube is variously dilated, its course tortuous, its coats thickened; the mucous membrane is tumefied, purple, slate-colored or of a blackish-blue tint; the passage contains a viscid, transparent, milky white or creamy, or a bluish-gray, or yellow, purulent mucus.

Catarrh of the Fallopian tube, by spreading to the fimbriated extremity gives rise to peritoneal inflammation in the vicinity of the orifice, and thus the free termination may become adherent to the neighboring tissues and be closed up, whilst the uterine orifice is obstructed and occluded by the catarrhal tumefaction of the mucous membrane. Catarrhal inflammation in this manner induces sterility.

The chief seat of catarrh is the external distended portion of the channel, and it is here that we find the greatest accumulation of blennorrhoic secretion.

Under the above-mentioned condition, viz. occlusion of the orifices, catarrh of the tube is very often converted into dropsy of the tube, a condition similar to that which we have already become acquainted with in various other mucous channels and cavities. In consequence of the accumulation of secretion from obstruction of the orifices, the tube, especially towards its fimbriated extremity, becomes so much distended, that that which before represented a tortuous or bent channel, is now converted into a simple sac. At other times, several saccular dilatations form between the separate angles and the projecting duplicatures of the tubal parietes, and give rise to an imperfectly loculated pouch, which,

as in the former case, may contain blennorrhoic mucus, a puriform secretion, a true purulent inflammatory product, or, if the mucous membrane has become altered, fluids of another description. It is to be observed, that as the dilatation proceeds, the texture of the mucous membrane is changed, and the latter is converted into a serous membrane; its secretion may be a thin, watery, serous, or albuminous synovoid, colorless liquid, giving the tube the appearance of a transparent serofibrous bladder; or it may be variously colored, yellowish, brown, blackish-green, chocolate-colored, inky, and more thick and flocculent, consisting in part of inflammatory products on the internal surface of the membrane.

The hydropic Fallopian tube not unfrequently attains the size of a duck's or goose's egg, or even of a man's fist; although not a usual occurrence, still it is satisfactorily proved that the contents are sometimes discharged into the uterus, and thus carried off.

In extremely rare instances chronic catarrh of the Fallopian tube becomes acute, and passes into suppuration; its contents may then be either poured into a cavity of the peritoneum, which has been circumscribed by adhesive inflammation, or into the perforated intestine, which has been previously agglutinated to the tube.

b. Exudative processes.—An exudative process scarcely occurs on the mucous membrane of the Fallopian tube, except in combination with a similar condition of the internal uterine surface after childbirth. The tubes are tumefied and infiltrated; their mucous membrane is variously reddened, discolored, excoriated, softened, and everted at the fimbriated extremity; the passage of the tube is dilated, especially at its outer end, and filled with various products, purulent and sanious fluids, and in uterine croup with coagulable lymph, assuming the shape of a tubular concretion. The exudative process has extended from the uterus to the tube.

3. *Adventitious growths.* *a. Cysts.*—Serous cysts are very often formed at the fimbriated extremity of the tubes, and in its vicinity; and they are generally attached by a pedicle, which sometimes attains a considerable length. They scarcely ever become larger than a bean or hazel-nut.

b. Fibroid tumors.—These are not frequent; they are rarely larger than a pea, and occupy the parenchyma of the tube in the shape of round or discoid tumors.

c. Tubercle.—Tubercle of the Fallopian tubes (Fallopian mucous membrane) is generally associated with uterine tubercle; but it is remarkable that it sometimes occurs independently of the latter, or in a condition of higher development. It therefore follows that in many cases of tubercular affection of the internal sexual organs, the mucous membrane of the Fallopian tube is the primary seat of disease.

Tubercle of the tube is almost always presented to us in the dead subject, in the shape of tubercular infiltration and complete disorganization of the mucous membrane; the latter being converted into a softened purulent layer of yellowish-white, cheesy, lardaceous matter, which is cracked and friable, and chokes up the passage. The tube is more or less swollen, its course tortuous, it is hard to the touch, and its parenchymatous coat thickened, and converted into a dense lardaceous tissue.

The fimbriated extremity presents a very peculiar appearance; the mucous membrane, which is infiltrated with tubercular matter, being pushed out in the shape of a cauliflower excrescence, and everted upon the peritoneum.

Opportunities are very rarely afforded of observing the disease at its commencement, which occurs in the shape of a deposit of crude, gray, discrete, or agglomerated tubercular granulations. In the above-described shape, it must doubtless be viewed as the result of a tumultuous localization of the general disease, occurring under symptoms of congestive inflammation. The remarks made in reference to uterine tubercle apply to this affection.

d. Carcinoma.—Except when involved in cancer of the peritoneum, the tube is not affected by this disease; and even an extension from the uterus or other adjoining tissues by mere contiguity, after pseudomembranous attachments have been effected, is very rare. Still I have noticed one case of ovarian cancer, in which the tubes, without being agglutinated to the former, were thoroughly diseased; the parietes were very much thickened, callous, contracted in their long diameter, and curled up.

SECT. IV.—ABNORMITIES OF THE OVARIES.

§ 1. *Defect of Formation.*—It is very unusual for one of the ovaries to be wanting, if the sexual apparatus is otherwise normal.

The ovaries often appear, together with the other portions of the sexual organs, in a state of imperfect development, and small; and, on account of the depth at which the Graafian follicles are placed, of uniform density and hardness, and with an even and smooth surface.

§ 2. *Deviations of Size.*—We find various enlargements occurring in the ovaries, which form a contrast with the just-mentioned smallness of the ovaries and their diminution at the decline of life; the latter affection only comes within the domains of pathology if it occurs prematurely. We shall have occasion to notice them all under the head of textural disease, and therefore do not here enter into a more minute examination of the subject. We here merely allude to that form of ovarian dropsy which results from the excessive development or hypertrophy of one or more Graafian vesicles, as a subject coming under the above denomination; but it will be more practical to consider it fully when we speak of the formation of ovarian cysts.

§ 3. *Diseases of the Tissues.*—These diseases affect either the cellulofibrous substance (stroma) and the fibrous capsule of the ovary, or the follicles, or both together, as we shall have occasion to explain in the subsequent sections that relate more particularly to this point. We confine ourselves to the most important and conspicuous affections of the follicles and their contents.

1. *Hyperæmia, Apoplexy.*—Hyperæmia of the ovary, affecting both its stroma and the external layer of the follicle, occurs physiologically in menstruation; but it also accompanies numerous pathological pro-

cesses in the sexual apparatus, and is sometimes permanent. Its cha-
racters are tumefaction of the ovary, softening of its tissue, vascu-
larity, and darker color; permanent hyperæmia gives rise to a gradual
increase of size, to hypertrophy of the stroma, and enlargement of the
ovary.

Hyperæmia affecting the more developed follicles that are seated at
the surface of the ovary often induces effusion of blood into the cavity
of the follicle or apoplexy. One or more cysts, varying in size from a
pea to a hazel-nut, are found in the ovary; they project more or less
above its surface, after having perforated the fibrous sheath of the ovary,
and are at once recognized by their contents being visible through the
parietes of the follicle. If seen shortly after the occurrence of extrava-
sation, they are tense; but more commonly a certain amount of coagu-
lation has been effected in their contents, and they then appear slightly
collapsed, and present fluctuation. They now contain a dark-red
loose coagulum, which is invested by a white or colored fibrinous coa-
gulum varying in thickness. In the course of time the coagulum
assumes a rusty or yellow color, is converted into a pulp which
gradually becomes inspissated, and yields the above-mentioned fibrinous
coagulum and serosity, the latter being in its turn removed by exosmose
and absorption. The entire cyst contracts, retaining traces of the origi-
nal lining coagulum of fibrine and of its yellow deposit, and, perhaps,
also, a yellow, indurated, friable, chalky residue of the coagulated blood;
it may become reduced to less than the normal size of the follicle, and
from drawing in the fibrous sheath of the ovary, cause the appearance of
a cicatrix. The contents and parietes of the apoplectic cyst consequently
present an appearance which varies according to the length of time that
has elapsed. We very often find cysts of different dates in one or both
ovaries.

It is evident that this effusion of blood must induce a destruction of
the germ, and, at last, cause an entire obliteration of the follicle. The
cicatrix naturally always presents a greater or less resemblance to the
corpus luteum. Although the amount of effusion is often very consider-
able, rupture of the follicle and hemorrhage into the peritoneal cavity
is of very rare occurrence.

The most common cause of this affection is excessive menstrual con-
gestion, and it undoubtedly comes within the sphere of pathological in-
quiry (vide Negrier).

2. *Inflammation.*—Inflammation occurring in the ovary, independently
of the puerperal state, is limited to the follicles. The coats of a follicle
are occasionally found injected, reddened and softened, and friable; the
contents are opaque, flocculent, reddened by an admixture of blood, and
not unfrequently purulent. Each of these processes, even in its slightest
form, is followed by a destruction of the germ by means of the exuda-
tion; obliteration of the follicle soon ensues, and the first impulse is thus
given to its conversion into a common serous cyst, which in its turn may
grow into ovarian dropsy.

On the other hand, inflammation resulting from childbirth, puerperal
inflammation, involves the entire ovary, though probably in the first
instance the stroma only; it is this that generally gives rise to the sup-
puration and abscess of the ovary noticed by ancient and modern ob-

servers. It not only varies much in intensity, but, like the other puerperal processes, in kind also; this is particularly evidenced by the product and the state of the tissues. According to the manner in which it is complicated with other puerperal affections, it plays the chief, or only a secondary part, as will become apparent from the following remarks.

The ovary may be swollen to the size of a hen's, duck's, or goose's egg, presenting various discolorations, and being at the same time collapsed and pulpy, its tissue distended by a dirty yellowish-brown, brownish-green, chocolate-colored fluid, or converted into a fetid pulp; this is putrescence of the ovary.

Or the ovary may present a pale greenish, or yellowish, or reddish gelatinous viscid product, which is deposited in the stroma in large quantities; the latter being at the same time friable or semi-fluid, the follicles tumid, their coats swollen, and their contents opaque and flocculent. The ovary is at the same time enlarged and tense, as in the former case.

Again, the deposit may be serous (of a pale yellow or reddish color) or fibrinous (of a yellowish-white color), and fusible; filling the tissues, and causing the follicles to present an opaque appearance. The tissue of the ovary and the coats of the follicles are congested and more or less reddened, and both are softened and friable.

Again, the congested stroma of a moderately tumefied ovary may be infiltrated with a flocculent serosity, which is rendered opaque by plastic exudation.

In all these cases the parenchyma of the ovary is more or less ecchymosed; its sheath presents exudations of various kinds, under which differently-colored, spotted, or striated suffusions are found; the tissue at the same time being softened, and extremely friable.

These are the chief varieties and degrees of puerperal inflammation of the ovaries; they enter into complications with other puerperal processes, and especially with endometritis and peritonitis, and give rise to the same products; they differ, however, in intensity, and the inflammation of the ovary may either be the predominating disease, or, as is commonly the case, the subordinate or partial symptom of an extensive exudative process of the uterine or tubal mucous membrane, of the tissue of the uterus, or the adjoining accumulations of cellular tissue or of the peritoneum.

We have, lastly, to allude to the condition presented by the ovaries in puerperal exudative disease, when they are not themselves involved in the latter process; like the other tissues in the vicinity of the seat of disease, they are infiltrated with serum, softened, flabby, pale, and friable.

Exudative processes either affect one, or, more frequently, both ovaries at the same time, though generally not in the same degree. They may run a very rapid course, sometimes even assuming such violence as to induce a spontaneous rupture of the ovary; they prove fatal by the intensity of the general disease; or by the exudative processes with which they are complicated; or they may terminate, after a slower progress, in suppuration (phthisis) of the ovary. In the case of recovery, sterility is entailed upon the affected ovary, in consequence of destruction of the germs and obliteration of the follicles.

Suppuration either commences at separate points which gradually coalesce, or it is set up equally throughout. The parenchyma of the ovary is by degrees consumed, and the organ converted into a purulent cyst, which sometimes attains a very considerable size.

The abscess itself is sometimes borne for a long time without marked symptoms, and nature does her utmost to prevent a free discharge of it into the peritoneal cavity; for adhesions are formed between the ovary and the adjoining viscera, either in consequence of peritonitis having been combined with the inflammation of the ovary, or from circumscribed inflammations of the peritoneum having been set up in the course of the ovarian disease. Thus the ovary may become agglutinated to the broad ligaments, to the pelvic parietes, the uterus, the bladder, or the rectum and the sigmoid flexure, to the cæcum and the vermiform process and the small intestine; and it is generally attached to several of these viscera at the same time. When at last the suppurative process has eaten away the fibro-serous investment of the ovary, and caused its rupture, the discharge follows, from a yielding of the adhesions, into a circumscribed cavity; new partial inflammatory attacks of the peritoneum ensue, or the pus meets with an organ which presents firm attachments. In the former case, the circumscribed processes not unfrequently pass into universal peritonitis, or this is induced by an extravasation of the pus through the relaxed adhesions. Again, in either of these cases, the suppuration may extend to the adjoining viscera, and the contents of the abscess be discharged outwards, indirectly through a circumscribed peritoneal sac, or directly in the hypogastric or umbilical regions; or into a portion of the intestine, into the bladder or vagina. Suppuration occasionally takes place in the pelvic cellular tissue investing the iliac muscle; such abscesses pass through the femoral ring or through the ischiatic notch, and accordingly make their appearance on the thigh or the nates. They may thus discharge themselves at a considerable distance from the original nidus.

3. *Morbid growths.* *a. Cysts.*—In no part of the body are cysts so frequent, or so various as in the ovary, in the peritoneum, in the neighborhood of the internal sexual organs, or in the subperitoneal cellular tissue; as, for instance, between the laminæ of the broad ligaments, and at the fimbriated extremities of the tubes. Moreover, the size attained by the ovarian cysts is extraordinary. It is more practical to consider all the different cysts at this place, though we shall parenthetically indicate the position they occupy in morbid anatomy, and have to revert to them in the sequel. At the bedside the term ovarian dropsy is equally applied to all cysts, provided they fluctuate. We commence with the simple formations, and pass on to those which, in reference to original development, structure, growth, pathological importance, and contents, are more complicated.

a. Simple cysts.—They are of very common occurrence. There are either one or several unilocular cysts in the ovary; at times they are even so numerous, that the ovary appears converted into an aggregation of cysts. They are placed near one another, each one being formed from the stroma, independently of the other, and they have a rounded form. If they enlarge, they come into mutual contact, their parietes adhere to

one another, and they are flattened by reciprocal pressure; the impression may thus arise that several have, in the manner of the compound cysts, been formed within the parietes of the same matrix. They attain a considerable size, rarely, however, exceeding that of a man's head. In this case the solitary cyst, or one of several cysts, undergoes extreme development, whilst the remainder continue undeveloped. They generally have delicate sero-fibrous parietes, and may contain a colorless, or pale yellowish or greenish, serous, or a more consistent yellow, brownish, colloid substance, or an opaque chocolate-colored or inky fluid. In many cases they are undoubtedly formed from the Graafian follicles; and it appears that an inflammatory process is particularly liable to give the first impulse to this metamorphosis. They are probably, however, as often new formations from the beginning; and this is the more likely in those cases in which their number exceeds the average number of Graafian follicles. Allied to them are the adipose cysts of the ovaries; these we shall, however, discuss at a later period, on account of their numerous peculiarities.

β. *Compound cysts.*—They occur in the two forms described by Hodgkin. In the one, new cysts are formed in the coats of an older cyst, and although projecting into the cavity of the latter, they do not actually grow into it; the oftener this process is repeated, the more complicated the morbid product becomes. In the other, an endogenous generation of cysts is effected, cysts being formed upon the internal surface of another cyst, and being either sessile or pediculated; the matrix is sometimes entirely filled, the cysts discharge themselves into it and become adherent to it, and subsequently a third order of cysts may be formed within them, &c. The two forms are often seen in the same adventitious growth.

These cysts are capable of very extensive development; to them and to the following variety the large encysted ovarian dropsies are due. The separate cells or loculi contain the above-mentioned different substances, and their parietes, especially those of older cysts, are generally of considerable thickness, and of dense texture. They, too, may probably in the first instance be developed from a Graafian vesicle as simple cysts, or they may form as adventitious growths; the remaining substance of the ovary is spread out at the base of the cyst; it is, as it were, thrown open, and its tissue condensed.

γ. A third form, which very much resembles, and is closely allied to, the last, is of a cancerous nature, and belongs to the areolar variety of carcinoma. In the shape which we are about to describe, it rarely occurs anywhere but in the ovary. It is an accumulation of numerous fibrous sacs, which contain various substances, but for the most part a glutinous, viscid matter. They diminish in size from the circumference towards the interior, and especially towards the base of the morbid growth; so that the latter represents a condensed alveolar mass, the alveoli or follicles of which consist of a white, shining, fibrous tissue, and contain a colorless or grayish, yellowish, yellowish-green, or reddish viscid gelatine. We have here an areolar cancer, the peripheral follicles of which are converted into large sacs. This species of ovarian dropsy, which, for the sake of distinction from the other varieties, we term alveolar dropsy,

is proved to be malignant, not only by its being accompanied by well-marked cachexia, but also by its complication with cancer (especially of the medullary variety) in the same organ, and with other varieties of cancer in other organs, as the peritoneum, or the stomach, and moreover by its complication with mollities ossium.

As already remarked, it attains an enormous size, and like the composite cysts, occasionally exists in both ovaries at the same time. In the composite as in the alveolar cyst, one peripheral follicle is subject to preponderating growth, and establishes ovarian dropsy.

To the above special observations we add the following remarks as important for the diagnosis. Generally but one ovary is affected, though the two are often attacked successively, so that the increase of size is much more considerable in one than in the other.

The enlarged ovary remains within the pelvis as long as it does not exceed certain dimensions ; it either continues freely movable between the uterus and its lateral appendages and the rectum, or becomes fixed, and, as it were, wedged in by the formation of false membrane. If it increases still further, and is adherent to the pelvis, it grows into the abdominal cavity; otherwise it leaves its previous position, and rises into the abdomen, where it continues movable, until, in consequence of peritoneal inflammation, it has formed adhesions with adjoining viscera, or becomes fixed by entirely filling out the cavity. In the course of this change of position, it drags the uterus after it by means of its ligament, so that this organ, together with the vagina, is not only elongated, but obtains a slanting form, which is recognizable by the oblique and elevated position of the os tincæ. (Page 214 and 215.)

If both ovaries are involved in the disease, inasmuch as they are generally affected successively, and one is less enlarged than the other, the smaller one remains in the pelvis, and its retention is proportionate to the obstacles offered to its ascent by its fellow. It is wedged in between the uterus and the rectum, even if there are no adhesions. If we find the above-mentioned irregularity in the uterus and the vagina, and at the same time discover an immovable tumor in the pelvis, which weighs upon the posterior walls of the vagina, and pushes it, together with the uterus, forwards, it may be assumed, if there are no contraindications, that both ovaries are diseased.

The cysts very frequently become the seat of inflammation. This either attacks at different periods the peritoneum, investing the diseased ovary, and causes its adhesions and fixation in the abdominal cavity, or the fibro-serous parietes of the cysts themselves inflame, and the resulting products are deposited upon their internal surface or in their cavity. Thus we find not only all the exudations with their metamorphoses, that occur on the normal serous membranes, at this place, but also all the further effects of this variety of inflammation. Our observations, however, lead us to except the tubercular metamorphosis of the inflammatory product; we, at least, have never met with it, in spite of very extensive and various opportunities.

As the dropsical ovary enlarges, it occupies more and more of the abdominal cavity ; it distends the belly to an enormous extent, pushes the intestine into the inguinal regions, forces the epigastric viscera, to-

gether with the diaphragm, into the thorax, and causes universal emacia-tion, proportionate to the increase of the tumor. The adventitious growth enters into combination with fibroid and carcinomatous products, and especially with medullary cancer, in the manner which we shall have oc-casion to explain further on. It is the less frequently complicated with tubercle, the more it approaches the character of areolar cancer, and the more it compresses the thorax by its increase of size.

There are a few cases on record in which the dropsical ovary is said to have discharged its contents into the Fallopian tube, and thus into the uterus, and externally.

δ. The simple cyst, or the cyst with secondary endogenous formations, also occurs in the shape of cystosarcoma of the ovary; this, however, is much rarer than any of the above-mentioned three varieties, and scarcely ever attains the extreme size to which these are developed.

ε. Finally, we observe that cysts with anomalous contents, viz. en-cysted fatty tumors, occur nowhere so frequently as in the ovary; either, and most commonly, as a simple cyst, or as the composite cyst, in which one of the cysts of the secondary formation is distinguished from the rest by its adipose contents, or, though rarely, in the shape of a compound adi-pose cyst. We often find the fat associated with a formation of hair, fre-quently, too, of teeth, and sometimes with the formation of bone. Like the serous cysts, the adipose cysts are undoubtedly often formed from a Graa-fian vesicle; they occur most frequently in the prime of life, rarely at the period of puberty, and still less frequently in childhood. We have, how-ever, one case of adipose cyst of the ovary in the museum of Vienna, belonging to a child of six years. They grow very slowly, and rarely exceed the size of a child's head. There generally is but one adipose cyst in one of the ovaries; the two are rarely affected at the same time.

The inflammation to which this variety is equally subject with the other cysts, gives rise to a dilatation of the cyst, as well as to an essential alteration in its contents by means of the exudation. It occasionally ter-minates in suppuration, and discharge of the contents externally at the navel, in the hypogastric, or inguinal regions; the contents consist chiefly of pus mixed up with hairs. Under certain circumstances, which will be explained in the sequel, the partially liquefied contents of an adipose cyst assume a peculiar form. In a female, 46 years of age, who died of internal hernia, the right ovary was found converted into an ellipsoid fibrous sac of the size of a man's head, and nine inches in its long diame-ter; it had mounted above the pelvis, and lay obliquely in the left iliac fossa. Its inferior apex was attached to the ovarian ligament; the other, which was directed upwards and outwards, was attached to the anterior surface of the middle portion of the jejunum, by means of a cellular band of an inch in breadth. The sac had been twice turned upon its axis; it contained a brown, fatty, gelatinous fluid, in which, besides a ball of the size of a walnut, composed of hairs that were matted together, there floated seventy-two bodies of the size of a filbert, and a much larger number of smaller bodies of the size of a pea, consisting of a greasy fat. They were of a yellowish color, and from mutual pressure had a polyhe-dral surface, and presented concentric layers. The cyst was not only surrounded by coils of the small intestine, but two portions of intestine

also passed underneath it. It may therefore be said to have represented a capsule, which both from its form and attachment, and from the circumstances of its having been found rotated upon its axis, resembled a dredging-box (granulirbüchse), the rotations of which had converted the contained fat into the globular bodies above described.

b. Anomalous production of fibrous and osseous tissue.

a. Fibrous tissue is formed—

In the shape of fibroid exudation on the internal surface of the simple cysts, but more especially on that of the composite and areolar cysts.

As a subperitoneal (subserous) new growth (so-called cartilaginescence) in the cystic parietes.

As a fibroid tumor ; this rarely attains a larger size than that of a hemp seed or pea. We must except those cases in which the tumor has formed in the parietes of a compound cyst.

As a dirty white or yellow, plicated, curled, soft concretion, within which, not unfrequently, a cavity may be traced. These concretions appear to be Graafian follicles which, after having undergone inflammatory thickening, shrivel up and become obliterated ; after puerperal processes we find them occasionally in the shape of soft, collapsed, friable sacculi, whilst under other circumstances they appear as solid, dense, coriaceous cysts.

As a cicatrix, presenting a rounded, nodulated wheal, with a yellow, rusty, or black nucleus, resulting from follicular apoplexy of the ovary.

β. A formation of bone occurs—

In the shape of so-called ossification (earthy concretion) in the majority of the just-mentioned fibroid growths, and more particularly in the fibroid exudation, and in the subserous fibroid formations of the dropsical ovary.

As genuine bone, in various forms that offer but a weak analogy to one another, and in the adipose cysts.

c. Tubercle.—The occurrence of tubercle in the ovaries is at least doubtful ; so far as our own investigations and observations go, we must deny it altogether.

d. Carcinoma.—Cancer, on the other hand, if we collect all that comes under this denomination, is not unusual.

a. The most frequent form is areolar cancer in the above-described shape of areolar hydrops ovarii ; the conversion of the peripheral follicles of the ovary into large sacs, is a peculiarity which but rarely presents itself in other tissues. We have already alluded to all the important points connected with this subject.

β. Medullary carcinoma is less frequent than the former. There are two varieties. The first occurs in the shape of rounded adventitious growths, varying in size from a goose's egg, to a child's head, and invested with a fibrous sheath ; it sometimes perforates the latter, and grows freely into the peritoneal cavity. In the interior we occasionally find large masses of cellular tissue traversing the substance of the tumors in the shape of septa, and inducing considerable density of the mass ; at other times the entire ovary appears infiltrated with soft encephaloid matter, so as to present fluctuation. The carcinomatous matter is either genuine white cancer, or it contains pigment-cells, which vary in arrangement and number; in the latter case it is brown or black, spotted or

striated, or black throughout (cancer melanodes). It occasionally is combined with the formation of cysts, the latter being either developed on the free surface of the peritoneal sheath of the ovary, or underneath the latter, and in the peripheral layers of the stroma.

This variety occurs in complication with peritoneal cancer, with uterine, mammary, and ventricular cancer, with cancer of the lymphatic glands and the rectum, and universal cancerous deposit. Close adhesions are sometimes formed between it and the adjoining cancerous rectum, so that there is often considerable difficulty in ascertaining which of the two organs is the primary seat of disease. Both ovaries are very often affected.

In the second variety, racemose, fimbriated, fibrous, vascular excrescences, containing a milky or creamy juice, or an encephaloid pulpy mass, form on the internal surface of the peripheral follicles of areolar cancer, or of one of the sacs of the compound cysts, or even upon the internal surface of a small primary cyst. They are often very numerous and attain a considerable length; they become condensed into large masses, and after perforating the parietes of the cyst, sprout through it.

This form is often, though not invariably, coexistent with areolar cancer of the ovary or of other organs.

γ. Fibrous cancer (scirrhus) occurs very rarely in the ovary.

SECT. V.—ABNORMITIES OF THE MAMMARY GLANDS.

§ 1. *Arrest and Excess of Formation.*—Froriep has lately recorded an extremely rare case of absence of one of the mammary glands in a female; the muscles and bones of the corresponding or right side of the thorax were imperfectly developed. The mammæ are found imperfectly developed in those cases in which the sexual apparatus generally is defective, and where certain parts of the latter, or the entire individual, present an hermaphroditic appearance, approaching the male type. An excess of development occurs in various degrees and forms : in the first instance we find an increase in the number of nipples, one gland being provided with two or three ; or there may be supernumerary glands, a third one being placed under one of the normal or between the two breasts. Sometimes the accessory gland is situated externally in the armpit, or there may even be a third, fourth, and fifth, which are arranged symmetrically under the normal breasts, and are always smaller than the latter. We include under this head also, the precocious development of the mammæ in premature puberty, as well as the development occasionally found in the mammæ of man approaching the female character, either with or without an arrest of development in the genital organs. An excess of development is occasionally simulated by the gland being separated into several lobes.

§ 2. *Anomalies of Size.*—In addition to the anomalies spoken of at the end of the preceding section, we here allude to the increase in the size of one or, more commonly, of both breasts, developed spontaneously or after sexual excitement in either sex, or in the female sex after parturition. It consists in a hypertrophy of the gland and of the surrounding fat. The enlargement may attain a most extravagant extent, so as

even to overwhelm the powers of growth in other parts. Hypertrophy of the gland is very often introduced by violent congestion, and accompanied by a secretion of milk.

A diminution or atrophy of the gland, as a morbid process, occurs in the shape of premature involution, both in consequence of the effects of over-nursing, as well as from the sexual functions being completely in abeyance.

§ 3. *Diseases of Tissue.*

1. *Inflammation.*—Inflammation of the mammary gland occurs very rarely, except in consequence of various causes that operate during the puerperal state, and during suckling.

The gland is never attacked throughout, but the inflammation appears at distinct spots of various dimensions, or is, as it were, reduced to them in the course of its progress; and whilst it is here developed with greater intensity, becomes moderated and recedes at all other points. Its symptoms are, besides tumefaction of the gland at the seat of disease, congestion and reddening, by which the natural appearance of the gland is obliterated, and made to resemble flesh: there is also hardness and resistance, with increased density of the parenchyma, which has lost its toughness, and has become friable and lacerable. The gland is infiltrated with a coagulable product, containing more or less reddish serum. Cure may ensue by resolution or absorption of the product, or the process may pass into more or less considerable induration, which at times is very obstinate, or, again, it may terminate in suppuration or abscess of the gland.

Inflammation of the mammary gland not unfrequently coexists with one of the above-described puerperal diseases, though there is no essential relation between the two affections. The resulting abscess is to be carefully distinguished from the deposition of pus, consequent upon its absorption in metrophlebitis.

2. *Cirrhosis of the mammary gland.*—There is a certain condition of the breast which, from all that we know of it, seems comparable to cirrhosis of the liver and the lungs (bronchial dilatation). We have been unable to ascertain whether any particular disease gives rise to it, but there is every reason to suppose that it is the result of protracted suckling.

3. *Adventitious growths.*—A great variety of adventitious growths occur in the mammary gland; some of these are unusual, whilst others are very frequent. They affect mainly the female breast, and only exceptionally, and from very remarkable influences, the male breast.

a. Cysts.—The simple cyst, with serous, albuminous, or colloid contents, as well as the adipose cyst, with or without the formation of hair, is very uncommon; the compound cyst is equally rare. Not so, however,

b. Sarcoma.—This is of common occurrence, and it often assumes the shape of encysted sarcoma, the cysts being either simple, or presenting the endogenous development of secondary cysts; this form is the hydatid tumor or hydatid mammary carcinoma of English writers. All the sarcomatous growths are liable to attain a considerable size, they are frequently recognized as such, and may be extirpated successfully.

c. Fibroid tumors and enchondroma.—These are not frequent ; we have observed the former a few times, but only of small dimensions. Johann Müller has seen one instance of the latter.

d. Tubercle.—According to our own observations, tubercle never occurs in the mammary gland.

e. Cancer.—Cancer, on the other hand, which is found to occur in almost all its varieties, is the more frequent ; mammary and uterine cancer alone suffice to give to the female sex a vast proponderance over the male sex, as to the frequency of cancerous affections. The different forms may occur by themselves, or in combination with one another ; medullary carcinoma is particularly liable to form upon a scirrhous matrix.

a. True scirrhus, or fibrous cancer, and the following variety, are the most common. Scirrhus presents the well-known characters of a cartilaginoid, immovable, nodulated, branched tumor, which draws in the integument, and more particularly the nipple, with its areola, and is imbedded in fat. Its internal structure presents a lobulated appearance, and consists of a whitish fibrous stroma, and of a gray transparent crystalline substance, which is deposited in the interstices of the former. It is often traversed by lacteal ducts which contain a corrugated, whitish, or yellow cheesy matter. The ulcer that it gives rise to is cup-shaped, and presents a hard elevated margin and a sanious discharge ; extends in all directions, but especially backwards, so as to involve the pectoral and intercostal muscles, the periosteum of their ribs and their bony structure, and at last to fix itself immovably in the thoracic parietes. The margin as well as the base of the ulcer degenerate into a red, vascular, bleeding fungus, which is distended by a whitish encephaloid juice ; the immediate consequence is a development of lardaceo-medullary tumors in the most various tissues, either in the vicinity or at a distance. This constitutes—

β. Medullary carcinoma, which however occurs not only in combination with fibrous carcinoma, but also in a primary form ; in the latter case it is equally distinguished by its rapid growth, its large dimensions, by the much more speedy degeneration into universal cancerous cachexia, and by the sponginess of the ulcer.

γ. Cancer hyalinus is much less frequent than either of the former varieties ; it occasionally attains a considerable size, and has, in addition to other peculiarities, a remarkably lobulated structure.

Cancer of the mammary gland is generally developed after the thirty-fifth, though it is sometimes met with before the thirtieth year. It frequently exists by itself, but is more commonly combined with cancer of the adjoining axillary glands, with mediastinal, pleuritic, pulmonic, uterine, hepatic, and cerebral cancer, with universal cancerous cachexia, and with mollities ossium.

SECT. VI.—ABNORMITIES OF THE OVUM.

We shall first discuss the anomalies presented in the attachments of the ovum, i. e. its attachment and development at a point external to the uterine cavity, extra-uterine pregnancy and the degeneration of the ovum. We shall then examine the abnormities occurring in the separate parts

of the ovum, the membranes, the placenta, the funiculus umbilicalis, and the foetus.

§ 1. *Extra-uterine Pregnancy.*—Extra-uterine pregnancy may take place at different points; in the order of frequency these points are, the Fallopian tube, the parietes of the uterus, the ovary, and the vagina. We proceed to state the more important matters connected with each of these occurrences.

1. Pregnancy in the Fallopian tube (graviditas tubaria) is the most frequent of all; the ovum attaches itself either near the fimbriated extremity, or more towards the uterus; this part of the tube becomes dilated into an oval sac, with eccentric development. This variety of pregnancy generally proves fatal in the third or fourth month by hemorrhage into the peritoneal cavity, from rupture of the sac either with or without an escape of the foetus. We have however observed this occurrence in one case at the sixth week, and in another a fortnight after conception. On the other hand, an old preparation existing in the Viennese Museum, appears to prove that pregnancy may continue to the sixth or seventh month. Of six cases of tubal pregnancy preserved in the same collection, five are on the right side.

2. *Pregnancy in the parietes of the uterus.*—This kind of pregnancy, which has also received the name of interstitial pregnancy, and about the seat of which various opinions have been promulgated, is probably nothing more than a pregnancy of the Fallopian tube, i. e. a pregnancy occurring in that portion of the tube which traverses the uterine tissue. It is, consequently, in a close relation with the uterus, and necessarily involves the uterine parenchyma in such a manner that the cavity which contains the foetus with its membranes, appears to have been developed within the tissue of the uterus. It will consequently be most appropriate to consider it as tubo-uterine pregnancy. The pregnant sac consists of uterine tissue, its walls are of considerable thickness, and are in direct connection with the uterus; the sac is more or less distinctly seated at the side of the fundus uteri, and uterine fibres are traceable into it.

This pregnancy generally proves rapidly fatal by rupture; however, it is evident, both from our own observations and those of other morbid anatomists, that it may terminate in a different manner. At various periods of the normal progress of pregnancy, the sac, probably in consequence of the traction exerted upon it, becomes the seat of chronic inflammation, which passes into suppuration and gangrene; after causing the death of the foetus, this either proves fatal by itself or by inducing peritonitis. The sac may suppurate chiefly in one direction, and thus involve the adjoining viscera; so that, after their destruction, it may discharge its contents externally or into the cavities of neighboring organs; this is generally effected slowly and piecemeal. In other cases, pregnancy not only attains, but even extends beyond the full period. The parietes of the sac in this case are of considerable substance and thickness; the sac itself is capacious, and contains a mature or even an over-ripe hypertrophied foetus. In one case of this description the pregnancy lasted sixteen months, and the over-ripe foetus, which had died some time previously, was extracted by opening the abdominal cavity and the sac.

3. Peritoneal pregnancy, graviditis peritonealis, occurs within the cavity of the peritoneum; the fœtus with its membranes is surrounded by an organic exudation attached to the peritoneum, and the placenta is found connected with the most different parts of the parietal and visceral laminæ of the peritoneum. This form of pregnancy generally terminates fatally at various periods by peritoneal inflammation; sometimes the peritonitis ends in suppuration, which may cause the effete fœtus, together with the formative organs, to be discharged by various passages.

4. Ovarian pregnancy is the most unfrequent form of the extra-uterine pregnancy; it either terminates in laceration, which is at once fatal, or in inflammation and suppuration of the sac, which in their turn prove fatal, or cause a diminution of the fœtus.

5. Vaginal pregnancy is not only the rarest of all, but altogether problematical.

Although we have given an anatomical account of the chief terminations occurring in extra-uterine pregnancy, we will add a comparative analysis, and also subjoin one that we have not yet mentioned.

Interstitial, ovarian, and tubal pregnancy terminate by laceration; the last two at an early period, the first much later, and even after the usual duration of pregnancy.

All the varieties of extra-uterine pregnancy may terminate in inflammation with consequent suppuration, owing to the decomposition of the defunct fœtus, and the putrescence of its involucra. Inflammation may, in advanced interstitial pregnancy, doubtless arise in the sac primarily, from the traction exerted upon its tissue and upon the peritoneal investment, and thus induce the death of the fœtus. If the suppurative process attacks the organs adjoining and adherent to the sac, the latter may discharge itself externally through the abdominal parietes or into the cavity of an adjoining organ; the fœtus may thus be eliminated entire or piecemeal, in a state of maceration, saponification, or putrefaction. This has been observed repeatedly; single bones belonging to the fœtus having been seen to pass through the navel, at different parts of the hypogastric region, and still more frequently by the anus or the urinary passages. It is stated that, after this has taken place, a cure may follow; but death from exhaustion is a more common consequence. The discharge may occur long after the usual period of pregnancy has terminated.

Besides the above-mentioned modes of termination there is another, which must be viewed as the most favorable one; in this case the fœtus dies before or after it has attained maturity, and, after the surrounding fluids have been removed by absorption, becomes mummefied and indurated; or if the soft tissues have already undergone a certain degree of decomposition, it may become incrusted by a greasy, chalky substance (lithopædion), and in this shape be borne for a long time in the contracted sac, without much inconvenience. This termination occurs chiefly in peritoneal pregnancy, it has however also been observed in pregnancy in the Fallopian tube.

In all extra-uterine pregnancies we commonly observe the formation of a deciduous membrane on the inner surface of the uterus; and the latter undergoes, up to a certain period, the same changes that it

would present if it contained the fœtus. It scarcely ever exceeds the dimensions which it exhibits about the first and second month of normal pregnancy. This development of the uterus is undoubtedly more considerable in interstitial and tubal pregnancy, than in the other varieties of extra-uterine pregnancy, and may be considered as analogous to the development of the unimpregnated half of the uterus bicornis or bilocularis.

§ 2. *Degeneration of the Ovum.*—We here but allude to those cases of degeneration of the ovum which are called moles, and which are distinguished according to their external appearance, structure, and density as carneous, vesicular moles, &c. We exclude all abnormities in the formation of the fœtus, and remark that moles generally contain no fœtus, as the degeneration of the membranes and the placenta mostly occurs at a very early period of pregnancy, in consequence of which the embryo dies and disappears, the cavity of the amnios remaining persistent or becoming obliterated. The diseases in which the moles of which we speak originate, will be adverted to in the following pages, and we shall take care to point out the connection.

§ 3. *Abnormities of the separate parts of the Ovum.*

1. *Abnormities of the Membranes and of the Liquor Amnii.*—The membranes of the ovum may undoubtedly become the seat of hemorrhage and inflammation at a very early period of pregnancy; these affections are probably the cause of the formation of moles, but they may also give rise to abortion. The dirty white and ash-colored spots occurring in various sizes upon the amnion, and accompanied by thickening of the membranes, and the opacity sometimes affecting the greater part of the amnion, evidence previous inflammation. But under this head we must more particularly instance the deposits of a grayish-red, whitish, or yellowish substance, occurring on the chorion and the decidua, in the shape of patches. The membranes here present a thickening without distinct edges, or flattened rounded nodules; the tissues being either softened and friable, or indurated and dense. Occasionally the membranes, in consequence of a cretification of the deposit, are incrusted, or contain a chalky grit.

The albuminoid layer which represents the so-called tunica media, is not unfrequently morbidly increased in quantity; sometimes a thin, brawny, or watery accumulation is substituted for it, and to the latter the discharge of the so-called spurious liquor amnii in the second half of pregnancy, is probably due (hydrallantois). In other cases its amount is very small, or it presents greater consistency, and may even be tough and membranous.

The villi of the chorion degenerate at an early period of pregnancy, into pedunculated vesicles or cysts, which are placed at their extremities; a degeneration which is also seen in the placenta; when occurring in an extensive and advanced degree they represent what has been termed the vesicular or hydatid mole.

Occasionally we discover more or less extensive adhesions between the embryo and the amnion, which is a circumstance of some importance, on account of the impediment it offers to the proper development of the fœtus.

The liquor amnii is sometimes so much reduced in quantity, that the fœtus is closely invested by the amnion, at others it is excessive (hydramnios); it is also found more or less opaque, discolored, and fetid.

2. *Abnormities of the Placenta.*—*a.* The placenta offers considerable variations as to size, without being morbidly affected. We have here only to mention that extreme development of the intervascular substance of the decidua which compresses the vessels; as well as the occurrence of atrophy with relaxation, a contraction accompanied by coriaceous toughness, the causes and original seat of which doubtless vary, but with the exception of inflammation are unknown.

β. The placenta presents no remarkable pathological changes in point of form, with the exception of its division into a few large or numerous smaller lobules. The various shapes presented by the placenta offer no interest in reference to pathology.

γ. The placenta presents numerous deviations in regard to position; its position at the inferior segment of the uterus deserves particular mention, as its eccentric or concentric development at the os uteri induces considerable perils towards the end of pregnancy, by giving rise to frequent and lasting hemorrhage.

δ. Allied to this hemorrhage are the floodings which, though the placenta occupies its normal position, are brought on by concussion or contusion of the pregnant womb, inducing a forcible separation of the placenta from the uterus, or a laceration of the former, or which are the result of rupture of the uterine vessels of the placenta caused by extreme congestion. Whether or not accompanied by external bleeding, they represent apoplexy of the placenta, one of the most frequent causes of abortion; the blood may be diffused through the parenchyma to a greater or less extent, or be extravasated and accumulated at one spot, which may either adjoin the uterine parietes or be situated deeply within the placental tissue. The placenta is undoubtedly sometimes affected with plethora, congestion, and diminution in the rapidity of the circulation, a condition which is important on account of the impeded vivification of the fœtal blood. It is then of a darker color, externally and internally of a dark violet or livid hue, the vessels are overcharged, the entire organ appears enlarged and heavier, and feels harder and denser.

ε. Among the textural diseases inflammation is the most frequent; it generally occurs in the lobular form, as it attacks small sections or lobules, and rarely larger portions of the placenta. Still a considerable portion may be simultaneously or successively attacked even in the first instance, inasmuch as the products of the process are found scattered over numerous spots. It recurs at different or new points to the end of pregnancy; an opportunity is thus presented of observing it, if not in its first stage, at least shortly after the formation of the product, and of tracing the metamorphoses of the latter.

Inflammation of the placenta generally gives rise to a plastic fibrinous deposit, which is reddened by the coloring matter of the blood which it contains, and by which the diseased portion is rendered denser and more lacerable. This may be termed hepatization of the placenta; it may be recognized by the increased resistance and nodulated tumefaction pre-

sented to the touch.   In the course of time the deposit assumes a pale red, grayish or yellowish-red, or even yellowish-white tinge ; at the same time it becomes firmer, and together with the included obliterated tissue, contracts and shrivels.   The inflammation has thus terminated as it usually does, in induration and obliteration of the placental tissue, which is converted into an ashy, tough, leathery callus, resembling elastic tissue.

It appears an established fact that an adhesion may form between the placenta and the uterus in consequence of a process of this kind ; the extent as well as the intimacy of the union naturally varies.

In rare cases the inflammation may give rise to a purulent product, causing suppuration of the placenta in the shape of circumscribed globular abscesses, or of diffused infiltration and fusion of the placental tissue —phthisis placentæ.

The question as to the seat of the inflammatory process, or rather as to which of the vascular systems is affected, has not as yet been answered ; doubtless either of the two may be involved, and it is to be presumed that in both cases the results will be the same, on account of the intimate connection existing between them.

Inflammation and consequent obliteration of the placenta are the more likely to induce imperfect nutrition, and consequent tabes of the fœtus, the more they are diffused and the greater the number of placental sections attacked at the same time.

The fœtal portion of the placenta, as we have already mentioned when speaking of the villi of the chorion, is not unfrequently attacked by a degeneration in the shape of round or oval, fusiform, pedunculated, serous vesicles or hydatids, which diminish, and even entirely obliterate the cavity of the amnion.   This is a vesicular or hydatid mole, Laennec's acephalocystis racemosa.

Foreign observers have given instances of osseous deposits in, or ossification of the placenta ; they are gibbous, nodulated, or cordate formations, which are probably developed in the placental tissue after it has been obliterated by inflammation, or in the fibrinous coagula caused by hemorrhages.

Tubercle does not occur in the placenta ; one must be careful not to confound the product of inflammation, which sometimes has a cheesy, friable, and chalky appearance, or a fibrinous coagulum caused by hemorrhage, which is undergoing a similar metamorphosis, with placental tubercle.

The observations recorded of scirrhus, or scirrhous degeneration of the placenta, may justly be considered as erroneous ; the cases described as such, are obliterations of the placental tissue after inflammation, indurated inflammatory products, or old, shrivelled, decolorized extravasations of blood, &c.   Adhesions similar to those which we mentioned when speaking of the membranes, are found to occur between the placenta and the fœtus.

3. *Abnormities of the Umbilical Cord.*—The instances of absence of the funiculus umbilicalis, recorded by ancient writers, must evidently, as Meckel has pointed out, be considered as cases of extreme shortness of the cord.   The subject is of considerable importance on account of its

influence upon arrest of development, particularly of the inferior half of the fœtus.

The cord varies considerably in length. Davis has seen one of two, Montault of four, Meissner and myself of five inches only; whilst on the other hand, Baudelocque has noticed one of forty-six, and Heriter one of fifty-seven inches. In the Viennese Museum there is a cord attached to a large placenta, which measures fifty-four inches; and if we allow five or six inches for the distance at which it was probably severed from the navel, the whole cord must have been from fifty-nine to sixty inches in length.

The chief deviations as to the insertion of the cord are the marginal attachment, and the insertion external to the placenta into the membranes. Occasionally we notice a premature separation of the vessels, and of the two arteries; one not unfrequently is absent, and the other is then commonly a direct continuation of the abdominal aorta.

The true knots of the umbilical cord that occur in rare cases, are of importance, as they may occasion obstacles to, or a cessation of, the circulation, in consequence of the traction exerted upon them during parturition.

Irregularities in the position of the umbilical cord very frequently present themselves in the shape of circumvolutions round different parts of the fœtus; if traction is exerted upon them, the circulation may be impeded in the cord, as well as in the part of the fœtus which they surround.

Rupture of the umbilical cord, or of its component vessels, and especially of the vein, are of very rare occurrence. They result from extreme traction, when the cord is either too short or twisted.

Adhesions have been noticed in rare cases between the funiculus umbilicalis, and the fœtus and the membranes.

The gelatinous matter of the cord is either excessive, and the latter then appears much enlarged; or it is diminished in quantity, in which case the cord is thin, flabby, and corrugated.

The umbilical vein presents varicose dilatations and contractions, the latter particularly in the vicinity of the navel. The sheath of the cord occasionally contains serous cysts.

4. *Abnormities of the Fœtus.*—We pass over those deviations of congenital development which we have already discussed, and devote this section to the consideration of the remaining anomalies, although we have already cursorily touched upon most of them.

We not unfrequently meet with a hypertrophy of the fœtus, or of individual organs and sections of the fœtal body. Children are sometimes born, either at the proper period or later, of excessive dimensions, over-nourished and endowed with all the characters of over-ripeness. Among the partial hypertrophies we have to specify those of the brain, of the thyroid and thymus glands, the hypertrophy of certain parts of the skeleton, as the premature development of the cranium, the excess in the digital phalanges of the hands or feet, the so-called hypertrophies of the liver and the spleen.

The entire fœtus may be atrophic, in consequence of the cachectic state of the mother; but those cases are of greater importance which result from disease of the membranes, the placenta, and the cord; and if

occurring at the earliest period of embryonic life, may cause the embryo to disappear entirely, or so far as to leave but mere traces. Partial atrophy, as a result of disease in the nervous centres, occasionally affects the extremities; in rare cases, it may be the consequence of pressure exerted by the uterus on the funiculus umbilicalis.

The most various curvatures and malpositions of the bones (pes equinus), curvatures and dislocations of the joints, may be brought on by contraction of the uterine cavity, external pressure, convulsions, and tonic spasms of the fœtus. Convulsions or traumatic injuries may induce fractures of the bones, and even rupture of the abdominal viscera, and especially of the liver and the intestines.

To these lesions of continuity we have to add the spontaneous amputations of extremities (Simpson, Montgomery), occurring at an early stage of embryonic life; they have not as yet been sufficiently accounted for, and are to be carefully distinguished from cases of arrest of development.

The fœtus may be affected by general plethora, as well as by congestion of separate organs, which ultimately degenerates into hemorrhage and apoplexy. The brain and spinal marrow and their membranes, the thyroid gland, the liver, the kidneys, and the suprarenal capsules are particularly liable to be attacked.

A most important morbid process and one of very frequent occurrence, the peculiarities of which in the fœtus are almost unknown, is inflammation. Its terminations and consequences are of great value in regard to the doctrine of deviations of formations, for a reconstruction of which we are particularly indebted to Simpson, whose views on the subject are of peculiar interest. Inflammation attacks almost all the organs, including those peculiar to fœtal life, the thymus, and the suprarenal capsules. Its terminations and consequences the more resemble those which it presents in the infant and adult, the more mature the fœtus, the more developed the tissues attacked, are. It gives rise to plastic products with adhesions between adjoining organs, accompanied by the most various new formations; it induces suppuration and suppurative destruction, though very rarely induration. On the other hand, it causes in very delicate embryos, and in particular tissues of peculiar delicacy, a very rapid dissolution, or liquefaction, either of a benignant or a malignant character.

Dropsy of the different cavities and anasarca, have been noticed in the fœtus; among the former, hydrocephalus and hydrorrhachis are both in themselves, and on account of the consequent arrest of development and the malformation of the cranium and of other parts, of chief importance. Cutaneous dropsy sometimes attacks the fœtus in a very eminent degree, and is generally combined with dropsical accumulations in the large cavities. It has been particularly and repeatedly observed in the children born of women who had themselves suffered from dropsy during pregnancy (West); it also occurs under other circumstances, and in these cases probably originates in obstacles to the circulation through the umbilical vessels and the fœtal portion of the placenta.

Even adventitious growths occur, though rarely, in the fœtus; we occasionally discover cysts, particularly the simple cyst, lipomatous, sarcomatous, and even cancerous products. Although many of the cases that

have been considered as cancerous, rest upon a misapprehension, the occurrence of carcinoma is undoubted and of peculiar interest. Fœtal cancer is allied to the cancer, and especially to the medullary variety, occurring during infancy ; we communicate the following case that we have ourselves observed, as one of peculiar interest.

A female infant, of 16½ inches in length, and 27 days old, presented in the region of the pudenda, anus, and sacrum, a tumor tensely invested by the cutaneous covering. It was of the size of a goose's egg, and had, towards the sacral and lower lumbar vertebræ, a conoid process of the size of a hazel-nut. On its left side were noticed the vaginal and anal opening, which were pushed downwards and separated, the latter appearing in the shape of an excoriated semilunar fissure. The child was born with this tumor. It lay external to the pelvis, under the skin and the perineal muscles ; posteriorly, fibres of the glutæus magnus spread over it. It was surrounded by a fibrous sheath, and consisted of various tissues ; the inferior third, and the portion that lay more to the right, resembled the reddish-gray cerebral substance of an infant's brain, whereas the two upper thirds presented a follicular tissue with small loculi, containing a grayish gelatine. The tumor extended into the pelvis by the inferior aperture, in the shape of an oval serous cyst of the size of a walnut, the internal surface of which appeared here and there to contain a black pigment, and presented a few ridge-like duplicatures. The follicular portion of the morbid product also forced its way through the sacral fissure into the medullary canal, and then presented a process closely resembling the external conical process, except that it was smaller.

This adventitious product is nothing more than a combination of areolar and medullary cancer, the circumference of which is converted into a cyst.

Tubercle occurs but in very rare cases in the fœtus ; we ourselves have never observed it.

The fœtal fluids are undoubtedly liable to numerous acute and chronic morbid affections, as is evidenced by the occurrence of various exanthematic and impetiginous diseases, as well as cachectic disorders, such as rickets, syphilis, tubercle, peculiar hypertrophies of the liver, the spleen, the lymphatic glands.

Variola, measles, and various cicatrices have been noticed on the fœtal integuments. To this head also pertains pemphigus and various vesicular eruptions, the vesicles of which contain a livid, sero-purulent fluid, are converted into ulcers, and may be traced to syphilitic causes. We also find ecchymoses, petechial suffusions of the skin, shallow or elevated nævi, of a brown or livid hue, and of different sizes. The subcutaneous cellular tissue is the seat of anasarca and of many of the above-mentioned tumors and morbid growths ; shortly after birth, it is frequently attacked with induration.

The serous membranes are found more frequently inflamed than any other tissue, or the previous existence of inflammation in them is evidenced by adhesions of the organs which they invest ; these, undoubtedly, give rise to many of the anomalies in the position of these organs, which are in part at least looked upon as the consequence of original malformation and arrest of development.

Peritoneal inflammation is, doubtless, the most frequent, and it is upon

this fact and upon the adhesive termination of the disease, that Simpson bases his views regarding many of the anomalies that are commonly considered as cases of arrest of development. Inflammation of the pleura and pericardium are less frequent.

Among the mucous membranes, that of the alimentary tube is the chief seat of disease, as we shall have occasion to explain more fully in the sequel.

In addition to the above-mentioned fractures, dislocations, and spontaneous amputations, we find the osseous system liable to suppurative inflammation (caries), hyperostosis, and an exuberant deposit of callus, an arrest in the process of ossification, which is allied to rhachitism (rhachitis congenita).

The morbid processes in the muscular system which we chiefly meet with, are contractions; they depend mainly upon diseases of the nervous centres.

The heart is liable to be affected, in the first instance, by pericarditis; in the second, by endocarditis. It is very remarkable, that the relation of the latter to the cavities of the heart is the reverse of what occurs after birth. The dilatations and valvular affections observed at the left ventricle and its arterial orifice in the adult, as a consequence of endocarditis, are here found to attack the right ventricle and the valves of the pulmonary artery.

The stenoses observed in the latter, which originate in a morbid metamorphosis of the valves, are to be carefully distinguished from cases of arrest of development occurring here, especially in the shape of atrophy of the pulmonary artery, resulting from anomalies in the structure of the heart.

The ductus Botalli is in rare cases liable to an aneurismatic dilatation, and in this respect resembles the aorta in extra-uterine life.

The brain and the spinal cord are particularly subject to disease in the foetus; and these affections are undoubtedly the cause of many defects and malformations of the brain and spinal cord, their membranous sheaths and osseous cases, and of defect and malformation of other organs which have hitherto been considered as anomalies of original development.

Thus we observe hypertrophy of the brain, which in rare cases attains such an extent as to cause the development of the cranium to appear entirely arrested.

Apoplexy occurs very rarely as hemorrhage within the substance of the brain; but we often find both in the foetus and the new-born infant a vascular apoplexy, and extravasation into the tissue of the membranes and into the cavity of the arachnoid.

Inflammation and its consequences, inflammatory softening and complete liquefaction of the brain, are much more frequent. These and hydrocephalus are doubtless the commonest cerebral diseases of the foetus, and upon the former the defects and numerous malformations depend which are found in hydrocephalic foetuses.

Hydrocephalus and hydrorrhachis are, in the foetus as at a later period, the result of repeated exudative processes affecting the investments of the cerebral cavities and the spinal canal. They are well known often to attain such a degree, that not only the dilated and imperfectly ossified

cranium offers an obstacle to parturition, but that the brain and the spinal cord are gradually destroyed by compression ; that they are ruptured at an early stage of embryonic life, or are dislocated in various directions, and forced out of the cranium at later periods.

In the respiratory apparatus of the fœtus we find that the pulmonary parenchyma and the bronchi occasionally become diseased. The former is said to have been found in a state of hepatization, and even abscesses have been seen in the lungs. The bronchi are frequently charged with mucus, and atelectasis neonatorum is probably caused by a mere catarrhal affection of the mucous membrane, and an obstruction of the capillary bronchi by mucus. (Vid. Vol. III., Acute Catarrh of Respir. Org.)

The thymus gland of the fœtus may, according to Veron, be attacked by inflammation and suppuration.

In the digestive apparatus, the peritoneum, the entire intestine, and its appendages may become diseased during uterine existence.

The peritoneum is frequently the seat of inflammation of an acute or chronic character, causing exudations, that vary in quantity and quality. It may be limited to one portion, or be universal. It not only induces thickening of the peritoneum, but also adhesions among the abdominal viscera, and between them and the parietes ; the sooner it sets in, the more it is likely to operate as the cause of numerous anomalies in the abdomen, which have been hitherto considered as cases of arrest of development. (Simpson.) The inflammation may originate in unknown causes, or in such as are anatomically demonstrable, as constriction of the intestine, hemorrhage of the liver into the peritoneal cavity, extravasation of the contents of the intestines, or of urine ; the latter may occur at a very early period, for it has been noticed in a fœtus of four months. It sometimes kills the fœtus before maturity, at others death ensues shortly after birth.

As regards the alimentary tube, both hyperæmia of its mucous membrane and anæmia, with waxy paleness and softening, have been frequently observed. The latter affection is of particlar importance in the fœtus. The former not unfrequently attains such a degree as to warrant the application of the term apoplexy ; it is generally associated with hyperæmia of other abdominal viscera and general plethora, and may be accompanied by ecchymoses in the tissues and extravasation of blood into the intestinal cavity.

Inflammation and the allied processes are generally limited to the follicular apparatus of the ventricular and intestinal mucous membrane, which are comparatively very much developed. The former occasionally presents a hemorrhagic fusion (erosion) of the follicles in a very marked degree ; the follicular apparatus of the intestinal mucous membrane is still more frequently diseased. In the small intestine, the glands of Peyer are chiefly found more or less swollen, reddened, and of a fleshy sarcomatous appearance, or pale, i. e. yellow or grayish-red, containing like the solitary follicles, a variety of reddish or grayish, more or less dense, opaque, milky or curdled, serous, flocculent fluids. These morbid enlargements of the intestinal follicles, caused by tumefaction and imbibition of the tissues, and the presence of a variously modified product,

are here too undoubtedly (vide p. 78) closely associated with anomalies in the vital fluids, with morbid conditions of the mesenteric glands, abnormal enlargement of the thymus, or with tumefaction of the spleen, but their real nature remains an enigma. The indurated swellings of the Peyerian glands occasionally resemble closely the typhoid infiltration found in adults, and may indeed result, if not from an identical, at least from a very similar process. Follicular tumefaction also occurs frequently in the large intestine of the fœtus and the new-born infant ; and, as in the adult, without any coexistent affection of the small intestine. The follicles appear much reddened, the entire mucous membrane is swollen, and invested with a yellowish-white secretion.

In very rare cases we meet with a diffused croupy inflammation, and a corresponding product in the intestinal mucous membrane.

The processes which we have just considered, do not appear to lead to ulceration in the fœtus ; still there are cases on record of extensive ulcerative affections of the œsophagus and the entire alimentary tract.

Tumors and excrescences have been observed on the internal surface of the intestine, as well as callosities of the interstitial cellular tissue, especially of the stomach.

The fœtal liver is very often the seat of hyperæmia, which, on account of the delicacy of the hepatic tissue, easily degenerates into apoplexy with rupture.

Various infiltrations of the hepatic parenchyma may form even during fœta existence, and we thus find the fatty, the waxy, the lardaceous liver accompanied by the characteristic enlargement, in which the left lobe, which at that period is comparatively of large dimensions, necessarily participates.

In the same manner, the spleen of the fœtus may suffer ; it is often found in a state of acute or of chronic tumefaction, presenting in the first case looseness, in the second, remarkable condensation, resistency, and frangibility of texture. Tumors of the spleen bear the same import as in the adult, and result from the relation existing between the spleen and dyscrasic conditions of the blood. They occasionally attain a considerable size.

The salivary glands, and especially the pancreas, are very rarely affected in the fœtus ; the cancerous induration of the pancreas noticed at page 140 must be considered as extremely remarkable.

The urinary organs of the fœtus do not frequently become diseased, but their affections occasionally attain a very considerable development. The kidneys are subject to hyperæmia and apoplexy ; the passages have been found excessively dilated, to an extent varying with the seat of contraction or obturation ; and the bladder has even been ruptured from the same cause. We quote the following case, taken from a preparation which is preserved in the Viennese Museum, both because it presents analogies to the various observations of this character that have been recorded, and on account of several peculiarities which it offers : A newborn male infant with a large abdominal cavity, is provided with a bladder of the size of a child's head, collapsed and agglutinated to the abdominal parietes by a thick exudation. The membranes are traversed by varicose veins, which presented a blue color when fresh, and the urethra

is narrow, though patent.   The inferior portions of the ureters are di-
lated to the size of the small intestine of an adult, the upper portions less
so, the right being of the size of a little finger, the left one larger; the
pelves and calices of the kidneys, which are of the size of walnuts, are
moderately dilated.   The ureters are coiled up and bent, and their
curvatures are bound down by a tense cellular sheath: both terminate
blindly at the sides of the bladder.   The right one is accompanied by an
unsymmetrical umbilical artery, which proceeds from the point of origin
of the renal arteries.   All the other abdominal viscera are pushed up-
wards, and the entire intestine is very narrow, scarcely presenting a
diameter of one line; the abdominal integuments are œdematous, and are
superficially ulcerated, below the insertion of the umbilical cord, to an
extent of three inches by nine lines or one inch.   They are considerably
thinned, and of an irregular thickness, presenting a cribriform perfora-
tion at one spot, and adhering to the anterior surface of the bladder.
The peritoneum was invested by a plastic exudation, and contained six
ounces of a yellow, opaque fluid.

The cysts of which we have spoken at page 159, also occur in the fœtal
kidneys, and may be so numerous that the latter appear converted into
one mass of cysts.

We frequently have opportunities of observing spots in the fœtal kid-
neys which are variously discolored, and which present a morbid indu-
ration of the tissue.   They most probably result from inflammation, and
are occasionally associated with traces of a similar process in the adipose
sheath of the kidneys.

The suprarenal capsules have been found in a state of suppuration.

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