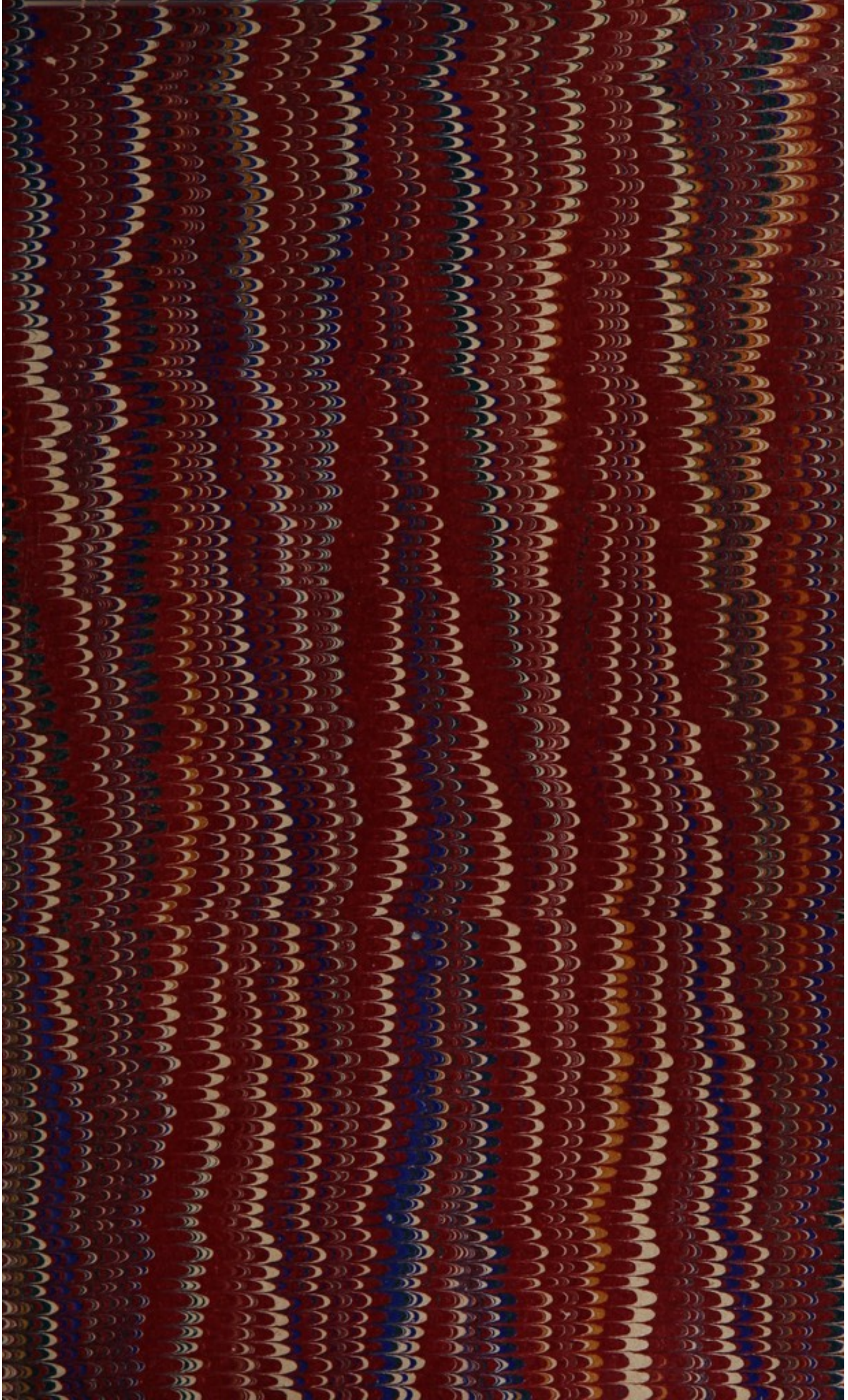
A treatise on pathological anatomy / by Carl Rokitansky ; translated from the German, with additions of diagnosis from Schoenlein, Skoda, and others by John C. Peters.



A TREATISE

ON

PATHOLOGICAL ANATOMY:

BY

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PART I.

CONTAINING:

THE ABNORMAL CONDITIONS OF THE ORGANS OF RESPIRATION.

TRANSLATED FROM THE GERMAN, WITH ADDITIONS ON DIAGNOSIS FROM SCHOENLEIN, SKODA, AND OTHERS.

BY

DR. JOHN C. PETERS.

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TO

ALEXANDER S. WOTHERSPOON, M. D.

ASSISTANT SURGEON U. S. ARMY, AND LATE RESIDENT-PHYSICIAN TO THE NEW-YORK HOSPITAL.

THIS TRANSLATION

Is respectfully Dedicated,

AS A TRIBUTE TO HIS ACQUIREMENTS IN PATHOLOGICAL SCIENCE,

AND A TESTIMONY OF THE TRANSLATOR'S NUMEROUS OBLIGATIONS,

BY HIS FRIEND,

J. C. PETERS.

TRANSLATOR'S PREFACE.

It has long been admitted that the great impulse which has lately been given to the studies of anatomy, physiology, microscopy, and pathological chemistry, had its origin in Germany; faint rumors, too, have reached this country of equally great advances made in pathology and pathological anatomy, and many expectations have been excited as to the nature of the rich harvest said to have been reaped by the proverbially profound and studious Germans. It is already considered a disgrace for an American physician to be unacquainted with the labors of the French, English, and Irish pathologists, and, as the Germans have now outstripped their neighbors, the time is not far distant when the same stigma will rest on those who are ignorant of German pathological literature. Hence, we think that we need make but few excuses for undertaking the translation of any reputable German work on morbid anatomy, and are still less inclined to make any for selecting that of Rokitansky; but we would fain beg the indulgence of our readers for the manner in which our task has been accomplished, as it was no easy one. "Rokitansky's book is what it professes to be: it is morbid anatomy in its densest and most compact form, scarcely ever alleviated by cases, histories, or hypotheses; it is just such a work as might be expected from its author, who is said to have written in it the results of his experience gained in the careful examination of over 12,000 bodies, and who is possessed of a truly marvellous power of observing and amassing facts;"* but his style of writing is often rough, and even ungrammatical, although his difficult and uncouth language is ever forcible and explicit, and

* See British and Foreign Med. Rev. Jan. 1843, p. 84.

expresses his meaning with all the accuracy and perspicuity which half of the European tongues compressed into one are capable. Hence, we are scarcely ashamed to own that the present is the fourth version which we have made; twice we essayed the task unaided, and although we had read the work repeatedly and carefully, and were familiar with Rokitansky's style, from hearing his lectures and enjoying familiar private intercourse with him, we could not succeed in making even a passable translation. We had thrown up the task in despair, although our admiration and friendship for Rokitansky had drawn us to it as to a "labor of love," and we had received not only his permission but his request to undertake it, when Dr. Wotherspoon volunteered his assistance and almost forced us to the third trial. Even the result of our joint labors did not meet a very low ideal of what the work ought to be, and hence we not only revised and compared it with the original text, but re-wrote the whole. We pledge ourselves for the faithfulness of the translation, and although Dr. Wotherspoon took considerable pains to render the style more English, it will still be found foreign and rude in many respects; as it is, we flatter ourselves that it will bear a comparison with the translation of parts of it, given in the January, 1843, No. of the British and Foreign Medical Review, the editor of which, it is well known, is a great stickler for purity and accuracy of style.

We beg also distinctly to state that all the notes on diagnosis are mere compilations, in which we have not only used the materials of the authors whose names will be found appended to each, but also their language; we are even constrained to apologize for the mutilation of the latter, which want of space often forced us to make at the expense of elegance. The names of Skoda and Schoenlein are here introduced for the first time to the American medical public, as able diagnosticians; the latter enjoys the reputation of being the greatest master of general diagnosis in Germany, and the former as the most original and accurate auscultator and percussor. It is a sufficient pledge of their ability, to state that the majority of the novel and important addi-

tions to diagnosis made by Graves, Stokes, and many others, may be traced to these two sources. Occasional notices have been made of Rokitansky's work in English, Irish, and American journals; and as most reviewers think themselves called upon to make an exhibit of the superior knowledge which they possess upon subjects to which others have devoted the undivided attention of a whole lifetime, it is not to be expected that Rokitansky should escape unscathed, especially from those who have never seen his work, and who are farthest removed from the field of his labors. The editor of the Dublin Medical Journal (see July No. for 1843) ventures a wholesale condemnation of Rokitansky's method of studying and teaching pathological anatomy, and that after merely reading a short but favorable account of the Vienna School of Medicine in Wilde's Austria; he says, "The readers of this [the Dublin] journal, who have attended the instructive meetings of the Dublin Pathological Society, will have no hesitation in coinciding with us in opinion that the plan of teaching pursued by Rokitansky is little better than useless. He must have observed, that the cases brought forward at that Society, unaccompanied by an accurate detail of symptoms, and faithful records of the effects of treatment, were little better than worthless; as specimens of diseased structure they were interesting, no doubt, to the mere morbid anatomist, but any of our museums conld have furnished, perhaps, infinitely better examples of the same alteration of structure. It is not in this way that the science of medicine can be advanced; that object can only be obtained by placing on record an accurate account of the symptoms present during the life of the patient, the result of treatment, and the morbid alterations disclosed on dissection—this is the true method of studying pathology, and it is for this reason that the works of Andral, Cruvelhier, and others, who have pursued the same path of investigation, have succeeded in leading to improvements in the practice of medicine."

We cheerfully admit that the above would be serious and fair objections, if they were true; but all these things and more are done in Vienna, but not by the same laborer. It is well known

to every one who has studied in the huge "General Vienna Hospital," that there are clinical professors there, who have successively risen from the post of resident physician to that of *secundarius*, thence to that of *primarius*, and professor extraordinary, and finally to professor in ordinary; that there are aged resident physicians who have been elected by "Concours," and if found worthy are sure of professorships; that medical students are not allowed to walk the hospitals before they have studied three years, and that then each is put in charge of a patient, under the supervision of the resident physician and clinical professor; that an accurate record, containing the history of each patient, with the variations of his symptoms from day to day, and the effects of treatment, is kept hanging over the bed of each patient; that in case of the death of the patient, the resident physician and the pupil who had him in charge, and, on all important occasions, also the clinical professor, and his whole class, are in attendance at the dead-house on the following morning; that when the name and number of the patient is called, these take their station at the post-mortem table, and the autopsy is performed by Rokitansky or one of his assistants; that a detail of the post-mortem appearances is dictated in a loud voice to a secretary, who writes them down on sheets similar in size and shape to the papers on which the Hospital records are kept; that when the examination is finished, the clinical professor or the resident physician is called upon for a short *resumé* of the case; that, finally, the record of the post-mortem and of the history of the case is placed among the archives of the pathological museum. It is well to add, that Rokitansky was first educated as a hospital physician; next, he was trained up in the dead-house in the celebrated Sifft, and finally was elected to the professorship of pathological anatomy; that all medico-legal, or so-called coroner's cases, are submitted to him for examination, so that life and death hang upon his verdict almost every day, and he is forced to acquire a most extended knowledge of practical medicine, which is the more necessary to him as he is daily called upon to verify or condemn the diagnoses of numerous clinical professors. Such is the

training to which his naturally researchful and truthful impulses have been subjected through a long series of years. Right honestly and modestly does he perform his delicate duties; ever inclining to the side of mercy, he is firm enough to add the weight of his testimony to that which has been accumulated against a trembling or hardened criminal, and never is found weak enough or mean enough to gloss over the mistakes or flatter the vanity of hospital physicians. In order to fully appreciate Rokitansky, he should be seen and heard when a discussion ensues between him and the clinical professors; at such moments the fruits of his extensive knowledge, gained by long service in a hospital where nearly thirty thousand patients are treated yearly, by nearly twenty years' service in the dead-house, and by a most extended and accurate course of reading, are brought to bear upon the case in question, in a rapid and condensed, but graphic and truthful manner. These ever-occurring discussions would alone prevent his sinking into a mere morbid anatomist, for daily he is thrown in collision with learned clinical professors, and their opinions of the origin, nature, progress, and proper treatment of disease are constantly dinned into his ears, and are as frequently contrasted with Andral's, Louis's, Cruvelhier's, and other morbid anatomists, and his own deductions drawn from the cadaveric appearances, and quickly he is transformed into a learned general pathologist. Impartial reviewers should be cautious how they hazard a lance against the victor in a thousand conflicts.

It is interesting to inquire what effects are produced upon the clinical professors and resident physicians: daily they appear in the presence of their classes, before a reserved but kindly man—before an experienced and truthful pathological anatomist, as it were, to render an account of their convictions as to the nature and treatment of disease. Frequently we have seen the cheeks of proud clinical professors and vain resident physicians mantled with shame, when an impartial and careful post-mortem has revealed their errors in diagnosis and treatment; but still more frequently we have seen the accurate predictions of Skoda, Helm, Schuh, &c., &c., confirmed and received with plaudits by the assembled host of students and foreign physicians, who have

watched the case during life, and now witness the examination after death. The consequence is, that those professors who are too proud to learn never show themselves in the dead-house, their cliniques are deserted, and government waits but for a plausible pretext to depose them from their rank; but the zealous and honest become doubly anxious to excel in diagnosis and treatment, for the various shades of structural alteration revealed by the scalpel of Rokitansky, form so many problems for them to solve. Many of these new diseases are quickly diagnosed, and suddenly a "*New or Young Vienna School of Medicine*" springs into existence, at the head of which the modest Rokitansky is thrust by universal acclaim long before he had commenced to be ambitious of worldly distinction.

Hence we cannot see why the Dublin Journal should so quickly take the alarm because Rokitansky, different from all other pathologists, does not engage in the study or treatment of disease during life—because he is not now a practical physician, and seldom sees one of the many hundreds of sick, the bodies of which he dissects. This anomaly, although peculiar to this school, undoubtedly possesses many advantages: as Wilde truly observes, "We all know how difficult it is to dispossess the mind of any previously conceived and long cherished idea, by which we either treat or explain the phenomena of disease. We have all witnessed how frequently men generalize from a few particular cases, and how easily they find the morbid appearances to agree with the previous diagnosis."—Vide Broussais's gastro-enterite. "This is because the physician and pathological anatomist are one and the same person. The Vienna school, previous to the present mode of examining diseased structures, offered a well marked example of this defect. The *protokols* of the different medical and surgical sections of the hospital teemed with numbers of cases whose post-mortem appearances fully corroborated their previous diagnosis, yet but little advance was made in pathological science in those times. Furthermore, although I [WILDE] do not believe that diseases have altered, yet we now find pathological appearances quite different from what they seemed to be prior to the introduction of Rokitansky's me-

thod. The first emancipated himself from the incubus of ideal pathology and false diagnosis, and now teaches general pathology and morbid anatomy unobstructed by them." It is sufficient for Rokitansky's class to witness at least four or six, and frequently ten or twelve, post-mortems every morning; to hear the record of each case read, and the post-mortem appearances dictated aloud; to be required every afternoon to perform an autopsy in turn, describe the healthy and morbid appearances they meet with in each cavity and tissue, and answer the questions of the professor upon the subject; to listen to Rokitansky's remarks upon all the morbid specimens which have been collected during the day, and finally, when thus grounded in the principles of pathological science, and their eyes made familiar with diseased appearances, to follow him to the museum and hear an elaborate course of lectures, illustrated by the largest and most valuable collection of morbid specimens now extant. The rest of practical medicine can be learned at the bedside in the hospital, from teachers as competent in their peculiar departments as Rokitansky is in his, and mainly rendered so by the impulse which has been given to their studies by the peculiar labors of Rokitansky.

The Philadelphia Medical Examiner (see No. for August 5, 1843) has taken its cue and its materials from the Dublin Journal: from it we learn that "The present Vienna school of Pathological Anatomy, under the celebrated Rokitansky, has of late acquired much notoriety. Though an unrivalled pathological anatomist, he is in reality no pathologist, and as all his deductions are drawn from cadaveric appearances, his opinions on disease should be received with great reserve. We hazard this little caution here, because Rokitansky's reputation is a rapidly growing one, and as authority is every thing now-a-days in medicine, in a few years we shall not be surprised if the most astounding theories become the vogue, solely because they emanate from the fashionable Vienna professor. His late theory on Typhus is a specimen of what we may anticipate from a man whose studies are thus exclusive. As Rokitansky is the acknowledged head of the Young Vienna school, and as he is a man of great

abilities, as well as a clear and attractive writer, his influence is by no means limited."

Rokitansky's theory of typhus consists in the statement of fact, that a peculiar typhous-matter is found in the intestinal ulcers, mesenteric and bronchial glands; that the quality of the blood is much altered, as evinced by chemical analysis and its outward appearance; and he suggests that typhous fever is an acute dyscrasia, which may be classed and compared with the cancerous and tuberculous, which are also marked by a peculiar alteration of the blood, and the deposition of heterologous deposits.

Those who have never read his work may pronounce Rokitansky a clear and attractive writer; those who have, will be more apt to agree with the British and Foreign Review, that "it is written in such vile, crabbed Bohemian-German, that few, even of those to whom good German is familiar, will ever wade through it;" we may add that those who do, will also agree with the latter, that "no modern volume on morbid anatomy contains half so many genuine facts as Rokitansky's; and that it is alone sufficient to place its author in the highest rank of European medical observers." As to astounding theories becoming the vogue, merely because they emanate from the fashionable Vienna professor, the British Review declares that Rokitansky's work is no more than it pretends to be, viz., Pathological Anatomy, in its densest and most compact form, scarcely ever alleviated by a theory or hypothesis; and concludes by stating that he has said but little of its merits, as the best evidence of this is found in the extent to which he has abstracted from it, without passing the bounds of what is novel or important; and adds, nor would that fault have been committed though much more had been borrowed.

In conclusion, we would state that the second part of the work is almost ready for publication, but will not be forthcoming unless the sale of the first part warrants it. In the mean time we intend revising and re-writing it; we also hope to compile better notes on diagnosis than those which are appended to the first part, which was already in the printer's hands before our publisher insisted upon their being added.

ABNORMAL CONDITIONS

OF THE

ORGANS OF RESPIRATION.

They may be divided into those of the tracheal trunk and its branches, viz. (the larynx, the trachea, and the bronchi ;) into those of the lungs and of the pleural sacs. Departures from the normal state, in the thyroid, and thymus glands, will be treated of in an appendix.

I. ABNORMAL CONDITIONS OF THE AIR PASSAGES.

1. EXCESS AND DEFICIENCY OF DEVELOPMENT.

An entire absence of the air passages always occurs, when there is a simultaneous deficiency of the lungs. A partial deficiency, as of the trachea, may exist without the absence of the lungs, the bronchi then being given off immediately from the larynx. Occasionally we find deficient development of these organs, owing either to the absence of individual component parts, as of single laryngeal cartilages, tracheal rings, &c. ; or to imperfect development of the same in length and breadth, producing proportionate deformities.

An excess of development is seen in the duplication of the lungs in double monsters, the upper, or lower section only of the apparatus being double. We may also mention the occasional presence of a supernumerary third bronchial tube, sometimes seen

on the right side, in persons otherwise normally developed. Finally, supernumerary laryngeal cartilages and tracheal rings occur, and increase in a greater or less degree, the length of the tracheal trunk.

2. ALTERATIONS IN SIZE.

As regards the calibre of the air passages, we shall here take up the consideration of acquired dilatations and contractions, setting aside individual peculiarities, in which the whole apparatus is either very much increased in size, or where, on the other hand, it remains undeveloped with its walls slightly and delicately constructed. With regard to the thickness of the walls of the air passages, we shall have to consider the hypertrophy and atrophy of their component tissues.

a. ABNORMAL DILATATIONS OF THE AIR PASSAGES.

These occur in various forms in the larynx, trachea, and bronchi, existing either as dilatations of the whole apparatus, or more frequently, as dilatations of single parts only, as of the bronchi.

1. *Dilatations of the larynx and trachea.*

Uniform dilatation of these canals is not unfrequently seen in Marasmus, or Atrophia senilis. Its existence in extreme old age is interesting, because it always occurs in connection with, and in proportion to, an Emphysema senile, which is a Marasmus senilis of the lungs. They are all dependent upon an emaciation of the component tissues of the parts affected.

Another form of dilatation is one proceeding from an hypertrophy and relaxation of the posterior tracheal wall, with or without saccular or hernial protrusion of the mucous membrane. This, as might be expected from the protected state of the interstices between its cartilages, is extremely rare in the larynx, even in a slight degree; while on the other hand, it is not only of frequent occurrence in the trachea, but is at times developed to an astonishing extent. Notwithstanding that dilatations of the bronchial tubes have been well known since the time of Laennec, this cannot be said either of dilatations of the trachea in general, or of this form in particular.

The posterior tracheal wall is relaxed, and exhibits a remark-

able increase in capacity, particularly in the direction of its breadth. At the same time its mucous membrane, the transverse muscular fibres, and the muciparous glands, are all in a state of hypertrophy, and the excretory ducts of the latter dilated; on the other hand the elastic yellow longitudinal fibres are atrophied and scarcely visible. If the protrusion of the mucous membrane, mentioned above, should now take place, it gradually passes through between the thickened muscular fibres, presenting at first a fissure, or a funnel-shaped cavity, finally becoming a transverse sac, deepest at both extremities of the tracheal rings, where we generally find the distorted fissure-like mouth of the excretory duct of a muciparous gland. The larger this hernia or false diverticulum becomes, the more prominently do the muscular fibres, between which it protrudes, project on the inner surface of the trachea; and if the herniæ be numerous, and close together, the projecting muscular fibres form a species of lattice-work; their ends are sometimes single, sometimes forked.

This condition arises in consequence of repeated and chronic tracheal catarrhs, and may be compared with many similar diseases, arising in other parts from analogous causes. In the saccular dilatation with protrusion, the tracheal mucous membrane may, in the very commencement of the disease, be drawn through the transverse muscular fibres, by the traction exerted on it by the hypertrophied mucous glands through the intervention of their excretory ducts. At times these protrusions are found along the whole course of the trachea, sometimes extending even beyond it in the bronchi.

These dilatations of the trachea have a striking similarity to the equiform dilatations of the bronchi, proceeding from hypertrophy and paralysis of their walls.

2. *Dilatations of the bronchi. Bronchectasis.*

Besides dilatations of the bronchi from emaciation, as seen in Marasmus senilis, other forms of this affection occur. Indeed, this section of the lungs is distinguished by their frequent occurrence, and the extent and degree of development which they attain. They are among the most important diseases of the air passages.

We distinguish in particular, *two forms* of dilatation.

a. In the first, we find a bronchial tube *uniformly dilated* for a certain distance i. e., the dilatation, has taken place equally to-

ward all points of its periphery, so that a bronchus, which in its normal state will hardly admit of a fine probe, attains to or even surpasses the volume of a crow or goose quill. This enlargement is remarkably distinct, when we find a dilated bronchial tube far exceeding in size the branch from which it springs. It is rarely confined to a single tube, but, as a rule, affects a whole division of the bronchial tree. The branches and twigs may be increased in size in a degree proportionate to their normal volume, or, as is more frequently the case, they are found the more dilated the nearer we approach the extremities of their ramifications. In this they observe a law to be treated of hereafter.

b. The second form is the *saccular* dilatation. In this, we find a bronchial tube enlarged, so as to form a spindle-shaped or roundish sac. In the latter case, the dilatation is often greatest in some particular direction, and then the greatest diameter of the sac may fall without the axis of the entering or departing tube. Such sacs may in rare instances attain the size of a hen's egg, more commonly that of a bean, a hazel or walnut. We find also, that one or more portions of a bronchial tube may be dilated, so as to form a sac, while the remainder retains its normal calibre; or the whole of the bronchial ramification may be affected. In the latter instance, numerous similar sacs of various sizes are so arranged with regard to each other that they form a large, many branched, sinuous cavity, the individual excavations of which are bounded off, and separated from each other by ridges, or valve-like duplicatures of the bronchial walls. The saccular dilatation of the terminal branches of the bronchi forms a peculiar subdivision. We frequently see these distended in the form of thin, membranous vesicles, which are tightly filled with air; they are found either singly, or in groups, in the apices of the superior lobes of the lungs, or in the neighborhood of cicatrized tubercular cavities. This condition arises when a bronchial tube becomes compressed and almost obliterated in passing through the contracted parenchyma of the apex of a lung which has been rendered almost impermeable by obsolete and chalky tubercles, and by a deposit of pulmonary pigment; its termination then expands itself into one of the above described vesicles. If the bronchial tubes going to these are pervious, we can empty the sacs of their air by pressing on them; if they are not, they resist this and remain unchanged.

Of the two forms of bronchial dilatation, the second occurs the more frequently, especially in young subjects. The *degree*

of dilatation may be determined by measurement, with due attention to the original size of the tube, and the calibre of the parent branch from which it proceeds. Its *extent*, as may be gathered from the above, may be either very limited, or on the other hand so considerable, that all the bronchi of a single lobe, or a whole lung, may be affected.

Bronchial dilatation affects especially the smaller tubes, and as a general rule is most frequent in those of the third and fourth order, never occurring in the largest bronchial trunks, or at least very rarely, and then only in the manner described when speaking of tracheal dilatations.

The bronchi situated near the periphery and edges of the lungs, and especially those of the upper lobes, are most subject to this affection, and this among other circumstances shows its analogy to true vesicular emphysema.

The *walls* of the dilated bronchi are found in various conditions. At times the mucous membrane and fibrous sheaths are hypertrophied and thickened; the former showing the effects of a chronic catarrh, being swollen, spongy, villous, softened, easily torn, and of various shades of a dark red color. The walls of the bronchi are rigid; they gap open upon the cut surface of the lung; the thickened white fibrous sheaths contrast strongly with the internal red and swollen mucous membrane, from which a thick yellow purulent mucus is seen to exude. Such is generally the condition in the first or equiform variety of bronchial dilatation.

At other times, especially in the saccular form of this disease, the walls are found in a state of relaxation and emaciation. The mucous membrane of the sacs is but little if at all reddened; on the contrary, it is generally paler than usual; its tissue is but slightly if at all loosened or softened, but is smooth, and approaches the character of a serous membrane. The cavities contain a thin, puriform, pale yellow, or almost colorless glassy mucus.

This great difference in the condition of the walls in these two forms of dilatation would lead us to conjecture an equal difference in regard to their proximate and exciting causes, and these we shall proceed presently to point out.

The pulmonary tissue in the neighborhood of the dilated tubes is increased in density and unfit for the purposes of respiration. We will enter into a more thorough examination of the state of

this parenchyma, in connection with the formation of a theory of its origin, and its importance as a cause of bronchial dilatation.

Laennec and most of the succeeding pathologists believe that bronchial dilatation is always produced in a mechanical way, by the accumulation of catarrhal secretions in certain portions of the tubes, aided by powerful inspirations during paroxysms of coughing, the walls from some unknown causes being at one time rendered thicker, at another thinner than natural. The condensation of the surrounding parenchyma, according to them, is a consequence of the compression exerted by the dilating bronchus. Hence Laennec describes the dilatation of the tube as the primitive affection, and the condensation of the parenchyma as a secondary change.

Within a short time, Corrigan has put forth a theory directly opposed to this; and believing the disease to be exactly analogous in its anatomical elements to cirrhosis of the liver, he has given it the name of cirrhosis of the lungs. According to him, the atrophy and obliteration of the pulmonary tissue is the primitive effect in the development of the disease; the dilatation of the tubes a consecutive affection, arising not only in consequence of an endeavor to fill up the space left vacant in the contracting lung, by the forcible expansion of the bronchi during the act of inspiration, but also by a dragging apart of the walls of the tubes from the shrinking of the pulmonary tissue itself.

The alteration of the parenchyma of the lung in extreme cases consists in an atrophy, obsolescence, and contraction of the air cells, or even conversion of them into a cellulo-fibrous or callous fibro-cartilaginous tissue, which may either be white, or else punctated, streaked, or colored throughout with black pigment, and to which the fibrous sheaths of the bronchi become so intimately attached as to form a connected whole.

When we consider the vast extent to which the pulmonary tissue around the affected bronchus is atrophied, the nature and degree of this change, and recollect the fact that it does not always appear equally developed around the dilated tube, nor most marked the nearer we approach to it, then it becomes a matter of doubt if all this can be produced by the mere pressure of a thin, dilating bronchial tube, more especially as we see that simple condensation of tissue only, is induced by long continued and severe pressure from pleuritic effusions, &c.

A closer examination enables us to obtain other facts which

are sufficient to explain this metamorphosis, and which are of great importance in the production of dilated bronchi.

Under whatever form bronchial dilatation appears, a bronchitis may be regarded as the most frequent primary exciting cause. It acts in very different ways, but not mechanically, from the accumulation of secretion, as supposed by Laennec.

In the first variety, atony and paralysis of the contractile and irritable tissues of the tubes are present, occasioned by chronic inflammation and blenorrhœa. Their walls are then the more easily dilated by the inspirations and concussions in paroxysms of coughing, the more forcible these are obliged to be in order to throw out the accumulated secretions. This process is still farther aided by many of the smaller bronchi being completely filled with catarrhal mucus. This variety only arises in that portion of the bronchial system which forms the seat of the catarrh.

The second form, or the saccular dilatation of the bronchi, arises, not in the catarrhal section of the bronchial system, but beyond it. It is the consequence of a bronchitis in the terminal branches of the air tubes, causing, first obstruction of them by accumulation of secretion and swelling of their mucous membrane; and finally entire obliteration of them. The more labored and the more protracted the single inspirations, so much the more readily does the inspired air which is obstructed in its passage through the tubes tend to produce dilatation. (Reynaud.) The expansion takes place toward the perfectly impermeable portion of the bronchus, for the parenchyma and air cells which were supplied with air by it have now collapsed and become atrophied, thus giving rise to a space to be occupied by the dilating bronchus, which lies either entirely, or in some measure, in the midst of a collapsed and apparently compressed pulmonary parenchyma; hence this last appears the exciting cause, the dilatation the result. This, together with the fact that the collapsed tissues pass into the above described perfect atrophy, and contract still further, leaving additional space in the lung, approximates this theory to that of Corrigan. But we make the obliteration of the terminal tubes the first step; the obliteration of the parenchyma which they supplied, the second; the dilatation of the air tubes in order to supply the vacuum, the third.

According to Corrigan, the primitive affection is not Bronchitis, but a disease of the parenchyma of the lung, not so much an inflammation of the interstitial cellular tissue, as a peculiar pneumonic process, (to be treated of subsequently,) which slowly

extends from one lobule to another, depositing a product which becomes indurated, and unites closely with the pulmonary tissue itself, while the air cells become atrophied, obliterated, and transformed into the same substance. The resulting fibro-cellular tissue may in this, as well as in the first instance, tend to draw apart the walls of the bronchi during its contraction, and may thus lead to a farther enlargement of the bronchial sac and increased thinness of its walls.

Be this as it may, a smaller or larger portion of the lungs becomes contracted and obsolete in proportion to the extent of the bronchial affection; yea, when all the bronchi of a lung are dilated, we find its entire parenchyma atrophied, and contracted to a small portion of its normal volume, and drawn up towards the roots of the bronchi, as if in consequence of external pressure from an effusion into the pleura. The cavity of the chest becomes smaller than usual, for its walls sink in over the contracting lung.

In rare instances a bronchial sac is entirely separated by obliteration, not only from its own branches, but also from the tube to which it is attached. It then presents a closed cavity, which in consequence of the persisting activity of its mucous membrane, becomes still farther enlarged from the accumulation of its secretions. At a later period, should this action cease, the contents of the sac gradually become inspissated and of smaller volume; we then find a fibrous capsule inclosing a fatty chalky paste, or a solid earthy concretion.

When single saccular bronchial dilatations contain purulent matter, they may be easily mistaken for tubercular cavities, especially when they exist simultaneously with pulmonary tubercles, and are situated in the upper third of the superior lobes of the lung, viz., in the usual primary seat of tubercular disease. A closer examination will enable us to discriminate between them, for bronchial sacs and their cavities have a roundish shape; their lining membranes are smooth, and uncorroded; there is no ulceration of the bronchial tubes which open into them; their contents differ widely from tubercular pus; and the surrounding parenchyma is either not filled with tubercles, or with such only as are obsolete. They may also occur in portions of the lungs not usually the seat of tubercles. In other instances general principles will enable us to decide on the diagnosis, such as the fact that the usual seat of bronchial dilatation is in the peripheric portions of the pulmonary parenchyma, and toward the edges of the lungs, while

they are rarely seated in the very summit; finally, when the bronchial disease is very extensive, it is found to be incompatible with tubercles.

Bronchial dilatation, when extensive, owing to the atrophy of a large portion of the lungs, which attends it, occasions obstruction of the circulation, active dilatation of the right ventricle, congestion and dilatation of the whole venous system, cyanosis, excessive development and vicarious action of the permeable portions of the lungs, and is not unfrequently followed by bronchial and pulmonary hæmorrhage and hemoptoic infarction. If it attain a very high degree of development, it causes debility, emaciation, general cachectic appearance, dropsy, and finally, total exhaustion.

In consequence of the venous congestion and cyanosis which attends this disease, it affords a remarkable immunity not only from pulmonary tubercles, but from tuberculosis in general.* This fact has been known since the time of Laennec, with regard to bronchial dilatation; but the cause, and general applicability of the above rule have not been discovered until lately, and only led within a short time to various projects for the cure of consumption.*

* *Diagnosis.*—Dilatation of the bronchi may be easily mistaken for chronic phthisis; so close is the resemblance of these two diseases, that Laennec, Louis, Andral, and other great masters have repeatedly confounded them. It is attended with habitual dyspnœa, more or less cough, muco-purulent expectoration, which however is not flocculent and cheesy like the tuberculous, but sanious and often remarkable for its fetor; some lividity of the face, œdema of the feet, and dropsical effusions are common, owing to the frequency of its combination with dilatation of the heart; as these symptoms and this condition of the heart is much more rare in phthisis, Stokes justly affirms that they may assist us in forming a diagnosis. Another point of importance is that it developes itself slowly, and rarely attains such a degree as to prove the immediate cause of death. Again, some one of the characteristic signs of phthisis is generally absent; thus in a case which Louis mistook, there was absence of hæmoptysis, hectic fever, pain in chest, and dulness on percussion, but there was emaciation, cough with opaque greenish sputa, and mucous rattle with gurgling under the left clavicle; the case was of ten years' standing, and diarrhœa and night sweats did not set in until the last fifteen days of life; the face was pale and livid, the legs œdematous, and obscure fluctuation in abdomen. In a case mistaken by Lerminier and Andral there had been oppressed breathing for a year, one attack

CONTRACTIONS OF THE AIR PASSAGES.

These occur in every section of the respiratory apparatus, but present various differences in their extent and proximate causes. As regards the former they may proceed to closure, and perfect obliteration.

of hæmoptysis, cough, with copious expectoration of puriform and intolerably fetid sputa; during the day the pulse was scarcely frequent, but every evening he had a chill and a burning heat all night, *yet he never perspired;* emaciation, loss of strength, and diarrhœa for last six weeks; puerile respiration on the right side, feeble respiration on the left, with great resonance of the voice—the dilatation was on the left side.

The physical signs of bronchitis are also more marked and persistent than in phthisis.

The uniform dilatation is less easily to be mistaken for phthisis, than the saccular variety, but it is extremely difficult to distinguish it from chronic bronchitis; it occurs most commonly after an attack of pleurisy or pleuro-pneumonia; its most frequent seat is in the posterior and inferior portion of the left lung; it is always a consequence of extensive compression or obliteration of the parenchyma, and hence almost always attended with an evident flattening, or sinking in of the chest over the affected part. It is marked by extensive bronchial respiration, not only without dulness on percussion, but with a hollow, tubular sound like that produced when the trachea is percussed; extensive resonance of the voice, almost amounting to imperfect pectoriloquy; a puffing of the breath, and a strong blowing into the stethescope or ear of the observer; in some cases the veiled puff of Laennec, i. e. sensation as if a thin veil or wet membrane was interposed between the column of air and the ear, and vibrated at each breath of the patient. There are no physical signs of cavities. When the dilatation is confined to one or several bronchi, is moderate and nearly equiform, there is an habitual cough, neither very frequent or severe, a moderate mucous expectoration, precisely similar to that in subacute bronchitis, and diffused bronchophony. When many tubes are dilated there is a chronic and persistent cough, in severe and long continued fits, dyspnœa, abundant grayish-yellow, puriform expectoration, the voice and cough resounds under the ear, mucous rattle, a puffing of the breath. Laennec has laid great stress on the abundance of the expectoration, and its great fœtor; some of these cases present the symptoms of simple chronic catarrh with copious expectoration, and may last from ten to fifty years without any of the constitutional signs of phthisis. Immediately over the dilated tubes percussion is clear and often distinctly tubular—around them it is clear, of short duration, with increased sensation of resistance of the

1. Contractions may be occasioned by pressure from without, as in the larynx, and trachea, by an enlarged thyroid gland, in the trachea and bronchi by lymphatic ganglia of unusual size, aneurisms, extensive cancerous deposits in the neck and mediastinum, large thymus glands, effusions into the chest and cavities of the pleura, &c. According to King, the left bronchus may be compressed by a dilated left auricle. By these means, the air

walls of the chest, owing to the compression and obliteration of the parenchyma. Respiratory murmurs are harsh, bronchial, diffused, and blowing; the dry and humid rhonchi of bronchitis are present; also bronchophony, or imperfect pectoriloquy; bronchial cough. In phthisis the symptoms and physical signs are constantly increasing in degree and extent, in bronchial dilatation they often remain unaltered in both these respects for months and even years. The loss of flesh is rarely so great as in phthisis. In both the equiform and saccular varieties, the external signs of the tubercular diathesis are absent, while those of emphysema and disease of the heart are common.

The diagnosis of the saccular variety is much more difficult, and at times almost impracticable, as there is no point of absolute difference between its signs and those of cavities in the lungs. Still the long duration of the disease, the absence of hectic fever, except towards the last; although hæmoptysis may occur, yet if it be frequently repeated and abundant, it will almost decide the question in favor of the tuberculous nature of the disease. If the disease have been watched from the commencement, it will be found that the physical signs of a cavity are present without being preceded by dulness on percussion; at a later period, when the neighboring parenchyma becomes obsolete, dulness supervenes. In the majority of cases dulness of percussion precedes tuberculous cavities. In large sacs we find cavernous respiration, gurgling, and pectoriloquy, and to add still more to the resemblance to phthisis, they are generally seated in the upper lobes, although, according to Williams, nearer the mammary region than the apex of the lungs. Stokes says the diagnosis can only be made by watching the case; for in saccular dilatation the physical signs remain stationary; hence if the extension and enlargement of the cavity be too rapid to be explained by the supposition of a dilated tube, we may at once conclude on phthisis. Stokes has frequently had recourse to this method with success. According to Skoda, when several sacs are strung along one bronchus, and communicate with each other, they may present a characteristic auscultatory sign, viz., loud blowing, dry ringing, interrupted sound, like that made by geese in their flight; when the sac is large and its opening small, a very strong hissing sound precedes this; the expiration is attended by hissing or whistling.

tubes may be thrust from their natural situation, in various directions; their volume, as may be seen in contraction of the trachea, may be diminished so as to present mere fissures, either straight or curved, transverse or antero-posterior.

2. Contractions may also result from various diseases of the mucous or sub-mucous tissues, such as hypertrophy, inflammatory swelling, or œdema; or from the most various excrescences upon, or cancerous deposits about them, or from contraction of cicatrices, after solutions of continuity. The most frequent cause however is bronchitis, especially of the minuter tubes, which frequently become obliterated.

3. The volume of the air passages may be diminished, by the entrance of various foreign bodies from without; or from the intestinal canal, through the throat, or abnormal communications; or by the products of various diseases of the mucous membrane and the deeper seated tissues, among which we may enumerate, coagulæ of blood, lumps of mucus, bronchial froth, croup membranes, pus, tubercular masses, fragments of cancerous deposits, necrosed portions of cartilage and bone.*

c. HYPERTROPHY AND ATROPHY.

We have already alluded to an hypertrophy involving the mucous membrane of the air passages, the transverse muscular fibres, the mucous glands of the posterior wall of the trachea, and the fibrous sheath of the bronchial tubes, proceeding from catarrh and blenorrhœa of these parts. We must now speak of *hyper-*

* *Diagnosis.*—According to Walshe, if the contraction be limited to a single bronchus, the respiratory murmur is suppressed over an extent corresponding to the size of the affected tube; exaggerated and puerile respiration is heard in its neighborhood—sibilant rhonchus is much more common than sonorous, and often is attended with a well-marked and strong hissing; they are heard over the locality of the contraction, while sonorous rhonchus is heard in the neighboring larger bronchi.

If the contraction be general and uniform, the respiratory murmur will be very feeble, almost inaudible, or quite suppressed on the affected side; with exaggerated and peurile respiration on the sound. The percussion sound is not as full as in health, but far from as dull as in phthisis. Several of Andral's patients had a feeling of constriction at the contracted part, and sensation as if they did not breathe with that part of the chest.

trophy of the mucous membrane and the muciparous glands of the larynx and trachea.

When existing in a moderate degree, it presents the usual characteristics of hypertrophy of mucous membranes; but when extreme, it affects in particular the muciparous glands, occasions their enlargement, and attacks those parts most abundantly supplied with them, viz., the neighborhood of the superior vocal chords, the ventricles, above the musculus transversus, and the epiglottis.

In the trachea we notice the dilated mouths of the excretory ducts of the mucous follicles, situated behind the muscular coat, and gaping open on the swollen mucous membrane of its posterior wall. These glands are enlarged to the size of a hemp seed, pea, or cherry, and are changed either into simple, or sinuous, or imperfectly partitioned sacs, in the cavities of which a whitish, opaque, or transparent glassy mucus is accumulated.

In its highest degree, the swelling of the membrane and glands of the larynx becomes a polypous hypertrophy or gives rise to cellular or mucous polypi.

When we find the mucous membrane of the air passages and its glands atrophied, especially in the larynx and trachea, there is a diminished secretion of mucus, with simultaneous emaciation of the laryngeal muscles, followed by the dilatation of the larynx, trachea, and bronchi, already described as peculiar to aged persons. The epiglottis is occasionally the seat of atrophy with relaxation, arising apparently from subacute inflammation; or from similar causes it may become atrophied, indurated, and variously deformed, so as to allow only an imperfect closure of the glottis. Finally emaciation, and final absorption of the laryngeal cartilages, the tracheal and bronchial rings with their membranes, occurs in consequence of the pressure of various tumors, such as aneurisms, &c.*

* *Diagnosis.*—According to Williams, when the bronchi are hypertrophied, they expand less easily, and hence there is difficulty of inspiration, which is short, quick, and labored, especially on making any exertion; the lungs cannot accommodate themselves by a supplementary effort; hence, if pleurisy or pneumonia set in, the oppression of respiration is very great. *Expiration* is comparatively easy, but both acts are often attended by wheezing; the expansion of the chest is perceptibly limited; the vesicular murmur is feeble. The signs in fact are those of a permanent and not very severe spasmodic asthma.

3. DEVIATIONS IN FORM.

Among these belong the acquired deformities of the larynx, trachea, and bronchi, such as flattenings, indentations, curvatures caused by the pressure of enlarged thyroid glands, aneurisms, encysted tumors, &c., or from cicatrizations after ulceration.

The epiglottis in particular presents some remarkable anomalies, being frequently found irregularly flattened, its edges unnaturally curled, or its apex curved downwards, or else rolled together like a horn. These malformations are either the consequences of contracting cicatrices in its mucous membrane, submucous cellular tissue, or cartilage; or of inflammation of the latter with consecutive softening induration, or emaciation of its substance, and transformation of the same into a rigid fibrous tissue.

4. ALTERATION OF POSITION.

These occur especially in the larynx and trachea, which may be forced from their natural position to the right or left side, by partial enlargements of the thyroid gland, by aneurisms, encysted tumors, abscesses, cancerous deposits, or in consequence of wry neck. They may be thrust forward by swollen and inflamed cervical vertebræ, by abscesses in the same, by aneurism of the arch of the aorta, cancerous deposits in the anterior mediastinum, &c. The occasional, but very rare dislocation of one of the laryngeal cartilages, may also cause alteration in position.

5. SOLUTIONS OF CONTINUITY.

These may be divided into:

a. The various injuries of the air passages, from cutting or penetrating weapons, and gun-shot wounds; fractures and bendings of the hyoid bone, laryngeal and tracheal cartilages, from lacerations, blows, and severe pressure; and from the entrance of angular and pointed bodies into their cavities.

b. The gradual solution of continuity from absorption and atrophy, caused by the long continued pressure of aneurisms.

c. Lastly, the multiform solutions of continuity, the consequence of various ulcerative processes, acting from within outwards, and vice versa.

All of these, more or less rapidly, give rise to unnatural com-

munications of the air passages with the surrounding cellular tissue, or adjacent cavities and canals; as with the pleural sacs, blood-vessels, œsophagus; or with abscesses in the lungs, bronchial glands, vertebræ, and lateral or anterior walls of the chest. They allow, as well the exit of air in various directions out of the passages, as the far more dangerous entrance of blood, purulent ichorous fluids, food or drink, into them.

We might here allude to the so-called congenital tracheal fistula described by Dzondi, Aschersohn, Serres, and others; but it requires still farther investigation to form a theory of its mode of formation, and its importance. It is an arrest of development.

6. DISEASES OF THE TEXTURE OF THE AIR PASSAGES.

They occur in all the component tissues of these parts, but the mucous membrane is most frequently primitively affected, and in a greater variety of forms; hence its diseases require the most attention, particularly as they often extend themselves to the subjacent tissues, and cause their destruction.

(1.) DISEASES OF THE MUCOUS MEMBRANE AND SUB-MUCOUS CELLULAR TISSUE.

a. HYPERÆMIA AND ANÆMIA.

Hyperæmia of the air passages is a diseased condition of common occurrence. When seated in the minuter bronchial ramifications it becomes combined with hyperæmia of the parenchyma of the lungs. When it occurs in the larger bronchi, trachea, and larynx, it usually exists as an independent and uncomplicated affection. It becomes of more or less importance, in proportion as it occurs either as an active hyperæmia, or a mechanical one, from obstructed circulation; or as a passive, which is rare; in either case it may form an independent disease, which finally gives rise to hemorrhage, or under other circumstances to congestion, which may increase to acute or chronic inflammation. The hemorrhages, may proceed from the bronchial, tracheal, laryngeal, or epiglottideal mucous membranes. When they occur, we find the air passages of the cadaver filled to a varying extent with different quantities of coagulated or fluid blood; the mucous membrane is

dark-red, swollen, spongy, relaxed, and bleeds easily from a greater or less extent of its surface when pressed upon; we find no other certain source of the hemorrhage, viz., no pulmonary apoplexy, mechanical or ulcerative solutions of continuity, &c. Portions of the pulmonary parenchyma are often spotted of a dark or light-red color, from imbibition of blood, which has settled into the air cells and terminations of the bronchi; this, however, is not peculiar, but occurs in all other forms of hemorrhage in the lungs. Other parts of them are pale, puffed up, and emphysematous, as the air cannot pass out, in consequence of the obstruction of the bronchial tubes.

These hemorrhages are of an active nature during the evolution of the body, when general plethora is present, and in vicarious discharges for the menstrual or hemorrhoidal flux. They may originate from, and accompany the congestions, which so frequently precede and attend the developement and progress of tubercles of the lungs, and may be brought on by violent exertions, either of the whole body, or the organs of respiration. We see them occur from concussion of the lungs, from rapid rarefication of the atmospheric air, but in the vast majority of cases from mechanical hyperemia the result of hypertrophy and dilatation of the heart.

Anemia of the mucous membrane of the air passages is most remarked in atrophia senilis.*

1. CATARRHAL INFLAMMATION.

This is one of the most common diseases of the air-passages; it occurs either as an acute or chronic affection, and in one or the other form most frequently attacks single sections only of the bronchial system. The acute affection however occasionally

* *Diagnosis.*—According to Schoenlein, in simple hemorrhage from the bronchi there are no preceding signs of a congestive stage, no sensation of weight, pressure, or increased warmth in the chest; but the patient merely has a burning sensation in the larynx or trachea, or through a larger or smaller section of the bronchia. To this is added a peculiar irritation and a short paroxysmal tickling cough by which mucus is brought up, which is either saturated or tinged with blood of a bright red color, and sweetish taste. There are hardly any physical signs; nothing peculiar is elicited by percussion, and auscultation only affords a moist rattle of unequal sized bubbles, usually larger and more liquid than those of catarrh.

travels over the whole tract of the respiratory tubes, and the chronic form frequently affects their whole extent, the inflammatory action being then, however, much more intense in some parts than in others. We distinguish catarrhs of the larynx, trachea, and bronchi, or *laryngitis, tracheitis, and bronchitis catarrhosa.*

ACUTE CATARRHAL INFLAMMATION.

This presents the same pathological phenomena in whatever section of the lungs it occurs; viz., various degrees of redness, and relaxation of the membrane, which, according to the stage and intensity of the attack, either secretes increased or diminished quantities of muco-serous, frothy fluid, (sputum crudum,) or of thick, white, or yellow puriform mucus, (sputum coctum,) or finally, of a true purulent secretion, which latter marks the transition into superficial suppuration. The swelling of the mucous membrane and the subjacent cellular tissue is worthy of especial attention, on account of the diminution in the volume of the air-passages which it occasions. It is of the greatest importance when it attacks the lining membrane of the epiglottis, the duplicatures which bound the rima glottidis, cover the vocal chords and line the ventricles; and also when it affects the mucous tissue of the smaller bronchial tubes.

Acute bronchial catarrh is an important, and, in children, often a dangerous disease, not only from its extent, but from the partial closure and even perfect impermeability of the bronchial tubes, occasioned by the swelling of the mucous membrane, and accumulation of its secretions. In children also it is particularly apt to extend to the air cells, causing catarrhal pneumonia.

CHRONIC CATARRHAL INFLAMMATION,

Occurs with great frequency in certain portions of the air passages, is often remarkable for its great intensity, and is of the greatest importance from its results. These remarks refer particularly to chronic bronchial catarrh. This species of inflammation is by no means rare in the larynx and trachea, and at times extends over the whole respiratory tract; generally, however, is most intensely developed in some one particular locality. Scarce any other acute catarrh suffers so many relapses, or so easily becomes habitual; while the chronic variety frequently increases to acute inflammation, or degenerates to a mere

blenorrhœa. It presents the general anatomical characters of chronic mucous inflammations, and is a very common cause of swelling in those portions of the larynx already described as well supplied with mucous follicles, giving rise to glandular hypertrophy, mucous polypi, and condylomatous or cauliflower-like vegetations of the epithelium. In the trachea, and still more in the bronchi, it may cause spongy thickening of the lining membrane. The conditions just mentioned may lead to hypertrophy and relaxation of the sub-mucous muscular layer of the fibrous portions of the vocal apparatus, and of the fibrous sheaths of the bronchi; or they may cause ulcerative loss of substance, especially of the larynx, in the form of a diffuse catarrhal suppuration, or of catarrhal follicular ulceration.

Chronic catarrhal inflammation may induce a diminution of the volume of the air passages, sometimes amounting to perfect closure; in the bronchi, adhesion and perfect obliteration is often the result of previous loss of substance by ulceration. At other times it gives rise to dilatation, with hypertrophy and paralysis of the tissues.

The quantity of whitish cream-like, or yellow purulent secretion, that is effused from a blenorrhoic bronchial mucous membrane, is very great, especially where dilatation is present. Such is the condition of the lung in the so-called phthisis pituitosa, asthma humidum, bronchial blenorrhœa; hence, if it be incised large quantities of mucus flow from the divided bronchi, and spread over its cut surfaces.

The acute as well as the chronic form may occur as an independent and idiopathic disease, although they are frequently associated with catarrhs of other mucous membranes. The acute is frequently of an exanthematic character, arising in the course of measles, small pox, and typhus. The chronic form frequently accompanies the most different pseudo-plastic processes upon the mucous and sub-mucous tissues, and is often of a syphilitic, arthritic, or scrofulous nature; thus it frequently attends on tubercular phthisis. It may also arise from mechanical hyperæmia, induced by disease of the heart.

Gonorrhœal catarrh of the larynx deserves particular mention on account of its sequelæ. By metastasis, it attacks the mucous membrane of the epiglottis, the lateral duplicatures of the glottis itself, and the vocal chords, converting them and the subjacent cellular tissue into a fatty-fibrous, white, and resistent texture of considerable thickness, which causes a diminution or narrowing

of the *rima glottidis* and cavity of the larynx. (*Gonorrhœal laryngeal stenosis.*)

The parenchyma of the lung suffers from a chronic bronchial catarrh in two ways ; it causes emphysema at one time, at another collapse of the air cells, obliteration and consequent atrophy of them. Livor, cyanosis, active dilatation of the right ventricle, and hydro-thorax follow in its train, and it proves fatal either in consequence of these, or by producing asphyxia, or tabes under the form of phthisis pituitosa.*

* *Diagnosis.*—The symptoms of bronchitis vary according as the disease is seated in the larger, smaller, or capillary bronchi. Inflammation of the larger air tubes is rarely attended with such violent symptoms as when it attacks the minuter ones. Fever and dyspnœa are moderate, cough loud and deep, expectoration scanty and glairy at first, afterwards easy, profuse, and mucous. The percussion sound is normal, but the hand applied to the chest often detects rhonchal vibration, especially if intense sonorous rhonchus be present. In the first stage auscultation detects sonorous rhonchus, which when dry is of a grave tone, resembling the prolonged note of a bass violin, or the cooing of a dove; when free expectoration comes on, the sonorous rhonchus is intermixed with mucous rattle of large, scattered, and uneven bubbles. The presence of sonorous rhonchus alone, usually implies that the case is not a dangerous one.

The more common form of bronchitis is that in which the larger bronchi, and those of the second, third, and fourth orders, are simultaneously inflamed. In this variety there is often burning, piercing pain under the manubrium sterni, which commonly extends towards the middle of the sternum, more rarely to the sides of the chest; there is comparatively great oppression of the chest and dyspnœa, but the patient is able to expand the chest fully ; the cough is deep, metallic ringing, dry and hard; the expectoration, according to Andral and Watson, is scanty in the first stage, often streaked with small lines of blood, saltish, transparent like the white of an egg, glairy and viscid ; it is stringy and viscid in proportion to the intensity of the inflammation, and at times may be drawn out into threads like those of melted glass. If expectorated after much coughing it will be frothy, with unequal sized large and small bubbles. When the inflammatory fever ceases a remarkable change in the expectoration ensues; the mucus gradually loses its transparency, and becomes mixed with yellowish white or greenish opake masses, which increase more and more until they compose the whole of the expectoration, which is then thick, consistent, muco-puriform, though somewhat viscid and confluent.

2. EXUDATIVE PROCESSES (CROUPOUS INFLAMMATION).

Under this head belong affections allied to each other from their originating in one general disease, but which differ widely in their local manifestations. These differences, so far as they are the objects of pathologico-anatomical investigation, are confined to variations in the physical qualities of the inflammatory products effused upon the free surfaces of the mucous membranes, and to variations in the condition of these membranes themselves, together with their sub-mucous cellular tissue. They occur,

Percussion sound remains comparatively clear, and this is of importance; for if, after three or four days of fever, cough, hurried and laborious breathing, we find that the chest sounds clear, the great probability is that the disease is bronchitis, and not pneumonia or pleurisy.—[Stokes.] Auscultation detects sonorous and sibillant rhonchus simultaneously, causing a strange medley of groaning and cooing, loud and hoarse sounds intermixed with chirping, whistling, and hissing, with sub-mucous rhonchus, and with the natural, gentle, rustling, breezy, vesicular murmur; the less the latter is heard, the more severe the disease; the more the sibilous rhonchus predominates over the sonorous, the less favorable the prognosis.—[Watson.]

Capillary bronchitis is a more serious disease than pneumonia; when the capillary tubes are extensively inflamed, the dyspnœa and oppression of the chest is very great, the whole disease is of an intense and severe character, and unless quickly relieved runs on to a fatal termination with great rapidity. According to Skoda, the commencement of the attack is often marked by a very loud, rough, vesicular murmur; this quickly changes to a dry and sharp wheezing, with sibilant rhonchus, hissing and whistling. According to Graves, when we hear numerous sounds, three, four, or even six or seven in number, proceeding from and audible over a small portion of lung, with wheezing from a great many points close together, we may be sure of capillary bronchitis: these sounds undergo rapid changes during the same inspiration—every moment some of them seem to cease, and to be replaced by new ones. The sibillant or whistling rhonchus depends on inflammation of the minuter tubes, while the sonorous, droning, humming, snoring, or cooing sounds, indicate that the larger bronchi are affected. In bronchitis of the larger tubes the sonorous rhonchus may outroar the vesicular murmur; but generally this is also heard mingling with it; but sibillous rhonchus proceeds from the immediate neighborhood of the air cells and abolishes the respiratory murmur—it does not mask it, but it takes

especially in true croup, either as primary independent diseases, or as secondary processes, the mere results of a degenerate or chronic affection.

True croup, which demands our first attention, is an exudative process, giving out a more or less plastic exudation. It rarely occurs, as a genuine primary process, upon any other than the mucous membrane of the air passages, and appears there under the forms of laryngeal, tracheal, or bronchial croup, i. e. *laryngitis tracheitis, bronchitis polyposa vel membranosa.* It occasionally extends over the whole of the air passages, progressing from the

its place. Sibillous is a more dangerous sign than sonorous rhonchus; when it is heard all over the chest, the whole of the capillary bronchi are affected, and the case is a very serious one.—[WATSON.]

When the first or dry stage passes away, *true sub*-crepitant rhonchus is heard; this is a small fine sound, like that heard when the ear is applied near the surface of a liquid slightly effervescing, such as bottled cider or champagne. We are indebted to Louis for establishing the frequency and pathognomonic character of *sub*-crepitant rhonchus at *both bases* of the lungs as a sign of capillary bronchitis: the only sound for which it can be mistaken is the crepitant rattle of pneumonia; but double pneumonia only occurs once in sixteen or seventeen cases—so that, in an immense majority of cases, when a sound approaching the crepitant rhonchus is heard at the base of both lungs, the case is one of capillary bronchitis and not of pneumonia. —[WALSHE.] The only exception to this rule is in the case of newborn infants, in whom double pneumonia is infinitely the most common form; in 128 cases observed by Valleix and Vernois, both lungs were inflamed 111 times.—[Ibid.] Severe capillary bronchitis of both lungs is remarkable for the rapidity with which collapse and extreme prostration and debility succeed to high fever, extreme dyspnœa, &c.

Bronchitis infantilis is very insidious in the beginning; there is common catarrh with coryza, without pain, fever, or dyspnœa, but a frequency and wheezing of the breathing may be noticed, with unusual paleness and depression; cough is frequently, and expectoration almost always, absent. The physician often does not take the alarm until extreme dyspnœa shows the imminency of the danger; the pulse then becomes very quick; remissions occur in which the child lies in a somnolent state, without cough, or quickness of pulse, but if the breathing continues quick the dyspnœa will return with increased severity, much greater quickness of the pulse, while stupor ensues, the lips become livid, and the little patient dies suffocated.—[WILLIAMS.]

epiglottis to the very minutest branches of the bronchi; at other times it spreads over the throat and pharynx, and even down the esophagus, although rarely. It attacks the mucous membrane either in large continuous sections, or only in small insulated patches which are indented at their edges. Hence the exudation or croup membranes either present the appearance of connected tubular coagulations, which ramify in accordance with the bronchial divisions, or of irregular patches or layers, as is most common in the larynx. In bronchial croup, the tubular exudations exhibit a thickness which is in inverse proportion to their calibre, being converted into solid cylinders in the minute bronchi.

These exudations vary very much in thickness and consistence, some presenting a hoop or ring-shaped or gauze-like coating of the bronchi, while others are a line or more in thickness. In consistence they vary from that of viscid cream to the most compact, leathery, and fibrous false membranes; but even these latter are not of the same density and thickness in all parts, for as a rule they become thinner and softer toward their edges, which are creamy and purulent. The layer applied to the mucous membrane is always found softer and looser than the others; it adheres sometimes very intimately, at others but slightly, as a viscid secretion is often effused beneath it. These false membranes are generally of a grayish-white or yellow color, but not unfrequently they have a greenish tinge. On the under surface of the layer, in juxtaposition with the mucous membrane, we occasionally notice red streaks and points, consisting in part of superficially adherent blood, in part of straight or tortuous streams of it, or of roundish extravasations, from which small radiating and branching streams proceed. The membrane itself lying beneath these exudations presents a very various aspect: its redness at times is very dark, even brownish; more frequently it is of a lighter, erysipelatous hue, but occasionally no traces of any injection whatever are to be found; its surface is excoriated, and bleeds from many small isolated spots; the whole presents various degrees of swelling; at times, however, so slight as to be scarcely perceptible. The sub-mucous cellular tissue is generally, although not always, the seat of serous infiltration.

Genuine croup of the air passages occurs most commonly during the years of childhood; rarely, however, before the end of the second year, when the laryngeal and tracheal variety is most frequent. In older persons, viz., during the years of youth and early manhood, bronchial croup is the more prevalent form, and is

often complicated with pneumonia. Croup of the terminal branches of the bronchi always occurs simultaneously with pneumonia ; it generally runs an acute course, but may become chronic, the process continuing in a lesser degree of intensity for a longer period, and being subject to paroxysmal exacerbations with renewed depositions of lymph. It becomes habitual in many persons, and bronchial croup in particular exhibits a certain periodicity in its attacks. It is frequently complicated with pneumonia, pleurisy, pericarditis, and at times with meningitis and acute and chronic hydrocephalus. In rare instances it appears on the stomach and degenerates into acute softening of that organ (gastro-malacia). It proves fatal in consequence of the obstruction of the air passages by the effusion, or, as is more commonly the case, by the swelling of the mucous membrane and the sub-mucous cellular tissue, coupled with spasmodic closure of the glottis. Suffocation frequently occurs from œdema of the lungs, or death may result from exhaustion consequent on the profuse exudation. No evident affection of the par-vagum has been found.

The other exudative processes deposit a softer and less plastic effusion, which in some cases is almost purulent ; or a thin, sero-purulent, gelatinous, discolored, ichorous exudation, wherein the mucous membrane becomes much attenuated, and finally entirely dissolved. The sub-mucous cellular tissue is infiltrated with similar matters, and is rendered friable, easily torn, and diffluent. These are in most instances secondary processes, arising from the localization of some degenerate constitutional affection, such as acute variola, or scarlatina.

The collective exudative processes are frequently complicated with similar diseased actions on other mucous or serous membranes. They may all degenerate to gangrene, or acute softening of the parts they affect ; and from the simultaneous enlargement of the spleen, lymphatic glands, and follicular apparatus of the intestinal mucous membrane, we conjecture their origin in a general dyscrasia of the blood and lymph.

Among such general dyscratic diseases belong apthæ of the air passages. They are generally confined to the larynx, trachea, and larger bronchi, rarely extending upon the pharynx. They scarcely ever occur as a primitive disease, but most commonly associate themselves with tubercular phthisis of the larynx and lungs.*

* *Diagnosis.* — According to Dr. Ware, of Boston, true croup

3. PUSTULAR INFLAMMATION.

The only but most perfect form of pustular inflammation occurring on the air passages is the variolous. It is seen when the disease is of very great severity, and attended with profuse eruption of the exanthem on the skin. Single discrete pustules are then found upon the mucous membrane of the epiglottis, the adjacent soft palate, the larynx, trachea, and occasionally in the larger bronchi; they are soft, easily rubbed off, sometimes confluent, and leave shallow, concave, roundish ulcers; the mucous membrane beneath is of a dark red or livid color, and appears excoriated; in the intervals between the pits it is more or less reddened, thickened, and coated with a tough, plastic, mucous, or croupy exudation. At times it is found swollen and, together with

rarely attacks children under two years of age; it is commonly gradual and insidious in its approach, for in thirty cases its onset was sudden in two only; this rule does not apply to it when it occurs as a sequel to scarlet fever, for then it is usually very rapid and almost inevitably fatal. In the simple inflammatory and spasmodic pseudo-croups the attack is almost always sudden, and usually sets in during the first sleep of the child, before the parents have retired to bed: the little patient awakes in great distress—*inspiration* is attended with great effort, is loud ringing and shrill, somewhat like the inspiration in hooping cough; expiration is comparatively easy and quiet; the voice is hoarse and broken, the cough is loud, hoarse, and barking; such cases almost always give way to an emetic.

Catarrhal pseudo-croup is more difficult to distinguish from true croup than the spasmodic variety. At first there are symptoms of catarrh; in a few days the voice becomes hoarse, the cough croupy; there is tightness and oppression of the chest, with some approach to croupy inspiration; at times sudden attacks of dyspnœa set in, with loud, shrill, sonorous breathing; but in a few days more the croupy character wears off, leaving simple catarrhal symptoms only.

The best diagnostic sign of true croup, according to Dr. Ware, is the presence of false membranes, most frequently and sometimes only on the tonsils, sometimes on the palate, uvula, and pharynx. Of thirty-three cases of true croup, in which the throat was examined, they were present in thirty-two; in one case there were none on the fauces, but some in the larynx; of forty-five cases of pseudo-croup they were not found in a single instance; in three cases, however, there was a thin, starch-like exudation on the tonsils—hence this sign failed only once in seventy-eight times. True croup is comparatively rare; Dr. Ware met with but twenty-two cases in twelve and a

the sub-mucous cellular tissue, in a state of serous infiltration. A very extensive confluent pustular eruption may bring on a variolous ulceration of the parts.

4. TYPHOUS PROCESS.

The typhous process of the air passages presents various peculiarities, both in its seat, its relation to typhus fever in general, and to ileo-typhus in particular.

It appears in all cases of typhus fever in the form of a peculiar typho-bronchial catarrh, attended with a secretion of tough, gelatinous mucus; it is present in a marked degree when the general affection is severe, and in those varieties characterized by predominant affection of the chest. It may occur as a genuine

half years' practice; of these nineteen died. In the same space of time he met with eighteen cases of simple inflammatory croup, all of which recovered; with thirty-five cases of spasmodic croup, none of which proved fatal; and with fifty-six cases of the catarrhal variety, without a single death.

In pseudo-croup the cough is often much louder and more violent in the beginning than in those which ultimately prove more alarming. In the less dangerous cases the breathing is generally loud, harsh, and suffocative from the commencement, and attended with great efforts, much loud coughing, creating great alarm, and calling for immediate relief; but in the commencement of true croup the breathing is comparatively quiet and unobtrusive; there is only a little more effort in drawing in the air, and a little more force exercised in its expulsion; soon there is a slight dilatation of the nostrils at each inspiration, and a slight whiz or buzz at the rima glottidis, heard by placing the ear on the back of the neck, or upon the larynx. In the latter stage the symptoms are more formidable and urgent than in the first stage of the less dangerous varieties: they are too well known to require a full detail.

According to Dr. Ware, the recovery from membranous croup is slow and unequal; the natural cure is brought about by a suppurative inflammation which commences in the trachea, beneath the false membranes, which are thus loosened and finally thrown off; it advances slowly about the glottis. The progress of the cure is attended with copious expectoration of pus, with or without pieces of false membrane, which often become dissolved by the pus; expectoration may also be mixed with blood — voice may not return for weeks. These accidents never occur even after the severest cases of simple inflammatory croup.

or degenerate typhous process; in the first instance being either a primary or secondary affection, in the latter always a secondary. Its seat is at times upon the bronchial, at others upon the laryngeal mucous membrane. On the former it frequently presents itself as primary broncho-typhus, which is a disease of great importance; on the latter it occurs as laryngo-typhus, which is always, at least with us in Vienna, a secondary affection.

a. The genuine typhous process upon the bronchi almucous membrane always appears in the form of an intense diffused congestion; the membrane itself is of a dark red, almost violet color, spongy and swollen; a gelatinous, and at times a dark, blood-streaked mucus is secreted, often in large quantities. It is most common in the bronchial ramifications of the inferior lobes of the lung, and is always limited to the stage of congestion; never giving rise to the visible production of those ulcerations which occur in such immense numbers in the follicular apparatus of the intestine, in abdominal typhus.

In primitive broncho-typhus, the typhous process localizes itself upon the bronchial mucous membrane, avoiding all others, even that of the intestine, for which it in general shows the most decided preference; although it is true that, even on the latter, we in many instances notice an evident, though comparatively trivial, secondary enlargement of the intestinal follicles, in which the neighboring mesenteric glands also take part. In such cases it is often a very difficult matter to recognize the presence of typhus fever in the above described affection of the bronchial mucous membrane; still the peculiar congestion of the spleen and of the greater cul de sac of the stomach, the remarkable tumefaction of the former, the well-known cherry-juice appearance of the blood, and the affection of the bronchial glands, will generally suffice to mark its typhous nature. The bronchial glands present similar diseased appearances to those of the mesentery in ileo-typhus: they are swollen, enlarged to the size of a pigeon or hen's egg, and at first of a dark bluish-red color; at a later period reddish-blue, soft, friable, and infiltrated with the marrowy typhous product. Like the mesenteric glands they may become the seat of sudden and tumultuous changes, leading to inflammation of the adjacent mediastinum and pleura, with or without perforation of them. It is frequently combined with pleuro and pneumo-typhus, and is undoubtedly the basis of the exanthematic and contagious form of this disease, and may also be present in the Irish and North American typhus, which, in a majority of

cases, seem to run their course without any intestinal affection. Broncho-typhus is rare with us in Vienna; at least, it cannot be compared in point of frequency with ileo-typhus.

Genuine secondary bronchial typhus presents the same morbid phenomena as the primitive, only it is rarely developed in so severe a degree. The *degenerate* form is still more rare, generally occurring as a species of bronchial croup, or as diffused gangrene of the bronchial mucous membrane.

b. Laryngo-typhus is with us in many epidemics an unusually frequent and extremely unfavorable phenomenon. It perhaps never occurs as a primitive, independent affection, but is always secondary, and dependent upon and growing out of various anomalies in the intestinal typhous affection.

Its seat is in the laryngeal mucous membrane, above the musculus transversus, and towards the posterior extremities of the ventricles (a situation for which, as we shall subsequently see, almost all the pseudo-plastic processes have a peculiar predilection). Next it is most frequent on the epiglottis, towards its lateral edges; at times, however, it attacks both of these places simultaneously.

It no doubt frequently occurs in a genuine form, but it is very rarely that we have an opportunity of seeing the typhous infiltration in its crude state, or while in the process of softening; we usually find ulcers similar to, but much smaller than, those of the intestine.

Far more frequently laryngo-typhus presents itself in a degenerate form, as an exudative or croupous process, or, what is more frequent, as a superficial gangrene. The latter, after the slough has fallen off, leaves an ulcer which cannot be distinguished from other degenerate typhus ulcers, so that from the cadaveric appearances alone we can form no correct idea of their origin.

These ulcers are of a circular shape, of the size of a lentil or pea; at times discrete, at others confluent from the coalition of two, three, or more. As we have already intimated, they are seated upon the posterior laryngeal wall and the lateral edges of the epiglottis, in both of which situations they occur as linear ulcers; while on the inferior surface of the epiglottis they always present a more or less circular form. They have all an atonic character; are lax, discolored, with black edges, and eat gradually into the musculus transversus, the ventricles, the vocal chords, the cricoid cartilage, and that of the epiglottis, in all of which they occasion softening, or necrosis, and exfoliation. Upon the

posterior laryngeal wall we not unfrequently notice abscesses of a larger or smaller size, in which the necrosed cartilages are found bathed in a brownish ichor. At times they perforate into the pharynx. The whole forms a typhous-laryngeal-phthisis.

Laryngo-typhus is frequently complicated with pneumonia, and with secondary broncho and pharyngo-typhus.*

c. INFLAMMATION OF THE SUB-MUCOUS CELLULAR TISSUE.

Besides the share which the sub-mucous cellular tissue takes in the inflammations of the mucous membrane of the air passages, it may also be the primitive seat of this affection. It is of rare occurrence, however, and when it does appear it is usually the

* *Diagnosis.* — According to Andral, broncho-typhus is more frequently latent than any other disease of the lungs, and it may lead to brown or livid softening of the parenchyma, before this is suspected to be the seat of any disease. It is most apt to supervene in the third or fourth week of typhus fever, and is only marked by a sudden increase of the prostration of the patient; the pulse becomes much more frequent and often extremely feeble, although occasionally hard; the features change, and the cheek bones present a livid redness, which contrasts strongly with the livid paleness of the rest of the face. There is often no appreciable difficulty of breathing, neither cough or expectoration; the patient will sink rapidly, apparently from mere nervous weakness, yet after death the bronchial mucous membrane will be found in a state of intense inflammation.

At other times the disease is only marked by hurried breathing, without cough or expectoration; sometimes auscultation discovers nothing unusual; the respiratory murmur seems normal, but if the patient be compelled to cough strongly, or to make several forced and deep inspirations, the auscultator will frequently be astounded at the number, variety, and intensity of the bronchial rales.—(Stokes.)

Whenever a sudden increase of debility sets in in a fever patient, it is a common practice among experienced auscultators to examine the chest of the patient at once and carefully, especially its posterior and inferior portions; dulness of percussion there is often the only sign which marks the existence of splenization, i. e. livid or brown softening of the base of the lungs, for the induration of the parenchyma is often not great enough for bronchial respiration to be formed. If bronchitis be present it is generally marked by the presence of dry and moist bronchial rales. It is of the utmost importance frequently to percuss and auscultate persons laboring under bad fevers, however exempt they may appear from any affection of the chest.—(Andral.)

result of a metastasis, and hence is particularly apt to run into suppuration and mortification of the cellular tissue and superincumbent mucous membrane. These inflammations are generally confined to the region of the larynx, though they occasionally extend to the neighboring pharyngeal and æsophageal textures, or to the intermuscular cellular tissue of the neck. When chronic they are apt to cause hypertrophy, thickening, callous induration of the tissues affected, and consequent diminution of the volume or calibre of the larynx.*

d. ULCERATIVE PROCESSES.

These with rare exceptions are seated upon the larynx and trachea, and commonly result from affections which commence in

* *Diagnosis.* — According to Howship, boils may suddenly stop discharging, and uneasiness in the throat come on: in a few days respiration will be impeded, from some cause evidently seated in the larynx; extreme difficulty of breathing ensues, features exhibit great anxiety and distress, death follows, and suppuration of the sub-mucous cellular tissue of the larynx will be found. The exact diagnosis of this rare affection is not yet known, but Johnson says such cases are not rare: they commence as simple sore throat, but soon the symptoms become severe beyond their apparent cause, and the expression of distress is disproportioned to it.

Stricture of the larynx, from thickening of the mucous and submucous cellular tissue, is, according to Schoenlein, not uncommon in persons who have lingering, badly treated, or suddenly suppressed gonorrhœa. It has permanent and paroxysmal symptoms.

Paroxysmal.—Suddenly the patient has a sensation of contraction of the larynx, and cannot expand the chest sufficiently: inspiration long, with a shrill, trembling sound; trachea is stretched as far as possible, hence the neck is thrust forward; the eye is dull; pulse small, weak, and quick; the patient grasps hold of every thing, has intense anxiety, and beckons for aid. Such paroxysms are spontaneous and generally occur at night, but they may be induced by hasty speaking and eating; they last from fifteen minutes to many hours.

Permanent.—A remarkable alteration of voice, rarely a dull, bass tone, but more of a fistulous and hoarse one, with a peculiar snarling speech; the breathing conveys the idea that the air forces itself through a contracted place, which becomes more evident when the ear is placed over the larynx; constant difficulty of breathing; constant want of more air; a peculiar sensation in the chest, induced by the small quantity of air in the lungs; inspiration is long, because

the mucous membranes, which they destroy from within outwards. We have already referred to the catarrhal suppuration; to the sloughing ulcer, which with the apthous are degenerate exudative processes; to the variolous ulcers; to the typhous ulcers in general, and to suppuration with gangrene of the mucous membrane, arising from a primitive affection of the sub-mucous cellular tissue. We have yet to treat of suppuration of the perichondrium with necrosis of the laryngeal cartilages, and to tuberculous and cancerous ulcerations.

Here, however, we only propose to treat of syphilitic ulceration of the air passages. The epiglottis is the favorite seat of this affection, which frequently spreads to this organ from the soft palate and root of the tongue. They present the well known characters of secondary chancres, and often occasion entire destruction of the epiglottis and the adjacent mucous membrane of the glottis. This destructive process generally confines itself to these localities, leaving after cure a more or less considerable deficiency of the epiglottis, which is covered by thick, hard, white, tendinous and cord-like cicatrices, crossing each other in various directions, causing diminution in size and deformity of the affected parts. In single rare instances they extend to the larynx and trachea, causing sloughing and apthous ulceration of the mucous membrane, contraction of the sub-mucous tissue, brittleness and friability of the cartilages.

The walls of the air passages may be ulcerated from without; this occurs most frequently in the bronchi, owing to their neighborhood to softened tubercles and tuberculous abscesses.*

but little air enters the lungs; expiration is short, because there is too little air there; remarkable sensation of weakness and prostration; skin cool; pulse weak and contracted. At a later period emaciation comes on; difficulty of swallowing ensues, as the epiglottis becomes swollen, so as not to close the larynx; hence a portion of food or drink often gets into the larynx, where it excites cough, by which the foreign matter is again forced out, often through the nose. Frequent inclination to cough, cough dry and croupy at first, with lividity of the face; afterwards it becomes moist with glassy expectoration, streaked with blood at times. This disease almost always proves fatal, sometimes in five or six months, at others not for years; most common in men about thirty years old; death ensues during a paroxysm, which may seem allayed, but it returns suddenly and treacherously, and the patient quickly dies of suffocation.

* *Diagnosis.*—It is well known that, in an immense majority of

It is most frequent in the larynx, and there also attains its greatest intensity, and is followed by the most serious consequences.

It has attracted the particular attention of pathologists, under the name of œdema glottidis. In rare instances it extends to the mucous membrane of the posterior tracheal wall and upon the pharynx.

This disease, in those cases which strictly belong under this head, consists in an infiltration of the sub-mucous cellular tissue and the mucous membrane with a colorless or pale yellow serum. The seat of œdema glottidis is in the mucous membrane of the epiglottis, of the aryteno-epiglottic folds, of the vocal-chords, and ventricles. When fully developed, a transparent pale yellow fluctuating swelling is formed, which, according to its size and extent, diminishes or entirely closes the aperture of the glottis.

Œdema of the glottis may occur either as an acute or chronic affection, and is associated not only with all the previously mentioned inflammatory diseases of the laryngeal mucous membrane, but also with many irritations of this and the adjoining parts. It also arises as a sequel of catarrhal inflammations, more frequently from those of an exudative or exanthematic character, and from inflammation of the sub-mucous cellular tissue. It follows the typhous and all other ulcerative affections of these parts, such as the tuberculous, cancerous, &c.

It is important to be prepared for and to be able to diagnose those cases, for they arise from unknown exciting causes during

instances, ulceration of the larynx is of a tuberculous nature, and secondary to more or less extensive tuberculosis of the lungs. Yet Schoenlein admits a rheumatic inflammation and ulceration of this organ, comparatively frequent in those much exposed to cold and wet, such as washerwomen, &c.; the pains are said to be more frequently *piercing*. Also, a laryngo et tracheo-phthisis hysterica, most common in females who have frequently suffered from *globus hystericus;* in this the pains are commonly *contracting*. When the ulcers are seated high up in the larynx, there often is a flaming redness of the fauces and tonsils; when the vocal chords, ventricles, or arytenoid cartilages are ulcerated, there is hoarseness, alteration, or complete loss of voice; the larynx is not often painful to pressure, except an ulcer is seated on the crico-thyroid ligament; when the posterior surface of the larynx is ulcerated, difficulty and pain on swallowing is experienced. The cough is peculiar and characteristic;

the progress of the above mentioned affections of the laryngeal mucous membrane, of the soft palate, tonsils, and neighboring tissues, quickly attain a high degree of development, and cause rapid suffocation, though the primitive disease may appear very slight.

It is important to distinguish the sero-purulent infiltration from simple œdema glottidis, as the former is always the result of an intense inflammatory affection of the sub-mucous cellular tissue.*

f. GANGRENE OF THE AIR PASSAGES.

It occurs in two distinct forms: 1st. As a circumscribed eschar of the mucous membrane of the air passages, which immediately attacks the subjacent cellular tissue, in which, however, it may also occur primitively. 2d. As diffused gangrenous colliquescence of the same tissues. It arises under conditions similar to those causing gangrene of the lungs, and is at times asso-

it does not come from the depths of the chest, but is a tussis laryngea, being effected by the muscles of the throat and larynx only; cough may be absent, and its place supplied by a mere hawking up of pus and mucus; cough, however, may come on in violent paroxysms, in which the rima glottidis becomes contracted, with violent contracting pain in throat, a croup-like paroxysm, and danger of suffocation. The root of the tongue is often thickly coated on one side only, viz., on that corresponding to the seat of the ulcer; this coating may extend in a broad stripe from the root to the tip of the tongue; when present it is characteristic.

* *Diagnosis.*—The symptoms of œdema glottidis are so similar to those of croup, as to have led some physicians to believe that this latter disease is not so rare in adults as is usually supposed. Cruvelhier thinks that it is always preceded by and attended with inflammation and tumefaction of the throat, and fever; the voice becomes remarkably rough; patient complains of difficulty in the throat, and of the entrance of air during inspiration, while expiration is effected with comparative ease; in the course of the disease respiration becomes extremely difficult, rapid, and hissing; dyspnœa with extreme danger of suffocation set in; voice is extinct, or else hoarse, sharp, and croupal; by auscultation we hear a shrill, trembling, and whistling sound, as if the air met with an obstacle in its passage through the larynx. If the finger be passed rapidly and dexterously through the fauces, a roundish, painless, but soft swelling will be felt. Asphyctic paroxysms ensue; the face becomes swollen and livid; the eyes project from their sockets, and the patient often dies suddenly of suffocation.—(SCHOENLEIN.)

ciated with it. It generally occurs in a tissue in some way previously diseased, being however an accidental rather than a necessary termination. We have said that the circumscribed form arises in the train of inflammation of the perichondrium, from tuberculous laryngeal phthisis, typhous ulceration, and laryngeal croup. The diffused variety is far more frequent in the mucous membrane of the bronchi. In it, we find the membrane converted in various places into a soft, moist, shaggy, friable tissue, of a dirty brownish-green color, giving out the peculiar odor of gangrene, and occupying either a large extent of surface, or only spots of various dimensions. The bronchi are filled with a similarly discolored, frothy, fœtid, sero-ichorous fluid. It most commonly accompanies gangrene of the lungs.

B. DISEASES OF THE CARTILAGINOUS SKELETON OF THE AIR PASSAGES.

a. INFLAMMATION OF THE PERICHONDRIUM OF THE LARYNGEAL CARTILAGES (PERICHONDRITIS LARYNGEA).

We occasionally have opportunities of noticing in the cadaver a peculiar form of ulceration of the larynx, which without doubt is the consequence of an inflammation primitively seated in the perichondrium, which is detached either in single circumscribed spots, or more frequently from both surfaces of the whole cartilage; a quantity of pus is found collected in a membranous sac both anteriorly and posteriorly between it and the cartilage. The cartilages are rough, shreddy, necrosed, and perforated, and either laid bare by circumscribed abscesses, or else they lie entirely free, detached, discolored, attenuated, softened, either entire, or crumbled into several small pieces, the whole being situated in a large purulent cavity. This abscess may open and discharge its contents into the larynx, pharynx, trachea, or through the skin.

The disease attacks the cricoid cartilages by preference; it is generally considered of a rheumatic nature, and has been described as a rheumatic laryngeal phthisis. Still it may follow the acute exanthems, especially variola, or may arise from an abuse of mercury.

b. INFLAMMATION AND SOFTENING OF THE EPIGLOTTIS.

The epiglottis occasionally becomes the seat of chronic

inflammation, by which this organ is transformed into a dense, rigid, fibro-cartilaginous tissue, a change always attended by contraction and deformity.

In contrast with this, softening may occur, similar to that which affects the yellow coat of the arteries, and which possibly may be also the result of inflammation. In this condition, the epiglottis loses its elasticity, becomes soft and brittle, assumes a dirty yellow color, and wastes away.

c. OSSIFICATIONS OF THE CARTILAGES.

In the later years of manhood, the cartilages of the larynx, in the male, almost always become ossified; hence this change only deserves particular attention when it commences at an unusually early period, is finished in a short space of time, or extends itself to parts not usually its seat. They ossify in the following order: 1st, the thyroid; 2d, the cricoid; more rarely and at a later period the tracheal, and finally the bronchial rings. This ossification is a true transformation of the cartilages into bone, not a mere deposition of osseous particles into their tissue. It occurs either spontaneously, or may be induced by an inflammatory vascular activity of the perichondrium, and is frequently noticed in and about the seats of ulcers, especially those of a tuberculous character. Fracture and injuries of the cartilages often prove exciting causes of the deposition of osseous callus. The new bone, when developed in the course of laryngeal phthisis, may become the seat of caries and necrosis; it then frequently happens that fragments of bone may be coughed up, which however can be readily distinguished by their peculiar structure from calcareous concretions.

In rare instances not only do the most minute bronchial cartilages become ossified, but even the walls of the smallest terminal twigs. We then find rigid tubes passing through the lungs, which do not collapse when cut across; and if the finger be passed over the incised surface, it detects prominent, sharp, sand-like granules. This condition only occurs in very aged persons.

The epiglottis is never truly ossified, but is sometimes, though rarely, the seat of bone-earthy concretions, especially when in consequence of inflammation it loses its normal structure and becomes changed into a fibrous tissue. These concretions necessarily cause various kinds of deformity.

d. HETEROLOGOUS FORMATIONS OF AIR PASSAGES.

These become of especial importance when they project into the cavity of the air passages in the form of broad or pediculated vegetations, causing different degrees of diminution in their calibre. These vegetations occur almost exclusively in the larynx, and, under the common head of laryngeal tumors, are generally divided and treated of according to their external characters. Taking their internal structure as our guide, we shall reduce them to the following species, which are also found in the substance of, or beneath, all other mucous membranes.

1. *Epithelial Formations.*

These occur in the form of rounded cauliflower-like or warty vegetations, varying in size from that of a hemp seed to that of a hazel-nut, or even larger. They are attached to the mucous membrane by a short neck, have a laminated structure, and consist almost wholly of exuberant epithelial cells, and prolongations of delicate vessels from the mucous membrane. Their favorite seats are the vocal chords and arytenoid cartilages; still they have been found attached to the inferior surface of the epiglottis and to the cricoid cartilage. As they may spring from a cancerous basis, they are frequently of a malignant nature; but they also occur in a benign form in persons of various ages who have suffered from frequent attacks of catarrh or croup. They are the most common of all laryngeal tumors.

2. *Cellular or Mucous Polypi and Condylomatous Excrescences.*

These generally grow upon, or by the side of previously existing ulcerations, though they may also arise from healthy mucous membrane. They form hard, spongy, bluish-red bodies, of the size of pins' heads, hemp seeds, or peas; lying side by side, and covering large sections of the laryngeal mucous membrane, particularly that which clothes the vocal chords. They are in all probability of a syphilitic origin, and when, as occasionally happens, they arise in connection with tubercular laryngeal phthisis, they lead us to suspect a combination of the syphilitic with the tubercular dyscrasia.

3. *Erectile Tumors.*

Erectile tumors occur in the form of soft, broad-based, dilatable vegetations, or as morbid growths from the free extremities of mucous polypi. The first variety not unfrequently springs from a cancerous basis.

4. *Fibrous Tumors.*

These tumors are very rarely found in the sub-mucous cellular tissue of the larynx, but are of more frequent occurrence in that portion of the pharyngeal mucous membrane which covers the posterior laryngeal wall. In this situation they are met with not only of their usual inconsiderable size, but at times are distinguished for their great volume, and by their adhesion to the perichondrium, reminding us of the large pharyngeal polypi that grow from the sub-mucous periosteum at the back of the throat.

5. *Cancer.*

As has already been remarked, this morbid change not unfrequently forms the basis of exuberant epithelial formations and erectile tumors. In addition it may occur in the fibrous form in the larynx, in the medullary form in the sub-mucous cellular tissue, and, what is extremely remarkable, cancerous degeneration of the arytenoid cartilages occasionally takes place. These tumors project into the laryngeal cavity in the form of knobby, rounded protuberances of various sizes, and diminish its volume to a greater or less degree. They for the most part prove fatal while yet in their crude state, but at times pass into the stage of softening and degenerate into cancerous ulcers.

Cancer may also occur in the trachea and bronchi, but only as a secondary disease. Thus in rare instances large medullary cancerous masses open their way from the neck into the trachea, or from the posterior mediastinum into the bronchi, into which esophageal cancer may also perforate and grow. We occasionally notice a malignant degeneration of the fibrous bronchial sheath, which at times extends from a bronchial trunk to a greater or less extent along its ramifications. We then find the walls of the bronchi thickened and rigid, their volume diminished, their inner surface tuberous and uneven. This degeneration seems to spring from a primitive affection of one or several bronchial glands.

6. *Tuberculosis.*

Tubercles are of very common occurrence in the air passages, but are not equally frequent in all parts. They are most frequent in the larynx, extremely rare in the trachea and larger bronchi, and again become frequent in the terminal bronchial twigs. By their softening they cause tuberculous ulcerations of the affected tissues, and according to their seat they produce laryngeal, tracheal, or bronchial phthisis.

a. TUBERCULOSIS OF THE LARYNX is so rarely a primitive independent disease, that its occurrence as such has been positively denied. It is developed in the train of tuberculous disease of the lungs, and generally only when the latter has progressed into pulmonary phthisis. The seat of the tubercles is almost constantly and exclusively confined to the mucous membrane and sub-mucous cellular tissue above the musculus transversus, and the adjacent arytenoid cartilages; still in exceptional cases they are met with upon the epiglottis and in other parts. The tuberculous matter is either deposited in the sub-mucous cellular tissue in the form of gray granulations, or is infiltrated into the mucous membrane as a yellow, cheesy, friable substance; in both instances, but especially in the latter, it passes rapidly into softening and ulceration. The softened gray granulations form little ulcers of the size of a mustard seed or lentil, with thick and hardened edges; they then coalesce and form secondary, irregular ulcerations with indented, serrated edges, and a cellulo-callous base, both of which may become the seat of a secondary deposition of tubercles. The yellow, cheesy infiltration softens in and together with the mucous tissue, forming larger, extremely irregular, gnawed, and, as it were, fissured ulcers. These are attended with evident inflammatory reaction, viz., redness, injection, swelling, œdema of the surrounding tissues, and apthous exudations in their immediate neighborhood.

These ulcers enlarge in consequence of the softening of secondary tubercular depositions in their edges, bases, and immediate neighborhood. They increase in depth as well as breadth, and gradually extend over the whole larynx, viz., upwards to the epiglottis, beyond it upon the soft palate and root of the tongue, and downwards into the trachea, causing suppuration and mortification of all the fibrous tissues and cartilages. They may even perforate the larynx from within outwards, and open into the surrounding cellular tissue, thus causing universal emphysema.

Secondary tubercular ulcerations at times assume a peculiar aspect from a condylomatous development of the mucous membrane of their edges, or of those of the muco-membranous insular patches which frequently remain on the bases of the ulcers during their enlargement. It is not improbable in these cases that the tuberculous dyscrasia is complicated with the syphilitic.

These ulcers may heal up in rare instances, when favored by the necessary general conditions, but always leave more or less callous, misshapen cicatrices, proportionate in size and depth to those of the previous ulcerations, and cause a greater or less degree of contraction and deformity of the larynx. We must be on our guard, however, not to imagine every cicatrix existing in the larynx or trachea adjacent to true tuberculous ulcerations to be the remains of similar ulcers.

b. TUBERCULOSIS OF THE TRACHEA is extremely rare, for the laryngeal affection scarcely ever extends itself, even upon the superior portion of this tube. In laryngeal phthisis, however, nothing is more common than for us to find small ulcers upon the tracheal mucous membrane, often, too, in such numbers that they become confluent and cover the whole of its surface. It is to these cicatrices which we just now referred, when we warned against mistaking them for the scars of tuberculous ulcers. They are small and shallow, generally of an oval, occasionally of a linear form, with concave bases, and often so very superficial as only to be observed when the light is thrown obliquely upon them. They are either clean or covered with a cream-like, dissolving exudation of a croupy character, and are surrounded either by a fiery redness or a sharply circumscribed, red areola. They are seated for the most part on the posterior tracheal wall; frequently they extend into the bronchial trunks, being found in greater numbers on the right or left side of the trachea and corresponding bronchus, according as one or the other lung is predominantly or exclusively the seat of tubercular disease. They also occur with considerable frequency in the pharynx and upon the mucous membrane of the mouth, but are in no wise connected with tuberculous ulcerations, and may be at a glance recognized as exudative apthous ulcers, which, however, frequently associate themselves with florid laryngeal phthisis. When the tuberculous disease is stationary, or makes actual retrograde movements towards a cure, these apthous ulcers heal, leaving delicate, white, shining, radiated, star-like cicatrices.

c. BRONCHIAL TUBERCULOSIS occurs in the mucous mem-

brane of the tubes, which may be infiltrated with yellow, fatty, cheesy, tuberculous matter to such a degree as to seem almost entirely converted into it. The tube itself becomes enlarged, its cavity gradually and completely filled with tuberculous matter, while its fibrous sheath undergoes a fatty infiltration and becomes callous and thickened.

This variety of degeneration is at times a secondary process; it is then developed in those bronchi which open into tuberculous vomicæ, and is dependent on pulmonary phthisis.

Primitive bronchial tuberculosis is a more important disease. As we have already mentioned, it is an affection of the terminal bronchial twigs, arising in these originally, and from them spreading along those of a larger size. Like pulmonary tuberculosis it occurs most frequently in the superior lobes, but unlike it is often found in their periphery. At times it attacks a large portion of the bronchial tree, and should we then make a section of the lung, we find the parenchyma traversed by numerous thick-walled, dilated bronchial tubes, which are filled with cheesy, tuberculous matter.

Bronchial tuberculosis is often complicated with a fatty gelatinous, or fatty, cheesy, tuberculous infiltration of the parenchyma of the lungs; sometimes, on the contrary, it is a perfectly uncomplicated and independent disease. In the latter case the obstruction of the tubes is followed by obliteration of the air cells, and atrophy of that portion of the pulmonary parenchyma supplied by them with air. If we then make a section of the affected part, we find the obstructed bronchi contracted into the form of ribbons, which ramify through a dense elastic tissue.

The tuberculous matter softens, destroys the bronchial walls, and creates abscesses in the adjoining pulmonary parenchyma. These abscesses, the first step of which is the destruction of the bronchial walls, are by no means so frequent as those arising from the softening of pulmonary tubercles. This mode of softening is most frequent when simultaneous tuberculous infiltration of the lung exists. Another, but a rarer change is, the transformation into chalky concretions. This metamorphosis is most frequent when the tube has been entirely obstructed with tuberculous matter, and the parenchyma of the lungs, to which it leads, has in consequence become atrophied. Under favoring constitutional causes, the matter is changed into a cheesy pap, which, instead of softening, begins to thicken, and is finally changed into a chalky mass, around which the bronchial tube contracts, and becomes atrophied.

Bronchial tuberculosis occurs as a primitive disease during the years of childhood; is generally associated with the tuberculosis of the other tissues peculiar to this period of life, but most frequently with the disease of the bronchial glands.

It presents many resemblances to tuberculous deposits on the fallopian and uterine mucous membranes.*

7. *Foreign Bodies in the Air Passages.*

Under this head we must consider:

1. The various diseased products of the entire mucous membrane of the air passages, such as blood; various quantities, often very great, of mucus of different qualities, viz., gray, pearl-colored, transparent, colorless, watery, tough, glassy, cream-like, whitish, yellow, and purulent; actual pus, membranous concretions or croup-membranes, ichorous fluids, tubercles, tuberculous pus, necrosed fragments of cartilage, ossified bronchial cartilages, &c.

**Diagnosis.*—Tubercular ulcerations of the larynx are more common in males than females; from Louis's statistics we learn that in eighty cases of phthisis in females they were found nineteen times, or in about one-fourth; while in one hundred and thirteen cases in males they were found forty-four times, or in about two-fifths. It is not sufficiently known that in some cases the affection of the larynx forms not only the first evident commencement of phthisis, but so masks the disease of the lungs that cough, dyspnœa, hæmoptysis, emaciation, and hectic fever, seem to spring from it, and not from the lungs, which are apparently sound to the mere symptomatologist. But nothing is more uncommon than laryngeal phthisis independent of pulmonary phthisis. In upwards of five hundred non-tuberculous subjects dying of various chronic diseases, Louis did not find one example of laryngeal ulceration; as chronic laryngitis with ulceration is found in from one-fourth to two-fifths of all cases of consumption, the conclusion is irresistible that in an immense majority of instances this affection is but a part of consumption. If we find a case of chronic laryngitis with emaciation, hectic, &c., the probabilities amount almost to certainty that there are softened tubercles in the lungs. Louis says that moderate pain of limited duration in the region of the larynx, coupled with more or less marked alteration of the voice, signifies superficial ulceration of the larynx; whereas severe and continuous pain with persistent loss of voice results from deep ulceration. But this affection is generally painless; commonly there is but a little heat and constriction in the part, coupled with more or less hoarseness or loss of voice. According to Andral, ulcerations of the mucous membrane lining the ventricles and covering

2. The products of diseases external to the air passages, and which enter either through natural or artificial channels: as blood in various but generally very large quantities, from the bursting of an aneurism; bronchial froth, composed of serous and frothy fluids from the parenchyma of the lungs; pus and ichor, flowing from abcesses of the pulmonary tissue, bronchial glands, or vertebræ. In consequence of the direct corrosion of a bronchial trunk, pus may be discharged from the cavity of the pleura; the same may happen with an hepatic abscess; cancerous ichor, from malignant disease of the esophagus; tuberculous matter; bone-earthy and stony concretions, including chalky transformations of tubercles; catarrhal mucus; acephalocysts, which, according to Portal, get into the air passages from the lungs, liver, and thyroid body.

3. Foreign bodies which,

a. Enter into the air passages from the pharynx, esophagus, or even from the stomach and intestinal canal, through natural or unnatural communications. Among these belong fluids which

the chordæ vocales often cause an alteration of the voice, which is not greater than when these parts are red and slightly swollen: a hoarse and rasping voice generally depends upon a considerable tumefaction of the mucous membrane of the ventricles: the destruction of one chorda vocalis may cause either hoarseness or loss of voice; of both, always complete aphonia. Fixed pain at the upper part of or immediately above the thyroid cartilage, difficulty of swallowing, and escape of drink through the nose, announces ulceration of the epiglottis, provided the throat and tonsils be healthy.

Apthous ulceration of the trachea is commonly attended by no special symptom; in a single instance only did Louis observe a slight feeling of heat and obstruction behind the sternum. It is not known if the successive crops of apthæ upon the trachea occur simultaneously with those which take place so frequently in the mouths of phthisical patients.

Tuberculosis of bronchial glands.—According to Rilliet and Barthez, serious results from pressure of enlarged bronchial glands occur less frequently than might have been expected: complete obliteration of a bronchus from the same cause is also rare; but compression of the trunk or branches of the pneumo-gastric nerve is common, and is attended with a spasmodic cough resembling hooping cough, alteration of the voice, even total aphonia, and very distressing attacks of asthma. When enlarged bronchial glands compress the superior vena cava, they may occasion, 1, œdema of the face; 2, dilatation of the veins of the neck; 3, a livid color of the face; 4, hæmorrhage into the cavity of the arachnoid. By compressing the pulmonary veins, 1, hæmoptysis; 2, œdema of the

flow into the cavities by ulcerated or cicatrized openings, or from strictures; ascarides, which sometimes find their way into the pharynx and thence into the glottis, and are said to have caused death by suffocation.

b. Articles of food, pieces of gristle, tough meat, which become wedged into the glottis in consequence of inflammation and degeneration of the muscular parts of the pharynx, of swelling of the tonsils, haste and inattention during the act of deglutition, especially in idiots, or when coughing, laughing, sneezing, &c. These accidents are still more apt to occur when atrophy, rigidity, deformity, and consequent insufficiency of the epiglottis, are present.

c. Foreign bodies which accidentally obtain entrance into the glottis without an intentional act of swallowing, and which fall, or are forced into the trachea or bronchi. Among these we have articles of the most various kinds, fruit-pits, coins, pins, natural

lung may result. By compressing the bronchi, they occasion, 1, loud, very persistent, sonorous rales, which are sometimes very singular in their tone and character; 2, feebleness, or absence of respiratory murmur in parts of the lungs; 3, they will convey sounds which are found in the bronchi in their normal condition, but not transmitted to the ear, such as prolonged expiration, bronchial respiration, &c., although the lung may be almost or altogether healthy; 4, if pulmonary lesions exist, their stethescopic signs will be exaggerated—thus, crude miliary tubercles will give rise to bronchial or cavernous respiration, or to pectoriloquy; and if they have begun to soften, or are attended with slight bronchitis, distinct gurgling may be heard; 5, the sounds arising from an affection of one lung may be transmitted to the sound side, and thus excite a suspicion of a double lesion.

These stethescopic phenomena are heard especially at the upper and posterior part of the lungs; they vary very much: thus, bronchial respiration will be heard one day, and give place on the next to simple prolonged expiration, while on the third cavernous respiration may be present; in fact, feeble respiration, prolonged expiration, bronchial breathing, cavernous respiration, pectoriloquy, gurgling, and mucous rhonchus, may alternate with, or succeed each other, without any regularity. This changeableness of the stethescopic signs, if attended with persistent dulness of percussion between the scapulæ, render it almost certain that there is tuberculosis of the bronchial glands; if cough, emaciation, fever, and sweats, are also present in a child from three to four years of age, scarcely a doubt can exist as to the nature of the disease.—[Rilliet and Barthez.]

or artificial teeth, nails, pebbles, pieces of glass, &c. In favorable cases they are soon ejected by coughing; in others they remain, causing not only inflammation of the air passages themselves, but pneumonia, suppuration of the lung, injuring the walls of the tubes in various ways, and occasionally penetrating through them into adjacent canals and blood-vessels. Here we may be permitted to refer to the following rare case: A little boy accidentally sucked an arrow from a blow-gun. It was drawn with its feathered portion downwards into the trachea, and from thence into the left bronchus. In consequence of hemorrhage from the lungs, he died in twelve days with symptoms of pneumonia. Bronchitis, especially of the left side, and hepatization of the left inferior lobe, were found after death. The arrow was lying loose in the left bronchus; its point upwards, and to the right. Near the junction of this bronchus with the trachea was a very small incised opening, and from this the perforation extended into the adjacent arteria innoninata. This mischief was caused by the point of the arrow being forced during the paroxysms of coughing against the right side of the trachea, which of course lay opposite in the axis of the left bronchus.

Key states an interesting and important fact, namely, that the majority of these foreign bodies fall into the right bronchus, owing no doubt to its greater size, the more obtuse angle at which it is given off from the trachea, and to the greater force of the current of air flowing into it at every inspiration. We are here reminded of the circumstance of the right lung in new-born children becoming more quickly and perfectly inflated than the left.*

* *Diagnosis.*—According to Hawkins and Ryland, in by far the greater number of cases the foreign body continues to be movable in the trachea; it may be distinguished from laryngitis by the absence of fever at first; by the very sudden manner in which the symptoms come on; by the intermissions in the difficulty of breathing, which sometimes continue an hour or two; by the noise which is heard occasionally when the foreign body is impelled against the vocal chords; by the excessively violent cough which follows this occurrence; but more particularly by the chief difficulty of breathing being during the time of expiration, while in laryngitis the chief difficulty is in inspiration.

When it is located in the larynx below the glottis—in the situation of the cricoid cartilage, the severe paroxysms of coughing are absent—hoarseness may be wanting in two cases out of three, but in all there is a sense of soreness and uneasiness in the spot where the foreign body is fixed; a noise is heard either during expiration

II. ABNORMAL CONDITIONS OF THE PLEURA.

1. DEFICIENCY AND EXCESS OF DEVELOPMENT.

The pleural sacs are absent when there is a perfect deficiency of the organs of respiration; the thoracic cavities then being filled with a dense fibro-cellular tissue. A partial deficiency of the pleura exists when, owing to the absence of the diaphragm, its serous membrane is continuous with that of the peritoneum.

Excess of development always occurs in the form of duplication of the costal pleura, except in the case of double monsters, in which the cavities of the chest are found double in a greater or less degree, and when both lungs are found seated in one large common cavity. It is extremely rare, especially when contrasted with the frequency of congenital duplications of the peritoneum. On account of its rarity, as well as the conditions under which it

or inspiration, or both, and in all the patient asserts that something has been swallowed.

When a plug of mucus obstructs a bronchus there may be a feeling as if something was lodged there, with a sense of great heat at the part; the clot may be large, weigh an half ounce, be yellow, opaque, viscid, and of a consistence intermediate between usual mucous sputa and false membrane: after its expulsion the sense of warmth at the spot may be changed to a painful heat, which may last many hours. When a plug of mucus obstructs a bronchus we all at once cease to hear the normal vesicular murmur, or the rales if bronchitis be present; the part of the chest above the obstruction retains its sonorousness; at the same time the patient is seized with greater or less dyspnœa; after a violent fit of coughing the plug may be expelled, the dyspnœa cease, and the respiratory murmur return; in rare cases the difficulty of breathing increases, suffocation becomes imminent, and patient dies of asphyxia.—(Andral.)

In one of Andral's cases the patient, in the midst of a violent fit of coughing, was seized all at once with extreme difficulty of breathing. The remainder of the day and all the night he had orthopnœa and almost continual efforts at coughing. The following morning there was imminent asphyxia, his face was swollen and violet; extremities livid, and pulse nearly extinct: with difficulty and panting he entreated them to relieve him of an enormous weight which he said he felt on a level with the right mamma, and which was smothering him. The sonorousness of the chest was not diminished—from the clavicle to below the breast, both before and behind, neither respiration nor rale was heard, although the chest was elevated with force; on opposite side there was exaggerated, puerile respiration.

arises, we may be allowed to allude to an increase of the right pleural sac, which as yet has not been noticed or described by any previous observer. This extends in the form of a fold from above and outwards, downwards and inwards, its free edge embracing the arch of the vena azygos, and lying in a supernumerary fissure, which divides the superior lobe of the lung into two parts.

2. ANOMALIES IN SIZE AND FORM.

The size or capacity of the pleural sac is always proportionate to the congenital or acquired volume of the lung ; thus large lungs have spacious pleuræ, those of a less size have smaller ones. In emphysema, dilatation of the serous membrane takes place ; in atrophy, diminution of it occurs. The pleural cavities may be enlarged to various extents by accumulations of gaseous or liquid fluids in them, and be lessened by an increase in size of the pericardium or peritoneum ; also by heterologous deposits in the mediastinum and by deformities of the walls of the chest.

The form of the pleura is regulated by that of the bony thoracic walls ; hence we must refer our reader to what has been said concerning anomalies in the shape of the thorax.

3. DISEASES OF THE PLEURA.

a. Hyperæmia.

A continuous increased flow of blood according to its extent occasions either a local or general opacity, also thickening or hypertrophy of the pleura, or finally the development of anomalous cartilaginous and bony substances in its texture or that of the sub-serous cellular tissue.

Congestion may also cause increased secretion, which, according to its character and the condition of the blood in general, may result in the transient or permanent accumulation of various quantities and qualities of serous fluids in the chest. [*Hydrops pleuræ or hydrothorax.*] In rare instances it may lead to hemorrhage in the pleura or hæmo-thorax.

6. *Inflammation of Pleura. Pleuritis, Pleuresia.*

This is the most frequent disease of the pleura : it generally appears as an idiopathic and primitive disease, and most frequently

is of a rheumatic nature. It may arise in consequence of injuries, or concussion of the chest, from exposure of the pleura to contact with atmospheric air, pus, gangrenous ichor, &c., which may reach it from within through the air passages; from the extension of inflammation and other affections of the neighboring tissues and organs, especially the lungs; or finally it occurs as a secondary or metastatic disease, and is often associated with inflammations of other serous membranes, particularly the peritoneum and pericardium. In general it bears a marked croupous or exudative character.

Pleurisy is either general, and then the disease for the most part is least severe upon the pulmonary pleura, or it is partial and circumscribed. It may also in either case be acute or chronic. As every thing which has been said of serous membranes in general holds good with regard to inflammation of the pleura, we shall here confine ourselves to the consideration of important peculiarities.

The exudations occur in all the varieties already described, two forms of which, the *purulent* exudation (Empyema) and the *hemorrhagic*, are of very frequent occurrence and of great importance. Acute, but more particularly chronic pleurisies deposit an exudation, the quantity of which is increased paroxysmally; when the inflammation is general it often becomes enormous, the fluid portions weighing as much as eight, ten, sixteen, and even twenty pounds and over. The thorax in these cases becomes dilated in a more or less striking manner; the intercostal spaces are enlarged from paralysis of their muscles, and the depressions so evident in the healthy state are completely effaced. The diaphragm is pushed down into the abdomen, the mediastinum and heart are thrust to the opposite side, and this way diminish its capacity. The lung itself is compressed in proportion to the quantity of fluid present; if no old adhesions exist to prevent it, it is always forced upwards and inwards, toward the mediastinum and spine; it may be reduced to one-fourth, sixth, or even the eighth part of its normal volume; it loses its natural arched form, and becomes like a flattened cake. Its substance is of a pale-reddish, or bluish-brown, or leaden-gray color; it is tough, leather-like, and destitute of blood and air. Its external surface is covered with a plastic effusion, which extends over the lung from the adjoining costal pleura; thus the lung is, properly speaking, shut out from the cavity of the sac formed by the pleuritic exudation. If adhesions already exist as the remains and conse-

quences of previous inflammations, then these will, in proportion to their nature and the tenacity of the tissues of which they are composed, prevent in some measure the above described dislocation of the lung. In partial pleurisies, this dislocation and compression of the lung is prevented in a degree and manner proportionate to the situation and extent of the adhesions.

A purulent exudation most frequently is found in weak cachectic individuals, and in those having a peculiar predisposition to the formation of pus. From the intensity and frequent renewal of the inflammation, it quickly leads to general debility and pyogenous admixture of the blood. The effused pus not unfrequently degenerates to ichor with disengagement of gas, so that from its decomposition a pneumo-thorax is added to the purulent effusion. Occasionally, suppuration of the walls of the chest with or without caries occurs, leading to a spontaneous discharge of the contained fluids, &c., by an external opening; or, on the other hand, this same process may take place on the surface of the lung, opening into and causing suppurative inflammation of the bronchial tubes, and giving rise to ejection of purulent matter from, and entrance of air into, the pleural sac.

Among the partial pleurisies, we may mention those occurring near the apices of the lungs in consequence of pulmonary tubercles, those in the lower sections of the pleural sac and pleura diaphragmatica, those of the mediastinal lamina, and finally those occurring in the interlobular fissures of the lungs.

Plastic exudations, the result of simple inflammatory processes, either acute or chronic, are transformed into cellular tissue of varying density or to cellulo-serous or fibrous texture, presenting various peculiarities as to their form and extent. When they are the product of general pleurisies they coat over the whole of the costal and pulmonary pleura; occasionally, however, they are only found in single spots of various sizes, in the form of circumscribed insular patches, surrounded by healthy serous membrane; they may, or may not, adhere to the opposite pleura. This cellular tissue of new formation appears either as dense and rigid, or as long, lax, movable, filamentous adhesions, according as a greater or smaller quantity of liquid effusion was present either originally or during their organization. They exist in the form of general cellular adhesions, when the whole costal and pulmonary pleura are attached, or as partial ones when only a portion of them adheres. They may become the seat of fresh inflammatory processes; but, as Laennec has remarked, they generally circum-

scribe and limit subsequent pleurisies. In general dropsy, or in the hydropic diathesis, they may become the seat of a serous infiltration.

This tissue is at times met with in the form of delicate flocculi or bundles, scattered over the costal and pulmonary pleura without forming adhesions, or even corresponding in situation. In most instances, however, previous cellular adhesions appear to have been torn asunder by the motions of the lungs and chest, and we then find cellular patches on both pleuræ, which correspond in position and shape; they may rise conically from a broad base, or extend into long string-like prolongations.

If, during the organization of the plastic exudation, a watery effusion was present in the pleura, sufficient in quantity to prevent the two layers from coming in contact, then the cellular tissue receives a serous coating, and the pleura is found covered with a second more or less perfectly adherent serous membrane of new formation.

If the exudation, under the same circumstances, was of some thickness, it will be converted into a smooth, bluish-white, fibrous lamella, which either covers the whole pleura or is only attached to it in single places, in the form of sharply circumscribed tendinous patches, with either thick and abrupt or sloping and shelving edges. Even in the first case these lamellæ are not of a uniform thickness in all parts, but present an areolar, perforated, and sieve-like appearance. Under such circumstances, it at times happens, after the quantity of the fluid effusion has diminished, that the lungs push through the parts where the false-membranes are thinnest, and project through their meshes in the form of teat-like vesicular appendices.

Chronic inflammation of the pleura, especially when it creeps slowly among the organizing exudations, occasions very important metamorphoses of the substance of the pleura and of the products of inflammation. It gives rise with great frequency to profuse hemorrhagic or serous effusions at the same time that it deposits remarkably thick and solid coagulæ, which are gradually transformed into very dense resistant fibrous swathes. Besides dilatation of the thorax, corresponding in degree to the quantity of fluid effused, and the already mentioned dislocation and compression of the lung, we have, as a result of general pleurisy, thick pseudo-membranous pleural sacs of new formation. These adhere both to the costal wall and the lung; but the parietal laminæ is generally the thickest, often measuring four, six, eight, ten lines, or even an inch in thickness.

If under such circumstances a quantity of the serous effusion be absorbed, then the lung, by reason of the thickness and resistance of its covering of fibrous false membrane, is completely prevented from recovering its former size, or may do this only very slowly and gradually. In the latter case the two lamellæ of the fibrous exudation approach each other by degrees, and after the complete absorption of the serum may come together and coalesce. The thorax in such cases, as Laennec has already shown, becomes permanently contracted; this contraction varies in degree from a scarcely perceptible flattening to a very evident ditch-like depression, and exerts a very marked influence upon the shape of the body. In these latter cases the greatest sinking in takes place as a general rule in the neighborhood of the sixth, seventh, and eighth ribs, and the lateral wall of the chest presents a concavity from the axilla to the edge of the ribs. The thorax appears contracted as well in its periphery as in every one of its diameters; the ribs sink in to such an extent as to touch, or even overlap one another. The thoracic muscles are found emaciated; the intercostals in particular are contracted to an extent proportionate to the degree and continuance of the paralysis they have undergone, and finally are transformed into a cellulo-fibrous structure. The dorsal spine inclines from its normal position, with a lateral curvature towards the healthy side; the shoulder of the affected side sinks down in proportion to this curvature. The lumbar portion of the spine forms a curvature proportionate to that of the dorsal, but towards the opposite side, and hence the pelvis of this side assumes a higher position, occasioning an apparent shortening of the corresponding leg; then the form of the hips and buttocks and the carriage of the body attains some similarity to that which occurs in coxalgia.

There are various causes for this contraction of the thorax: among these are, the pressure of the atmosphere on the chest, coupled with incapability on the part of the lung of returning to its former size with a rapidity equal to that of the absorption of the serous effusion, owing in part to the binding down of the organ by the fibrous swathe, but principally to the loss of its elasticity and power of expansion, from the long continued pressure it has endured. There is also a predisposition on the part of the costal lamina of the fibrous exudation to contract and increase in density, and a similar inclination to contract on the part of the cellulo-fibrous tissue which has taken the place of the atrophied intercostal muscles.

Contraction of the thorax may, even when the result of general pleurisy, affect only one portion of the chest, as the upper section, the lower remaining either absolutely dilated or only relatively so when compared with the contracted part. Thus it may happen, for instance, after a partial absorption of the effusion, that adhesions may take place superiorly, while below this is prevented, and the lamellæ are held apart by the remainder of the effusion which gravitates between them. Partial contractions of the thorax are generally the consequences of partial and circumscribed pleurisies, as seen in the clavicular regions from effusions about the apices of the lungs when pulmonary tubercles are present; and in contractions of the lower section of the lungs, from inflammations about their bases.

Ossification of the fibrous lamella, more particularly of its parietal portion, occurs sometimes before, but more generally after, the absorption of the serous effusion. Deposition of bony matter generally takes place in the thickest portion of the exudations, in the form of compact knobby strings and plates. In rare instances, the whole pseudo-membranous pleural cone, with the exception of the thin layer covering the lungs, passes into ossification; and should this take place before the entire absorption of the effusion, then the relics of this remain permanently inclosed in a bony sac.

As several of the causes of thoracic contraction are present in pleurisies followed by other forms of exudation, such contractions are frequently noticed to take place in them, though generally in a minor degree. Thus a slight degree of shrinking of the chest follows even when the pulmonary layer of the exudation has become an extensible, yielding, cellular tissue, and the lungs have re-attained their normal size; but then the costal lamina of the effusion is transformed into a thick fibrous sheath, which, from its condensation and shrinking, gives rise to the contraction. We see it take place after pleurisies with inconsiderable plastic effusions, but with profuse ones of pus; it is then caused by paralysis and atrophy of the lungs from long-continued pressure, and by paralysis and alterations of the tissue of the intercostal muscles, as these keep equal pace with the intensity and continuance of the inflammation and the quantity of the effusion.

Inflammations at times occur in both pleuræ, and may be either simultaneous in their occurrence or not.

Pleurisies with persisting effusions may produce cachexia, general dropsy, hydrothorax of the opposite side, hyperæmia and

œdema of the lung of the affected side, asphyxia, dilatation of the right side of the heart, various degrees of venous congestion and atrophy of the lungs, and may lead to the eradication of already existing tuberculosis.

Typhous pleurisy in its strict and proper sense is perhaps always associated with typhous pneumonia.

In addition to the suppuration of the pleura, already mentioned as one of the terminations of empyema, a similar destructive process may arise, originating either within the parenchyma of the lungs or from without, somewhere in the circumference of the thorax. Simple or tuberculous abscesses, especially when seated upon the sternum or its neighborhood or on the spinal column; softening encysted exudations from the peritoneum-diaphragmaticum; perforating hepatic or splenic abscesses; ichorous or cancerous deposits, may all prove exciting causes of pleural suppuration. Perforation of the costal pleura is not unfrequently prevented by the presence of thick resisting layers of false membrane, the result of previous pleurisies which have effected such intimate adhesions of the lungs that an abscess cannot open into the cavity of the thorax; sometimes, however, it is perforated, and ulcerous destruction of the lungs themselves ensues.

Gangrene of the pleura arises when the serous membrane is laid bare by collections of pus or ichor in the lung or walls of the thorax, occasioning its transformation into yellowish-white, or more frequently into black or greenish-brown, soft, deliquescent eschars, coupled with superficial gangrene of the lungs. It is easily distinguished from the acute black softening, which the pleura-diaphragmatica undergoes from the stomach, or which the left mediastinal layer is subject to from its contact with the esophagus.*

* *Diagnosis.—Acute manifest pleurisy* is marked by the occurrence of a chill followed by fever, with quick and hard pulse, stitch in the side, dry cough, difficulty of breathing, dulness on percussion, absence of respiratory murmur, at times ægophony, and by dilatation of the affected side.

The stitch in the side is regarded as one of the most constant symptoms of pleurisy, but Louis asserts that it is more frequently absent than present. When present, it may be slight and wandering for several days, apparently seated in the muscles of the chest, and then without farther exposure it becomes suddenly exasperated; more commonly it sets in suddenly as an acute, penetrating stitch

1. *Secondary Formations.*

We pass over the cellular and cellulo-serous formations to the consideration of anomalous fibrous and cartilaginous tissues and bony substances. They occur with great frequency upon the

in the side, as if a knife were thrust into it at each inspiration, motion, or cough. Excessively acute pain is regarded by some as diagnostic of hæmorrhagic pleurisy. Pain is thought to be most frequent on the left side, although Chomel states that two-thirds of all cases of pleurisy are, like pneumonia, on the right side. Laennec states that it is not unusual for the pain to shift from side to side without a transference of the disease; it was already noticed by Stoll that the pain may be on the right side, and the disease on the left. If the posterior mediastinum is the particular seat of the disease, the piercing pain is often located between the scapulæ, and is much increased by motion of the dorsal portion of the spine, while the difficulty of breathing is slight, and the increase of pain from inspiration comparatively so. According to Schoenlein, pleuritis postica is often mistaken for lumbago, as the pain may be felt in the lower dorsal and lumbar regions only, or there may be violent drawing pain in the small of the back extending to the sacral region, increased by touch and motion, attended with a feeling of tension in abdomen, and with crawling or numbness in one or both legs; if, as is commonly the case, the membranes of the spinal marrow become simultaneously affected, there is pain exactly in the centre of the spine, which is stiff, and very sensitive to touch and motion; paralysis of the legs and bladder may come on suddenly. Pleurisy of the anterior mediastinum is marked by sharp pain under the sternum, and supervention of dulness on percussion.—[Andral.] Pain along the cartilaginous border of the ribs, extending into one or both hypochrondria and flanks, marks diaphragmatic pleurisy. But in the great majority of cases the pain is felt a little below the nipple, although the seat of the disease is often elsewhere; this is as much of a mystery as the pain in the top of the shoulder in liver-complaint, and that in the knee in disease of the hip-joint. The pain is often so sharp and lancinating at each inspiration as to impart a peculiar expression of anxiety and suffering upon the countenance of the patient, which is almost pathognomonic to the eye of the observant physician.

Respiration is often marked by a peculiar nervous hurry; it is short and jerky; it is also *low*, as the chest cannot be fully expanded on account of the increase of the pain at every attempt to do so: this is a valuable diagnostic sign from pneumonia, in which respiration is *high*, as the volume of a hepatized lung rather exceeds that of one fully expanded.

costal pleura, and exhibit two varieties in their origin and seat. In the *one* case, they are the products of inflammation, which, as above stated, have become transformed into fibrous or cartilagi-

The cough is short and dry, and attended with a thin mucous and very scanty expectoration ; if sputa are more abundant we should suspect a complication with pneumonia, or bronchitis; cough is usually infrequent and moderate, and may be so slight as not to attract attention.

The pulse is frequently peculiar; it is frequent and hard, instead of being full, large, but compressible as in one variety of pneumonia, or small, soft, and frequent (one hundred and ten to one hundred and thirty) as in another variety.

As the pleuritic effusion begins almost simultaneously with the commencement of the pleurisy, dulness on percussion is quickly found to be present; it is usually first noticed at the base of one or the other lung behind ; it is attended with a great feebleness of the respiratory murmur, or entire absence of it at the dull part. When the effusion is very copious from the commencement, we may find dulness over a large portion of one side of the chest at the very first examination, and entire absence of vesicular murmur which has taken place so rapidly, equably and completely, that no effort of inspiration can render it perceptible—the extension of the dulness in pleurisy is usually much more rapid than in pneumonia. Rattling in the air passages is heard much less frequently than in pneumonia—a rattle with numerous bubbles renders it far more probable that the disease is not pleurisy. If the lung be adherent to the diaphragm, or to the back part of the chest, it cannot be forced upwards and inwards towards the roots of the lungs and spine, but will be flattened against the back or the side of the chest ; the air-cells will be compressed, but the bronchi remain pervious, hence there will be dulness on percussion and bronchial respiration, so that the case may be mistaken for the second stage of pneumonia. But the bronchial respiration has not been preceded by crepitant rattle, and in some part of the chest there will be dulness on percussion, with feebleness or entire absence of vesicular murmur, without bronchial respiration.

Alteration of the percussion sound from a change in the position of the patient, is frequently put down as one of the diagnostic signs of pleuritic effusion, but we have the great authority of Laennec and Skoda for stating that it generally happens that the patient cannot change his position ; or if he be able to do this, and a portion of the lung be already completely compressed by the effusion, he cannot retain his new position sufficiently long to allow the fluid to leave its first place and occupy another so as to compress other portions of the lung. Increase of dyspnœa from change of position is a more important sign of effusion. Patient generally lies on the affected

nous tissues, and from thence to bone-earthy concretions, which assume the form either of plates or strings. They are always seated inside the costal pleura, to which they have intimately

side; in pneumonia, he prefers lying on his back, or on the sound side.

The grazing variety of friction sound is much more rarely heard in the commencement of pleurisy than at a later period, when most of the serous effusion has become absorbed, and a portion of the pulmonary pleura rubs, at each inspiration and expiration, against a corresponding part of the costal pleura, both being coated with plastic exudation. Under such circumstances the friction sound may become rubbing or grating, as if two pieces of new leather were rubbed together; the sensation may even be felt by the patient who is thus enabled to point out the exact spot of its location.

Dilatation of the affected side is a valuable sign; Laennec has found it well marked as early as the second day of pleurisy; an increase of six lines, or half an inch, is very obvious to the sight.

Ægophony is at best but an uncertain sign of pleurisy; Chomel heard but eight times out of nineteen; in six cases the voice was transmitted without being broken or tremulous, or its pitch raised; in five cases there was no approach to ægophony.

When the effusion is great, the heart is usually forced to the opposite side; but occasionally it happens that dulness may extend quite up to the clavicles, and yet the heart retains its position. Chomel says when the heart is forced aside the danger is great, and death very probable; he prides himself upon the cure of two cases of pleuritic effusion with displacement of the heart, occurring during the course of the same winter; both recovered he says, "which is very rare."

When the effusion is copious on the right side, the liver is pushed down, and, as it then projects below the ribs, it may be mistaken for enlargement of the liver. Jaundice and vomiting of bilious matters may be present, owing probably to a simultaneous inflammation of the convex surface of the liver; at least Hasse has drawn attention to the frequency with which adhesions of the liver to the diaphragm have been found, when extensive adhesions of the right pleura are present: we too have found this coincidence in at least twenty cases.

When the effusion is copious the natural vibration of the chest when the patient coughs or speaks is lost.—(Reynaud.)

Acute latent pleurisy.—It is not generally known how frequently acute pleurisy is latent; we have already quoted Louis, who states that acute pain in the chest is more frequently absent than present. It is best illustrated by an example; young Dr. Jackson, during his stay in Paris, observed that one of his young friends looked rather

coalesced. In *the other case*, these fibrous or cartilaginous tissues are developed without any inflammatory process, merely in consequence of a hyperæmic state of the sub-serous cellular and

unwell, rather more so than is common from a slight catarrh, which was all he complained of; Dr. Jackson called to see him again at night; they laughed and talked together for some time, he appearing pretty well, and referring his complaint to a slight bronchitis. Dr. Jackson examined his chest, not expecting to find any thing, so slight were the general and local symptoms; auscultation and percussion, however, detected a considerable pleuritic effusion. In another case recorded by Dr. Jackson, a woman had been sick for fifteen days; she had had a chill but no heat, pain, loss of appetite, acceleration of the pulse or dyspnœa; there was slight cough only—yet a pleuritic effusion filled the lower half of the left chest. The frequency in which pleuritic adhesions are found after death without there having been any manifest pleurisy during life, speaks strongly for the frequency of latent pleurisy.

Acute latent pleurisy is very apt to occur towards the end of chronic diseases; sudden prostration with increased difficulty of breathing are often the only signs which mark it; the patient sinks apparently from mere debility, yet after death extensive pleuritic effusion may be found.

Acute double pleurisy is almost always latent; it is rarely attended by the acute, circumscribed, characteristic pleuritic pain; as the dulness and dilatation of both sides may be equal, inspection of the chest and percussion may throw no light upon the affection, as no comparison between them can be made. It may however cause death before effusion takes place, on account of its extent and the extreme dyspnœa it occasions. If it take place rapidly it is generally fatal.

Diaphragmatic pleurisy.—According to Andral, *risus sardonicus*, which was regarded as characteristic of this disease by Boerhaave, Van Swieten, Dr. Haen, &c., is not even common in it; when present, however, we should think of diaphragmitis. Andral regards a remarkable degree of anxiety, sudden alteration of the features, acute pain in one or both hypochondriæ, extending along the edges of the false ribs to the flank; complete immobility of the diaphragm; absence of abdominal respiration, with presence of costal; almost constant orthopnœa, with inclination of the trunk forwards, as by far the most characteristic symptoms; hiccup, nausea, vomiting, and convulsive movements of the face, especially of the lips, may be present, but are often absent. It is best exemplified by a case: A man was seized with a slight shivering, followed by a burning heat; two days after, an acute pain was felt in the left hypochondrium, along the cartilaginous edge of the ribs; consider-

fibrous layers of the pleura, or of the serous membrane itself. We first notice a white, more or less circumscribed opacity and thickening of the serous tissue, which becomes converted into a

able oppression of the chest; sleepless, restless, occasional hiccup; on third day, pain continued, dyspnœa increased, and cough became frequent; patient was found sitting up in bed, with body bent forwards, his hand constantly applied to his side, where the slightest pressure caused him to scream aloud; he pronounced a few words in a broken, interrupted voice, and with difficulty; inspirations were short and frequent, and effected by the elevation of the ribs only; the diaphragm was fixed and immovable; cough frequent; no expectoration; pulse very frequent and hard; skin burning hot and dry; auscultation and percussion normal; some remission of the symptoms during the day, aggravation of them and delirium every night; on the fourth day convulsive movements of the face set in; on the fifth, almost continual nausea; pressure on the epigastrium induced hiccup and nausea. Death on the sixth.

Chronic pleurisy may occur either as a primitive affection, or as one secondary to an acute attack. In the latter case the severe symptoms moderate and partly disappear; the skin loses its burning heat; there is no dyspnœa, except from unusual positions, exertion, or excitement; a slight acceleration of the pulse alone remains: patient complains of weakness only, and fancies he is on the eve of convalescence. At a still later period pulse is no longer frequent; strength returns; walking, talking, or excitement no longer produce the least dyspnœa; yet the chest may be half full of fluid, only to be detected by means of physical signs [Andral]; this may be the case although the acute attack have lasted but four or five days. It is rare, even in the mildest cases, for the effusion to be absorbed in less than a month, commonly not under two or three months. [Laennec.] Dilatation of the chest is the most obvious sign which meets the eye of the physician; the affected side is more full and prominent than the healthy one, although the enlargement seldom exceeds an inch and a half; the intercostal depressions are not only obliterated, but their place is supplied by an intercostal bulging; while the ribs and cartilages remain permanently as far apart, or even farther than during the fullest inspiration. When this dilatation of the chest is coupled with extreme dulness on percussion, and with entire absence of respiratory murmur, or excessive feebleness of it, no doubt can remain as to the nature of the affection. When bronchial respiration supplies the place of feeble or extinct vesicular murmur, the lung is generally adherent to and compressed against some part of the chest; in such cases, however, dilatation, dulness, and feeble or extinct respiratory murmur, will generally be found opposite to the situation of the bronchial respiration.

smooth or rough elastic patch, or into a group of fibrous or fibro-cartilaginous granulations, or into masses with irregularly rounded angles, of the size of a pea or larger, and which finally ossify.

According to Laennec, primary chronic pleurisy is generally latent, or at least extremely insidious; the stitch in the side either does not exist at all, or it is obscure and momentary, only felt at long intervals; a slow fever creeps on by degrees; cough, with mucous or even puriform expectoration, is much more common than in the acute variety; it may come on so suddenly and profusely as to lead to the apprehension that an abscess has burst, or that pus has made its way from the cavity of the pleura into the air tubes; more or less rapid emaciation ensues, and many of the symptoms of tubercular phthisis present themselves, such as night sweats and diarrhœa; the mere symptomatologist cannot distinguish them. Formerly an accurate diagnosis between the two affections was a matter of little moment, as both were nearly equally and certainly fatal. Broussais saved but one case out of eighteen, and Laennec regarded a cure as a rare accident; but Stokes has saved twenty cases in succession with hydriodate of potash and blisters; while Hope has cured thirty-five cases running with mercury and blisters. Hope insists strongly upon the danger of mistaking the febrile irritation from anemia which attends this disease, for inflammatory fever; Townsend, Broussais, and Laennec made this mistake, and kept their patients on low diet; Hope, however, allowed strong broths, animal food, and porter, during the intervals of the most violent hectic fever, occurring in two daily tremendous paroxysms; the pulse was running from 120 or 130, to 150 or 160, with what patient terms internal fever, thirst, craving for cold drinks, dryness and heat of skin, &c.; even when the dyspnœa, faintness and danger of suffocation was most urgent, Hope relied on the prompt use of mercury, and states that it was quite common, and happened in fact in the majority of cases that the fluid descended one-third or even one-half, within forty-eight to sixty hours, relieving the extreme dyspnœa and danger of impending dissolution. It must not be forgotten, however, that dilatation of the chest and pushing down of the diaphragm may take place to such a degree that air will again enter the upper portions of the lung, although the quantity of fluid be not diminished. In the great majority of cases an attrition murmur, or pleuritic rubbing sound, will be heard as the fluid becomes absorbed; this is always best heard along the line of the margin of the lungs from the heart, curling backwards to the base of the lower lobe. The longer this rubbing sound is heard the better, as then the adhesions are more apt to be loose and elongated, and the lung will generally recover its full size, marked by full resonance on percussion, and complete restoration of respiratory murmur. But if rubbing sound last for a few days only,

These latter are always seated beneath or without the serous membrane, and are covered over by it.

The two varieties may be easily distinguished. The fibrous exudations clothe the whole costal as well as the pulmonary pleura, but they only ossify upon the costal layer. The subserous new formations occur almost exclusively upon the costal and diaphragmetic pleura, and are frequently found in the intercostal spaces. They sometimes become detached in the form of rounded, knotty masses, which fall into the cavity of the chest as free cartilages.

2. *Tuberculosis of the Pleura.*

Tubercles of the pleura occur in all those forms common to serous membranes, namely:

a. As perfect or partial and rapid conversion of a pleuritic exudation into tuberculous matter. This is most common on the costal pleura.

b. As tubercular deposits in organizing, or more or less organized pseudo-membranous exudations, i. e. tuberculosis upon or in cellular or cellulo-fibrous tissue of new formation.

c. As primitive, acute, miliary tubercles.

The second variety of tubercular formation is frequently complicated with secondary inflammation of the false membranes in which the tubercles are seated, and is very frequently attended with a hemorrhagic effusion.

Tubercles of the pleura are generally the result of a marked tuberculous dyscrasia already evinced by the production of tubercles in other organs. They are mostly associated with tuberculosis of the lungs and bronchial glands; still they occasionally occur as the first in order of the successively developing tubercu-

the adhesions become so close and universal that expansion of the lung cannot take place, and patient may remain more or less delicate for eighteen or twenty-four months, or even for life.—[Hope.]

Acute pleurisy is generally regarded as a dangerous disease; but Louis states that recovery almost always takes place when the disease attacks a previously healthy person; he estimates the deaths at not more than one in one hundred, as he did not lose a single case out sixty-eight. Chomel also states, that the pleurisies which occur so often during the course of consumption, rarely increase the fatality of the disorder, even when attended with effusion; resolution appears to take place as promptly as in ordinary simple pleurisy.

lar depositions. They often complicate stationary or even retrograde pulmonary tuberculoses, and are then to be regarded as evidence of a tumultuous re-crudescence of the general disease, and from that time forward they are generally associated with profuse tubercular depositions in many organs.

Tubercles of the pleura frequently soften and deposit tuberculous pus in the various pseudo-membranous tissues in which they are seated; the abscesses thus formed may perforate the pleuræ and thoracic walls, with or without being attended by caries of the ribs.*

* *Diagnosis.*—But little is known of the diagnosis of tubercles of the pleura, and it is of comparatively little importance, as tubercles of the lungs are almost always simultaneously present. They seem to be more common in children under fifteen than in adults: thus in one hundred cases of tubercular disease in adults, Louis found the pleura affected only twice; while Papavoine found tubercles of the pleura seventeen times in fifty cases in children; and Rilliet and Barthez, in three hundred and fourteen children with tubercular deposits in various organs, found them in the pleura one hundred and nine times. Circumscribed pleurisy is so common in phthisis that we can derive no assistance from the presence of pain in forming an opinion whether tubercles are seated in the pleura or not; in fact, fixed or movable, transient or permanent, slight or severe pleuritic pains, are so common in the dorsal region, between the scapulæ, or beneath either of the clavicles, during the course of consumption, as to form a valuable diagnostic sign of the presence of tubercles in the lungs; but these pains in the majority of instances arise from circumscribed, simple pleurisies, which lead to partial adhesions of the pleura. According to Barth and Roger, when tubercles are deposited under the two serous laminæ of the pleura, and form resisting prominences on the surface of these, they may give rise to manifest friction sound; yet, according to Fournet, circumscribed pleurisies at the apex of the lung are the most common cause of the grazing variety of friction sound, and indicate the progress of tubercularization towards the surface of the lungs; hence it is evident that in the majority of cases we can only conjecture the existence of tubercles in the pleura. When present in pleuritic pseudo membranes, they increase the existing irritation and sustain the secretion of fresh quantities of fluid and plastic exudations, while they offer a permanent obstacle to the complete absorption or organization of those which have already been thrown out; hence when pleuritic pains and irritation exist for months in succession, it is probable that they are kept up by the irritation of tubercles.

3. *Cancer of the Pleura.*

This is far less frequently met with than tubercles; it never occurs as the first in order of the successively developing cancerous formations, but is always the result of a cancerous dyscrasia, which has already localized itself in other parts. It is frequently found in company with cancer of the breasts, mediastinum, or even of the bones, and is common when numerous cancers are present in many organs; most commonly it arises simultaneously with cancer of the lungs, especially after the extirpation of large cancerous masses in the breasts.

The pleura may be perforated from without inwards by adjacent cancerous deposits, which, after they have involved it in their ulcerous destruction, project themselves into its cavity in the form of knotty protuberances; or else it appears originally upon the smooth internal surface of the pleura, in the form of flattened, roundish, knobby, fatty, marrowy bosses of the size of a hemp seed, egg, or fist. They are either loosely or intimately attached to the serous membrane; in the latter case they always attack the tissues of the pleura itself, and generally prove to be medullary cancer.

Their presence always occasions the accumulation of various quantities of serous fluid in the pleural sacs.

4. *Abnormal Contents of the Pleural Sacs.*

Besides the anomalous contents of the pleural sacs which we have already mentioned, and those to which we will subsequently allude, we must speak particularly of the presence of air and serum, forming the so-called *pneumo* and *hydro-thorax*. Accumulations of various kinds of air and gas have many sources distinct from those arising from the entrance of atmospheric air from without or through the bronchial tubes, in consequence of penetrating wounds of the chest or of injuries of the lungs. Pneumo-thorax may recur under the following circumstances:

a. In consequence of the opening of a tuberculous abscess before adhesion of the costal and pulmonary pleura has taken place. This is the most frequent source of pneumo-thorax.

b. In consequence of the rupture of a superficial gangrenous eschar of the lungs.

c. From the softening of the so-called metastatic depositions

which perforate the pulmonary pleura before reactive hepatization can take place in the lungs.

d. From the opening of a pulmonary abscess which communicates with one or more of the bronchi.

e. From the disengagement of gas from decomposing purulent and ichorous exudations (empyema).

f. In rare instances a small quantity of air is found as a product of inflammation, in connection with a benign pleuritic effusion.

g. In consequence of corrosion of the pleura and of one of the bronchi by purulent or ichorous exudations.

h. In consequence of rupture of one or several pulmonary vesicles in vesicular emphysema, or from rupture of the pleura in interlobular and vesicular emphysema.

i. From perforation of the diaphragm or mediastinum in consequence of acute softening of the stomach and esophagus.

If the disease giving rise to the pneumo-thorax is not in itself fatal, as is always the case in softening of the stomach and esophagus, then the accumulated gas causes inflammation of the pleura, and exudations which vary according to the source of the gas, its nature, composition, and that of the other substances which are precipitated into the thorax simultaneously with it. Pneumo-thorax causes compression of the lungs and hydro-thorax.

Hydro-thorax is a very rare disease, except when it occurs as a result of general dropsy, and even then it is scarcely ever the first in order of successively occurring serous effusions, except when it occurs from disease of the heart and lungs. It may occur as a consequence of hyperæmia of the pleura, and the longer it continues the more it is associated with swelling and hypertrophy of the same. It attends cancerous vegetations on the pleura, but arises especially from diseases of the heart and large vessels, pericarditis, catarrh, bronchial dilatation, indurated hepatization, and pleurisy. Finally, it occurs in the general dropsy which follows exhausting diseases, such as typhus, puerperal fever, phthisis from tubercles and cancer, brights disease, &c.

The lungs are compressed and forced from their situation in hydro-thorax in the same way that we have seen them in pleuritic effusions.

It should be carefully distinguished from those serous effusions which occur as the product of a pleuritic process. The beginner may find some difficulty in distinguishing it from those pleurisies which deposit exudations with scanty plastic product, forming the

so-called active dropsies. He is particularly liable to be mistaken when these effusions have persisted for some time.*

III. ABNORMAL CONDITIONS OF THE LUNGS.

1. EXCESS AND DEFICIENCY OF DEVELOPMENT.

In very imperfect monsters, viz., in the acephalous, the lungs and central organs of circulation are entirely wanting. In lesser

* *Diagnosis.*—Laennec was the first who succeeded in diagnosing pneumo-thorax during the life of the patient. It arises in nearly nine-tenths of all medical cases from the bursting of a tuberculous abscess into the pleura. The perforation generally happens at the inferior part of the upper lobes, or at the upper part of the middle lobe; hence in a great majority of cases it will be found on a line with the third rib, or between the fourth and fifth, more posteriorly towards the axilla. It seems to be far more common on the left side: thus, of eight cases by Louis, seven were on the left side; Houghton thinks his cases show nearly the same proportion; of forty cases collected by Reynaud, no less than twenty-seven were on the left; but of eight cases recorded by Laennec, four were on the right and four on the left. It generally occurs only after phthisis has made considerable advances, yet it has set in at a period when only the faintest suspicion of phthisis exists; instances are recorded in which there was only one tubercle in the lungs. Patient often feels the abscess give way, and air and matter pass into the pleura; this is so common that physicians should always inquire for it. Louis regards the sudden supervention of acute pain and overwhelming dyspnœa as symptoms of constant occurrence, and always indicating the time of the perforation; in seven of his eight cases they were both present. The pain, however, is not always proportionate to the sudden breathlessness. Drs. Townsend and Houghton each had a case without pain, or marked dyspnœa; and Louis one without pain. The acute pain which ushers in the attack generally subsides, and patient may not be troubled with it afterwards. If there be much fluid in the chest, even the motion of turning or raising in bed often leaves patient speechless and breathless for some time. Dry, husky, and ringing cough is thought to be characteristic by Dr. Houghton. Patient generally lies on the affected side. Chest is slightly or not at all elevated during inspiration, and the affected side is generally,

degrees of monstrosity, and even in cases in which the organization is otherwise quite normal, we may find a deficiency of one or both lungs, owing to arrest of development. In some instances the development may be arrested to such an extent that the lungs are represented by rudimentary, scarcely perceptible, roundish little bodies which are attached to the roots of the bronchi. This latter condition may be the consequence of contraction of the thorax, or of the pressure exerted upon the lungs by the abdominal organs, when the diaphragm is wanting, but far more frequently it is owing to dropsical accumulations in the chest.

An *excess* of development occurs in double monsters, as various degrees of duplication of the lungs, either with or without simultaneous duplication of the pleura. An apparent excess is

although not always, larger than the sound one. Œdema of one arm, but not of the chest, has been frequently observed. Succussion of the chest is a very valuable diagnostic means, as it is available in this form of disease only; the splashing of the liquid is often distinctly felt by the patient, and heard when the ear is applied to the chest; if either air or fluid be absent it is not felt or heard. The percussion sound is commonly and very evidently tympanitic; but if the walls of the chest be very tense, it is less, or even not at all so. As the opposite lung takes on a vicarious action, and becomes dilated or even emphysematous, it in rare cases affords a much clearer sound than the affected one, but the respiratory murmur is always puerile; while on the affected side none at all is heard, except in cases where strong adhesions bind the lung to the ribs and prevent its compression by the air and fluid in the pleura. In the majority of cases percussion elicits a metallic resonance simultaneous with the tympanitic sound. It is usually laid down that there is a strong line of demarcation between the tympanitic sound above, furnished by the air, and the dull sound below, owing to the presence of fluid; but according to Skoda, this is not so, for the percussion sound remains tympanitic to some distance below the level of the fluid, and is scarcely altered by a very thick layer of it, so that in pneumothorax we must calculate that nearly twice as much fluid is present as is indicated by percussion. Metallic tinkling is very commonly present in pneumo-thorax; it may be very accurately imitated by dropping a pin into a large wine-glass, so that it may easily be recognized by one who has never heard it before; it is principally heard when the patient speaks or coughs, but sometimes it attends the respiration. When it has a louder and graver pitch, and is deepened in tone so as to resemble the noise produced by blowing into a decanter or large bottle, we may be sure that the fistulous opening is large. Metallic tinkling and amphoric resonance are not peculiar

present when the lungs are divided into an unusual number of lobes.

2. ANOMALIES IN SIZE.

Hypertrophy and Atrophy.

The various differences in the size of the lungs depend for the most part upon the number of the air cells, and their capacity. Enlargement of the lungs may depend upon the presence of a greater number than usual of air cells, which are at the same time larger; under opposite circumstances the lungs are reduced in volume. The first state is generally connected with a great development of the muscular and osseous systems, and a comparative smallness of the abdominal organs; hence it is most frequent in the male sex. The second, is generally found when the mus-

to pneumo-thorax, but are also heard at times in large tuberculous caverns; as these may also give forth a tympanitic sound on percussion, and auscultation detects no respiratory murmur, the diagnosis between the two diseases may be difficult; but when the abscess fills, the presence of a large mucous or cavernous rattle will decide the question. It may also be mistaken for emphysema; but in very severe cases of this, the patient is generally able to be up and about; while by far the greater number of cases of pneumo-thorax occur in patients already bedridden with phthisis. The two diseases have tympanitic percussion sound and bulging of the chest in common; but in emphysema the respiratory murmur is only feeble, never entirely suppressed as is usually the case in pneumo-thorax; loud and prolonged expiration is frequent in the first, and bronchial snoring, whistling, and rattling, are almost invariable attendants, while they are very rare in pneumo-thorax, and when present are almost always attended with a peculiar metallic resonance.

The prognosis is very bad—very many die in the first week, and but few linger out a few months.

To sum up, whenever a phthisical patient feels something give way in the chest, followed by sudden and severe pain, with great dyspnœa; if soon after the chest sound tympanitic above, with some dilatation of the side and extinction of the respiratory murmur; if the chest sound as flat below as in pleurisy with effusion; if the splashing of fluid in the chest be heard when the patient is shaken by the shoulders, or felt by himself when he turns in bed; if metallic tinkling or amphoric resonance be heard by auscultation, there can be no hesitation in deciding on the existence of pneumo-thorax.

cles are less large and firm, the bones more slender and delicate, and the abdominal organs large; hence it is most frequent in females.

The lungs may appear large, either within or beyond the bounds of normal development, when any given number of their air-cells are dilated; under an opposite condition of the cells, the lungs will seem small. The lungs may even appear larger with a smaller number of air-cells, than in other examples in which a large number of air vesicles are crowded into a small space. In the first case the tissue of the lungs is rarefied; in the second, it is denser and compressed.

Hence, in forming an opinion of the size of a lung, the density of its parenchyma requires especial attention. The two extremes of excessive rarefication and extreme density of the lungs constitute very important diseases, of which we will treat

Diagnosis of hydrothorax.—Simple idiopathic hydrothorax was formerly regarded as a very common disease; now as a very rare one. It was frequently confounded with emphysema, cyanosis, chronic affections of the heart; in fact all chronic affections attended with blueness of the face, dyspnœa, oppression of chest, and œdematous swellings, were at once put down as hydrothorax. Even enlargement of the liver, by its compression of the diaphragm and lungs, and consequent dyspnœa, especially if œdema of the feet and diminished urine were added, was often treated as dropsy of the chest. *Corvisart* was the first to demonstrate its rarity. It has few or no pathognomonic symptoms; the starting in the sleep, anxiety, inability to lie down, irregular pulse, œdema of the legs, lividity of the face, &c., which were once considered as characteristic, depend far more frequently upon disease of the heart, of which, however, it is not an uncommon effect. Its physical signs are similar to those of chronic pleurisy, from which it can rarely be distinguished. Almost the only symptoms and signs which attend it are, impeded respiration, dull percussion sound, absence of respiratory murmur at the base of one or the other lung, and œgophony. The absence of pain in the side and of fever may occasionally serve to distinguish it from chronic pleurisy with effusion. It commonly exists on one side only.

Laennec considers symptomatic hydrothorax as common as the idiopathic is rare; it is almost peculiar to the moribund, as nothing is more uncommon than to find signs of it, even as long as eight days before death. If the effusion take place on both sides simultaneously, very painful suffocation may arise; at times, however, neither dyspnœa or any other sign marks its presence.

more fully when we come to the consideration of the alterations of the texture of these organs.

Hypertrophy of the lungs doubtless results from a remarkable combination of dilatation of the air cells with simultaneous thickening of their tissues; it is at times observed in the vicarious development of one lung, when the other, from any given cause, has become unserviceable. This variety does not depend upon an increase in the number of the air-cells, but in a dilatation of the existing ones, the walls of which have also become more massive and thick, while their capillary vessels are enlarged in calibre, or even increased in number by the addition of vessels of new formation. The tissue of the lung is thus rendered more dense, but in particular more firm, and the lung itself resists the pressure of the air in a remarkable degree; it has in fact become *larger*, and its thoracic cavity wider.

Atrophy of the lungs is exactly the opposite of the preceding condition; it occurs in the most marked degree in old age, under the form of atrophia senilis of the lungs; whenever it is found at an earlier period it depends upon a premature involution of the respiratory organs, and comes more properly within the limits of pathology. It consists in a dilatation of the air-cells, (emphysema,) with an alteration of their normal angulo-concave form to a roundish or elliptical; and this dilatation is the consequence of an emaciation and thinning of their walls, the vessels of which finally become obliterated. In extreme cases the walls of the air-cells are atrophied to such a degree that several of them coalesce and form a larger cavity; the interlobular cellular layer has disappeared, and hence the lobular structure is destroyed; the parenchyma of the lung resembles an irregularly perforated net work, while the lungs themselves are blanched, pale-gray in color, but spotted with much black pigment; they are soft and downy to the feel, light in weight, small in size; they collapse as the thorax is opened; when cut into, the air exudes sluggishly, with a dull, diffused sound; and their tissue is dry and bloodless.

This marasmus of the pulmonary organs is generally connected with an equally marked emaciation of the tracheal passages, dilatation and thinning of their walls, and dryness of their mucous membrane, and the proximate cause of both is essentially the same. As a rule it attains its maximum of development in the peripheric portions of the superior lobes, and hence often occasions a remarkable dislocation of the interlobar fissure, which gradually assumes a vertical position.—[HOURMANN.]

The walls of the chest sink down upon the atrophied lungs, become flattened laterally, and take on a conical form; the spine bends backward with a bow-shape; the sternum is thrust forward; and the vertical diameter of the chest is diminished by the spinal curvature, the consequent absorption of the intervertebral cartilages, and even of the vertebræ themselves. The soft parts of the chest, but especially its muscles, are pale and emaciated; the diaphragm is thin, lax, and lies in folds; the heart is small.

The difficulty of breathing, the greater part of the weakness, pallor and lividity of the tissues, and the general atrophy of aged persons, are owing to the above condition of the lungs. The small size of the respiratory muscles renders every inspiration imperfect; the loss of contractility of the lungs, together with the above condition of the muscles, makes each expiration equally laborious and imperfect; while so large a portion of the capillary vessels of the lungs are obliterated, that but a small quantity of blood is offered for arterialization.

If atrophy of the lungs occur at an earlier period of life, and be far advanced, while that of the rest of the body is but little so, then the disease will acquire fresh importance from the superaddition of active dilatation of the right side of the heart.

A remarkable enlargement of the lungs is present in emphysema; lesser degrees, and in part only apparent increase in size, take place in hepatization, high degrees of tubercularization, and in cancer of the lungs, &c.

A diminution of the size of the lungs may be induced by contractions of the thorax, but in particular by accumulations of air, or fluid within the chest, as in pneumo-thorax, hydrothorax, empyema, &c., or by obliteration of thebronchi.*

3. ALTERATIONS OF FORM AND POSITION.

The *congenital* alterations in the *form* of the lungs are gen-

* *Diagnosis.*—In hypertrophy of the lungs the percussion is clear to a greater or less distance below the sixth right rib, where it normally commences to be dull, owing to the presence of the liver. As this organ is pushed down farther than usual, the indistinctly or quite evidently tympanitic sound of the intestines at the lower edge of the right false ribs is replaced by the dul sound of the liver. The enlarged lungs also overlap the heart, hence the normal dulness at the lower half of the sternum will be much less than common—

erally limited to irregularities in the division into lobes, at times, however, attended with lateral inversion.

The *acquired* are, the already described displacement of the interlobar fissure in atrophia senilis; the pitting of the surface, from obsolescence of a portion of the parenchyma, or from con traction after the cicatrization of cavities; the flattening of a whole lung, or of circumscribed portions of it, or mere indentations from large or circumscribed accumulations of fluid, air, or exudations, or from aneurisms, heterologous deposits, &c.

Besides, when the lung has been surrounded for a long time by a pleuritic effusion, and received a thin, but firm sero-fibrous coating, which prevents its perfect re-expansion, it undergoes a peculiar alteration in form, consisting in a loss of the sharpness of its edges, but in particular of the concavity of its base, and which reminds us of a similar alteration in the form of the liver, after inflammation of its peritoneal coat. In rare instances, when the pseudo-membranous coating is thinner in some places, portions of the lungs may push through these, so as to form teat-like processes, with contracted, neck-shaped bases.

Congenital alterations of position consist in the protrusion of the lungs through large fissures in the walls of the chest; and in the lateral transposition of these organs. Among the *acquired*, belong the prolapsus of the lungs through penetrating wounds of the thorax; their dislocation in various directions from enlargement of the abdomen, or of the abdominal organs; from effusions into the pericardium, enlargements of the heart, aneurisms of the aorta, heterologous formations in the mediastinum, but most frequently from the most various accumulations of fluid within the thorax. If the latter causes be in action, and there be no already existing adhesions to prevent it, the lungs will always be forced upwards and inwards towards the mediastinum and spinal column. They may undergo the same alteration of position from obsolescence of their texture, from internal causes, as they then shrink back upon the roots of the bronchi.

the same is the case to the left of the sternum over the seat of the heart and left lobe of the liver. The respiratory murmur is heard louder and clearer, and lower down in the chest than usual.

In atrophy of the lungs, the respiratory murmur is feeble and is not heard as low down as usual; the dull sound of the liver extends higher up than the sixth right rib; the dull sound over the heart is more marked as the small lungs overlap the heart but little.

4. DISEASES OF THE TEXTURES.

We commence with the consideration of two quite simple alterations of texture, which, though slight in themselves, are the more important from their consequences, which, singularly enough, are similar; we refer to *rarefaction of the pulmonary tissue*, i. e. *vesicular emphysema*, and to *condensation of the lungs*.

a. Rarefaction of the Lungs.

EMPHYSEMA.

Under *pulmonary emphysema* we comprehend, according to Laennec, two different conditions, of which one, and that by far the most important, does not properly deserve this name. But this inaccuracy has been partially corrected by the employment of the terms *emphysema vesiculare*, and *emphysema interlobulare*, which serve to point out the seats of the two diseases, and to distinguish them from one another.

In vesicular emphysema we meet with an affection of the peripheric portions of the lungs, similar in its nature to that which causes dilatation of the bronchi, and even of the trachea. The discovery and valuation of this disease alone would have sufficed to render Laennec's name immortal.

It consists in a permanent dilatation of the air-cells, and all the respired atmospheric air is absolutely contained within their walls, whereas in true emphysema a portion of it forces its way into the interstitial cellular tissue.

It often arises very rapidly, as a vicarious development of portions of the pulmonary parenchyma, when other and larger parts have become impermeable; and appears to be frequently formed in a high degree, during the last moments of life, in consequence of the forcible inspirations. Thus, in hepatization of the lungs, we find their edges, and in extreme degrees of tuberculariza-tion, the interstitial parenchyma between the tubercles, and also the peripheric layers of the lungs, puffed up, and emphysematous. In like manner it developes itself in the train of those acute and chronic diseases which prove fatal from paralysis of those respiratory nerves which preside over the chemical functions of the lungs, and are attended with the most labored action of the chest, and insatiate desire for air; also from suddenly checked expira-

tion, as in hemorrhages from the air-tubes, which then often become obstructed with blood.

In such cases the following appearances present themselves: the emphysematous portions are puffed up, and have a peculiar feel, which may be compared to that of cushions, or bladders filled with air; they are pale, varying from a palish-red, to a dull white color, and in a perfect anæmic condition; dry, collapse rapidly when cut into, but their crepitation is less distinct and duller; they float upon the very surface of the water; their cells are dilated in various degrees, and their walls are the thinner in proportion as an extreme degree of emphysema has been rapidly formed. Finally, lacerations of some of the dilated cells take place occasionally, and the emphysematous portion then resembles a bloated and torn net-work; this is generally noticed on the anterior edges of the lungs, and towards their bases only; it gives rise to the effusion of air beneath the pleura, and to peeling of it from the lungs.

The thinness of the walls of the dilated cells gives this variety of emphysema some similarity to atrophia senilis of the lungs.

Another form of vesicular emphysema is of slow growth, and spreads itself gradually over a large portion, or even over a whole lung; it arises in part from other causal conditions than those already mentioned, and forms a structural disease, which, as Laennec truly remarks, gives rise to most of the so-called nervous asthmas.

This variety presents many differences of degree and extent. By degree, we refer to the grade of the dilatation of the air-cells; still we must remark, that in emphysema of long standing we always find several grades of dilatation simultaneously, and that it is only in the commencement of the disease that all the affected cells are equally dilated. They may attain the size of a pin's head, barley-corn, hemp-seed, pea, or even bean; and depart the more from their original shape, the larger they become. In the beginning, the disease consists in a truly simple dilatation of the air cells; and as their walls also become thickened in some measure, and more rigid, it represents an active dilatation of the same, and becomes somewhat analogous to hypertrophy of the lungs. In higher degrees, on the contrary, the dilated cells unite to form larger cavities, for their walls are absorbed in consequence of the pressure which they reciprocally exert upon one another. Such hemp-seed, pea, or bean-sized cells always pre-

sent a very irregular and multi-cavous, although somewhat roundish, form. Their internal surface offers a remarkable appearance, for ridge-like prominences project to various heights within their cavities, traverse them in different directions, and form boundary lines and imperfect partition-walls between the individual pouches. We also notice delicate threads, either extending from one wall to the other, or else hanging free into the cavities; these and the ridges are the remnants of the contiguous wall of the air cells. The larger the cells become, the more pressure they exert upon the adjacent tissues, and finally cause wasting away of them; thus they gradually acquire thick and rigid walls, which either collapse very slowly or not at all, when cut into.

This form of emphysema, also, is most frequently developed, and in its highest degrees, in the peripheric parts of the lungs and along their edges. It is often associated with bronchial dilatation, and this among other signs betrays the affinity of the two diseases. It either attacks a small portion of the lungs only, and then is confined to the anterior edges of one or the other upper lobe, or else it spreads itself over a whole lobe, or a whole lung, or both lungs.

In emphysema of both lungs, the complex of all the anatomical signs affords the following description of the disease:

a. Barrel-shaped dilatation of the thorax, with persistence of the intercostal depressions; great dorsal curvature of the spine; hypertrophy of the respiratory muscles, and clear percussion-sound.

b. When the chest is opened the lungs press forwards out of their cavities; they are remarkably large, and do not collapse from the pressure of the atmosphere.

c. Upon their surface, especially along their anterior edges, we find roundish, hemp-seed, pea, or bean-sized prominences, either singly or in groups, which are nothing more than the above described dilated air-cells.

d. The lungs have a quite peculiar, soft, elastic feel, which may be compared to that of a pillow filled with down.

e. When cut into they collapse very slowly, the air exudes sluggishly, with a very diffused, scarcely crepitating noise, which may be compared to that produced by air flowing slowly out of a bellows.

f. Their parenchyma is pale throughout, bloodless, and remarkably dry.

If but one lung is emphysematous, then the corresponding

half of the chest only is dilated, but it is important to know that the mediastinum and heart are forced over to the opposite side. Finally, if single portions only of the lungs are affected, they may, if they be very numerous, and the disease is fully developed, prevent by pressure the expansion of the neighboring healthy air-cells, and thus retain them in a state of permanent compression.

The causal conditions for the origin of emphysema, and its mode of formation in general, are not well understood, notwithstanding the numerous attempts at explanation. Laennec attributes it to his so-called dry catarrh with pearl-colored secretion, and explains it in a mechanical manner. According to him, this secretion and the catarrhal swelling of mucous membrane obstruct the bronchi in such wise, that, though they permit the entrance of the inspired air into the air-cells, yet they offer an obstacle to its exit, which cannot be overcome by the less energetic act of expiration, and hence a portion of it is retained. In the course of the succeeding respirations fresh quantities of air are added and retained, and finally accomplish the dilatation of the air cells, which is also facilitated by the expansion of the air from increase of its temperature. He also supposed that the long continued retention of the air, when straining during parturition, or in the act of defecation, or from blowing wind instruments, &c., might lead to emphysema.

With regard to catarrh, cases of emphysema have been cited in which either no catarrh had ever been present, or else it followed the dyspnœa as a mere symptom of the already existing emphysema; as regards the last mentioned exciting causes, it is said to have occurred in persons who have never been exposed to them. Hence a spontaneous dilatation of the air-cells has been supposed, which from equally unknown causes becomes at one time associated with premature atrophy and thinning of the walls of the cells, at another with hypertrophy and thickening of them.

Laennec's views hold partially true in emphysema from catarrh. Still we do not believe that it is the long retention of the air which causes the forcible expansion of the air-cells, but, much rather, the very deep and powerful inspirations which finally follow the retarded expiration; this view gathers confirmation from the effects of the labored inspirations in croup, bronchial catarrh of children, and whooping-cough. Besides the forcible dilatation, they may also cause paralysis of the con-

tractility of the lungs, and consequent stagnation of air in the dilated cells.

Still emphysema undoubtedly developes itself in some cases in which such injurious influences have never been present, and in fact slowly in persons who lead a sedentary life. In these, the less frequent but so much the deeper inspirations are the more to be regarded, because they take place without the aid of the diaphragm, as the occupation of these persons generally requires a bent position, by which the abdominal cavity is compressed. The paralytic and atrophied condition of the diaphragm is of the greatest importance here, for the prevented abdominal respiration is compensated by the labored activity of the other great respiratory muscles; and hence we find an evident dilatation of the superior portions of the chest, while emphysema is primarily and most fully developed in the anterior portions of the upper lobes of the lungs.

The thickening of the walls of the dilated air-cells arises doubtless from the final coalition with them of the adjacent tissues, which have become atrophied from the compression exerted upon them. Notwithstanding this, if the dilatations increase, atrophy of the contiguous walls of the cells will ensue, from the persistent pressure which the adjacent cells exert upon one another, and several of them will unite to form larger cavities, as is also the case in rare instances with contiguous bronchial sacs.

The dyspnœa in emphysema arises from a complication of causes:

a. The excessive accumulation of air in the lungs prevents the circulation in the capillary vessels which ramify on the walls of the air cells, by the pressure which it exerts upon them, and hence renders the arterialization of a sufficient quantity of blood impossible.

b. In the higher grades of emphysema numerous capillary vessels become obliterated, not only in the walls of the air-cells, but also in the adjacent atrophied pulmonary parenchyma, and produces the above consequence in a still greater degree.

c. The diminished contractility of the lungs, and the frequent and labored inspirations to which the lungs are constantly urged, allow of only a very imperfect evacuation of the air-cells, and occasion a permanent accumulation of highly carbonized air in them, which in its turn prevents the arterialization of the blood.

The impermeability of numerous capillary vessels leads to an accumulation of blood in the pulmonary arteries, and gradually effects an active dilatation of the right ventricle, then of the right auricle and both venæ cavæ, and finally of the venous system generally. The predominant venosity and cyanosis which ensues occasions the immunity of asthmatic persons from tubercular diseases.

The impermeability of the capillary vascular system also occasions the anæmic condition of emphysematous lungs, and renders the occurrence of œdema, stasis, hemorrhage, and pneumonia in them impossible.

It proves fatal by final paralysis of the lungs, by asphyxia from the accumulation of highly carbonized air, by paralysis of the heart, and vascular apoplexy of the brain.

Emphysema interlobulare is the only variety which truly deserves the name of emphysema; it consists in an accumulation of air in the cellular tissue, between the lobules of the lungs. If we except the not impossible spontaneous development of gas in the interlobular cellular tissue, it can only be the result of a rupture of one or more air-cells, and the exit of air from them into the adjoining cellular interstices.

We then find air-bladders in the spaces between the lobules, especially on the surface of the lungs; they vary in number and size; are characterized by their paleness, transparency, and roundish or rather oblong shape; they may be pushed along in the direction of the interstices, or made to run together so as to form stripes, which branch off on the surface, and towards the roots of the lungs; they at times completely circumscribe and insulate the lobules, and have a wedge shape; their bases are seated on the surface of the lungs, and their apices point inwards towards the roots; if they be very small and congregated close together, they present the appearance of froth. Upon the cut surface of a dried piece of the lung, we find the interstitial cellular tissue converted into irregular cellular cavities, of larger or smaller size, heaped above and around each other in various positions, and which may be distinguished at a glance from the adjoining air-cells. As has already been mentioned, the most of the effused air is found accumulated near the surface of the lungs, so that the pleura is puffed up in various places; frequently it pushes along in the cellular tissue which attaches the pleura to the lungs, and peels off the former to such extent as to form large, flattish convex, movable air-bladders. In such cases it is to be feared

that air may be extravasated into the pleural sacs from the rupture of one or more of these; on the other hand, the air may force its way towards the roots of the lungs, pass into the cellular tissue of the mediastinum, from thence into that of the neck, and thus cause general emphysema.

This variety is usually attended with a bloated state of the lungs, but never coexists with well marked vesicular emphysema. It is most common in children, and in them, and in the rare instances in which it occurs in adults, it is occasioned by very rapid and deep inspirations, or by long retention of the air, when great exertions of strength are made, which require a fixed condition of the thorax. Its most frequent seat is in the upper lobes of the lungs, especially along their anterior edges.*

* *Diagnosis.*—Emphysema often begins in childhood and increases gradually; it is frequently hereditary: thus, in eighteen out of twenty-eight cases, Jackson found that one or the other of their parents had had the same disease; in fact most of the hereditary cases of asthma are examples of emphysema. When slight, the rational and physical signs are not well marked; there is habitual slight dyspnœa, as may readily be observed by the physician, but which the patient may not be aware of; habitual cough, infrequent and dry, with trifling morning expectoration of very viscid, grayish, transparent matter. In such cases we generally find dilatation on one side of the chest, most frequently a little below the clavicle; with unusual clearness on percussion there, and feebleness of respiratory murmur. The patient, however, is very liable to repeated attacks of congestion of the bronchial mucous membrane, which are commonly mistaken for paroxysms of spasmodic asthma, as the difficulty of breathing is often extreme, and the bronchial secretions are scanty and viscid.—(Stokes.)

When the disease is confirmed and extensive, the complexion is of a dusky, somewhat livid hue; the face is full and rather bloated, especially when compared with the rest of the body, which is rather thin; the nostrils are dilated, thickened, full, and vascular; the lower lip is enlarged, its mucous membrane everted, and livid; the eyes are dull and projecting, with lividity and fulness, especially of the lower lid; there is a general expression of heaviness, dulness, melancholy, and anxiety, upon the countenance; dilatation of the heart is almost always present when the above signs exist. There is constant difficulty of breathing, which is aggravated in paroxysms; a short, interrupted diaphragmatic cough is present, in which the walls of the chest move but little; this is the more distressing, as copious bronchial secretion often exists, and the cough is not sufficiently expulsive to raise the mucus, hence it occurs and reoccurs

b. Condensation of the Pulmonary Tissue.

A certain degree of density of the lungs occurs in children, and some adults as an individual peculiarity; in the latter it is frequently coupled with smallness of the lungs and pleural sacs. It is also present as a transient condition during pregnancy.

It only comes within the domains of pathology, when it is permanent, of high degree, and offers a persistent obstacle to the capillary circulation of the lungs.

Such an increase of density may arise from encroachments of the abdomen upon the thorax, in enlargements of the former; more frequently, however, from lateral sinking in of the chest, in atrophy of the great respiratory muscles; from rhachitis of the chest, curvatures of the spine, distentions of the pericardium, enlargements of the heart, large aneurisms, heterologous deposits, &c.; or from pressure on the lungs in accumulations of fluid or air in the chest, pleuritic exudations, bronchial dilatations, &c. According to its exciting causes it may arise equally in both lungs, or only in one, or merely in portions of it; thus in rapidly developed emphysema we may find single lobules in a state of compression; in atrophy of the external respiratory muscles, single circumscribed portions of the lungs are found in

in paroxysms. The ribs are held permanently elevated, hence respiration is high; and the shoulder-blades and collar-bones are raised so as to produce the high shoulders so common in asthmatic subjects. As the chest is expanded to its utmost, and the ribs sink but little during expiration, the respiration is mainly effected by the diaphragm and is abdominal; hence the recumbent position, flatulence, distention of the stomach; in short, any thing which impedes the motion of the diaphragm is particularly apt to bring on attack of asthma. The patient stoops habitually, a habit contracted in his frequent fits of orthopnœa and cough; the points of the shoulder-blades project in a remarkable degree; the clavicles are arched and prominent; the sternum, instead of being flat or slightly concave, is thrown forward and even arched, both longitudinally and transversely. But the most marked alteration in the form of the chest is a cylindrical, or barrel-shaped dilatation of it, most marked under the clavicles, and posteriorly and inferiorly, with persistence of the intercostal depressions, which are widened, but deeply grooved, not protruding and bulging as in empyema. The lateral portions of the chest are also remarkably deep, but not so convex as the anterior and superior, and the posterior and inferior parts. As the lungs are dilated, and so inelastic as to collapse very

the same state, under the bent-in ends of the anterior edges of the ribs.

There are different grades, varying from simple increase of density, characterized by greater firmness and consistence of the lungs, to a state marked by stasis and hyperæmia from obstruction of the circulation, and to such a degree of condensation and compression as to cause annihilation of the air-cells, obliteration of the capillary circulation, and atrophy of the lungs.

The most intense degree of compression is induced by large pleuritic effusions. Besides the already described alterations in form and position of the lungs, they then become so much denser, that first the entrance of air into their cells, and finally that of blood into their capillaries, is rendered impracticable. If they still contain blood, their red color gives them such a similarity to muscle, that this state has received the name of *carnificatio pulmonis*; at a later period, however, they change to a dirty brown, or more frequently to a bluish-gray, or lead color; they are then tough, leathery, and sink in water.

If extreme compression persist for a long time, the pulmonary tissue becomes obsolete, i. e. it is converted into a cellulo-fibrous tissue, a condition, by the by, which differs widely from atrophy of the lungs.

imperfectly, a large quantity of air is constantly retained in them, and hence the percussion sound is remarkably full, clear, sonorous, and almost tympanitic. The diaphragm is pushed down in the same, or even in a greater proportion than the chest is dilated; hence, if unusual clearness of percussion shows that the lungs extend down to near the edge of the false ribs, we may be sure of the existence of emphysema; if the percussion sound is less full in the upper parts of the chest than in the lower, then only the inferior portion of the lungs are emphysematous. The heart may be so completely overlapped by the lungs that its impulse is scarcely or at all felt. As the lungs are almost always distended to the utmost, they can receive but very little additional air at each inspiration; hence extreme feebleness, or almost complete absence of respiratory murmur, is a natural consequence. When a large portion of the lungs contains cells enlarged to the size of a pea or bean, we may hear a peculiar dry rattle with large bubbles, similar in sound to that produced by clucking the tongue in the mouth; it is heard towards the end of inspiration, and may or may not be preceded by a hissing, whistling, or some other rale. Expiratory murmur may be inaudible, or very much prolonged, or attended with hissing, whistling, snoring, rattling, &c.

Excessive condensation of the lungs produces consequences similar to those which arise from emphysema, for it prevents their capillary circulation, and hence causes stasis in the pulmonary arteries, followed by active dilatation of the right ventricle, venosity, and cyanosis. Hence it, like emphysema, affords a remarkable immunity from tubercles, especially when it is coupled with curvature of the spine.

Atelectasis of the lungs forms a peculiar variety of anomalous condensation of the lungs. It is perhaps owing to a congenital bronchial catarrh, or catarrhal pneumonia, and consists in a deficient development of the lungs of new-born children, portions of which remain in the fœtal state after birth. It offers various degrees of obstruction to the collapse of the ductus arteriosus, and to the closure of the foramen ovale, whence the predominance of the right side of the heart is maintained, and cyanosis ensues.*

* *Note.*—Atelectasis of the lungs has only been correctly described by Joerg and Hasse of Leipzig; the French pathologists almost invariably mistake it for the lobular pneumonia of new-born infants; Rilliet and Barthez regard it as a carnification of the lungs, owing to chronic pneumonia. Its importance may be gathered from the fact, that the majority of the cases of asphyxia of new-born infants, and many cases of permanent morbus cæruleus, are dependent upon it. It consists in an imperfect expansion of the lungs by the first inspirations after birth; if present to any extent it may prove fatal in a longer or shorter space of time, but any time in the course of a few weeks after birth a rapid and perfect recovery may take place if the child be forced to breathe forcibly and deeply; after the lapse of several weeks the walls of the air-cells coalesce, and the case is rendered almost incurable, although the child may live for a year or more; occasionally, however, a slow and imperfect recovery ensues. The most common causes are, undue quickness of delivery, and excessive pressure upon the child's head. The first act of respiration is delayed longer than usual; the child is weak, moves its limbs but feebly, and they hang as if palsied; the voice is thin and plaintive; its eyes but half opened; the movements of its chest are either hurried and superficial, or else unusually slow, or heaving and irregular; its skin is livid, but gradually becomes less so, and may change into extreme pallor, accompanied with a cold sweat. When it is likely to prove fatal, the skin, from being pale, suddenly again becomes blue, the pupils are fixed and the head drawn backward, with slight rattling in the throat. However alarming the symptoms may be, it rarely proves fatal on the first attack; if the

c. HYPERÆMIA AND STASIS.

Pulmonary Apoplexy.

No organ, except the brain, is so frequently subjected to hyperæmia as the lungs. It occurs in various degrees, and may develope itself, either gradually, or with excessive rapidity, and forms the anatomical basis of most sudden deaths.

In lesser degrees, as simple hyperæmia, it is frequently, either an habitual, or periodically returning affection, of an active nature. It often ensues very rapidly, and may prove fatal by itself, but more frequently by the superaddition of acute œdema of the lungs. We then find both lungs equally bloated, dark in color, and their vessels filled as far as the minutest capillaries with dark-red blood; their tissue is succulent, and softened, but crepitates. In the bronchi we find a grayish, at times a reddish,

subsequent attacks are slighter the child may recover, although even then the continuance of life is precarious, for sudden catarrhus suffocativus may terminate the scene; or the obstruction in the lungs may gradually bring about a patency of the foramen ovale, and permanent cyanosis ensues, while the breathing remains oppressed, and each act is short as in asthma.

The inferior and posterior portions of the lungs, particularly of the left, are most subject to it; a whole lobe may be affected, or only single lobules or cells; the affected parts are always depressed below the surface of the lungs, in lobular pneumonia they are higher; the portions of pleura which cover them are perfectly smooth and shining, instead of opaque, or covered with lymph, as in pneumonia; the cut surface is smooth, while in pneumonia it is granular; the parts are hard, not softened as in pneumonia; they are more of a violet-red, than of the dull brownish-red of inflammation; if pressed, thin and dark, or normal, and non-areated blood flows out, while in the first stage of inflammation a considerable quantity of a turbid, frothy fluid, mingled with minute fibrinous particles may be forced out; in the second stage of inflammation of lungs, viscid and dirty brownish-red fluid may be pressed out, and in the third stage, much grayish-yellow, pus-like matter flows over the cut surface.

If death takes place soon after birth, the body is well nourished, but marked with numerous livid spots; the hands are clenched and the feet balled; froth exudes from the nostrils and closed mouth; the brain is congested, and the foramen ovale open. If the child has lived for some time, the body is emaciated, and the skin flaccid and wrinkled.

mucus, intermixed with air-bubbles. The heart is usually somewhat dilated, and always contains large quantities of thin and fluid, or only slightly coagulated, dark blood, especially in its right cavities. The venous vessels of the cerebral membranes are generally much congested, and frequently we find serous effusion into the ventricles, as a consecutive complication. The external integuments are characterized by their lividity, and the rapid occurrence of large and very dark death spots; the face in particular is much swollen, and more or less livid; the eyes and mouth are generally more or less open, the conjunctivæ injected; the mucous membrane of the mouth and throat is livid and coated with tough mucus. Greyish or pale reddish frothy mucus is found in the trachea.

Higher degrees of hyperæmia form what is called *stasis*. In this the pulmonary parenchyma is dark bluish-red, or blackish-red, and completely saturated with blood, whence it acquires some similarity to the substance of the spleen, and therefore has received the name of *splenization*. Several other of the characteristics of this state are subject to various modifications, which for the most part depend, first upon the degree and continuance of the hyperæmia, next upon the nature of the stasis and the quality of the blood. When it is recent, and slight in degree, the parenchyma is denser, but easily torn; it crepitates, although less than normal; a large quantity of fluid blood gushes from its cut surface; the affected portion is bloated, and floats upon water. In higher degrees, and from long continuance of the stasis, the air-cells and interstitial tissue swell to such an extent, that the former are perfectly impermeable to air; the parenchyma thenceforth becomes denser, harder, and heavier; it does not crepitate, and a comparatively small quantity of thick fluid blood flows from its cut surface. It seems almost as if the blood had coalesced with the structure of the lung, the whole affected portion of which is somewhat shrunken.

According to the nature of the stasis and the quality of the blood, the part which is in a state of splenization presents various degrees of discoloration, viscidity, fluidity, or development of gas in the contained blood.

According to circumstances we find at times either a muco- or sero-sanguineous fluid in the bronchi.

Stasis is either the result of an active, passive, or mechanical hyperæmia. Each of these may prove fatal by themselves, when present to any great extent; and either may sooner or

later be changed into inflammatory stasis, or true inflammation. It never attacks both lungs at one time, and with equal severity; but it generally selects the lower lobes in preference to the upper, and when it is found extended over a whole lung it has usually commenced below.

Passive stases in the most dependent posterior and inferior portions of the lungs are important affections, as they are generally developed in bed-ridden old persons, or in such as have been confined to bed for a length of time by diseases of the brain, typhus fever, typhoid, and adynamic affections, &c. It is identical with the so-called *Pulmonary-Hypostasis of Piorry.*

Mechanical stasis is most frequently occasioned by organi diseases of the heart, although excessive density of the lungs may give rise to it. It is developed according to circumstances, either in the arterial, or venous portion of the pulmonary capillary system.

It is important to distinguish these stases from that which occurs in the cadaver after death, the more especially as this latter is very frequent, and often is superadded to the former. It is most marked in the posterior and inferior portions of the lungs, and gradually diminishes from thence upward and forward. The lungs are soft, crepitating, and apparently not so much saturated with actual blood, as with a dark-reddish, discolored, bloody serosity, which pours out in large quantities from the cut surface, frothing as it flows, and may be entirely forced out by very moderate pressure, after which a pale, discolored parenchyma remains, more or less compressed in proportion to the pressure applied. Various quantities of bloody discolored serosity are found to have transuded into the pleural cavities; and the pleuræ themselves are stained from imbibition.

These stases have been variously regarded with respect to their relation to pulmonary hemorrhage and apoplexy of the lungs, especially in latter times, when doubts have been raised against Laennec's views of hæmoptoic infarctus. We regard them as hyperæmiæ of greater or less degree, which may occasionally lead to hæmoptysis, and it also appears quite clear to us that they also represent *apoplexies*, viz., congestive apoplexies, when slighter in degree, and apoplexies with effusion of blood into the parenchyma of the lungs, when more severe. But as the hemorrhage does not take place originally within the cavity of the air-cells, they are not *necessarily* followed by *hæmoptysis*, and hence must be distinguished from it.

The latter variety, i. e., hemorrhage within the cavity of the air-cells, is identical with *Laennec's Apoplexy of the Lungs*, or *Hæmoptoic infarctus*, the existence of which, and in fact in the manner described by this great master, we consider as well substantiated, at least according to our observations. The higher grades of it are attended by laceration of the pulmonary parenchyma.

This apoplexy of Laennec presents the following characteristics:

We find blackish-red spots in the lungs, which attract the more attention by their color and consistence, and because they are sharply circumscribed. Upon the cut, but still more readily on the fractured surface, we notice a more or less coarsely granular structure, the unequal granulation of which is often striking. The affected part is dry and firm, but fragile, and of equal consistence in all parts, as well in its centre as towards its edges. The whole represents an extravasation of blood into the cavities of the air-cells, which distends them to a certain extent, and then coagulates within them, thus giving rise to the granular texture of the hæmoptoic infarctus. The interstitial tissues are compressed, and even infiltrated with blood, hence the coloring of the affected part is uniform throughout. The terminal ends of the bronchi are also filled with blood, and their walls are red from imbibition, like those of blood-vessels when saturated with blood. If we scrape the infarcted portion with the back of a scalpel, we only obtain a small quantity of thick blood, intermixed with many black grumous flocculi.

Hæmoptoic infarctus bears some similarity to red hepatization of the lungs, still the two may only be mistaken for each other by very inexperienced persons, for every one of the above characteristics of infarctus also includes a sign of distinction from hepatization. These briefly summed up are, the sharp circumscription of the infarctus, the uniformity of its consistence and color throughout its whole extent, *the coarse and unequally granular* appearance of its cut or fractured surface, the dry fragility of its structure, and the peculiarities of the product obtained by scraping and pressing it.

The pulmonary tissue adjacent to the infarctus is either in a perfectly healthy state, or else in different states of pre-existing or consecutive disease; in every case, however, it is sharply divided from the infarctus. The pre-existing diseases are usually tubercles or pneumonia; the most frequent consecutive, are em physema and œdema of the lungs.

At times we do not immediately recognize the sharp boundary of the hæmoptoic infarctus, but only after a close examination; this is particularly the case when the surrounding parenchyma forms the seat of an effusion of fluid blood, the limitation of which is never sharply marked, as it changes towards its circumference into a pale sero-sanguineous infiltration, and gradually makes a transition through this into the normal tissue. Still by a careful investigation one will discover the infarctus seated within the fluid effusion, and plainly bounded off from it by its greater firmness and darker color.

The size of an hæmoptoic extravasation is generally not great, for, as is correctly stated by Laennec, it scarcely ever exceeds the measure of four cubic inches, while it is frequently less than one. We frequently find but one infarctus; at times, however, several are present simultaneously either in one or both lungs.

The most common situation of them is in the deeper seated portions of the lungs, near their roots, or else in the posterior parts of the inferior lobes; but occasionally they are found near the surface, and then glimmer through the pleura, so as to be immediately recognized by external inspection. Sometimes a circumscribed pleurisy then arises above them.

They are often, but far from always attended with hæmoptysis of greater or less degree; and their size stands in no kind of proportion to the extent of the blood-spitting, for a very profuse hæmoptysis may have taken place, without our finding even a trace of hæmoptoic infarctus in the cadaver; or the effused blood may coagulate rapidly and perfectly within the air-cells, and then, notwithstanding the presence of infarctus, there will be no hæmoptysis; again, the blood may not coagulate at all, but be coughed up in a fluid state, and then, although there has been spitting of blood, no infarctus has ever existed. On the other hand, the first extravasation may coagulate and form an infarctus, and hæmoptysis may have its source in a secondary sanguineous infiltration of the parenchyma. (See above.)

It is this form of apoplexy which is so frequently found in connection with an active dilatation of the right side of the heart, and appears to be equally dependent upon it, as apoplexy of the brain is upon active dilatation of the left ventricle.

In latter times the definition of hæmoptoic infarctus as a pulmonary apoplexy has been cavilled at, and it has been suggested that it is the result of hemorrhage from the minuter bron-

chial tubes, and subsequent gravitation and coagulation of this blood in the air-cells. In our opinion, it cannot be denied that this may be sometimes the case; but in default of actual proof of this process, we prefer adopting Laennec's views, especially as hæmoptoic infarctus frequently occurs without being followed by hæmoptysis, while bronchial hemorrhage could scarce exist without being attended with cough and expectoration of blood. Besides, if, as is but just, we admit the influence of hypertrophy of the right ventricle in the production of this disease, it is evident that hemorrhage will be more apt to ensue from the pulmonary arteries, than from the bronchial vessels, although we are well aware that anastomoses occur between these two systems of blood-vessels.

In higher degrees, this variety of apoplexy is complicated with *laceration of the substance of the lungs;* we then find a cavity similar to those which occur in cerebral apoplexy, which contains varying quantities of more or less coagulated blood. The adjacent pulmonary tissue is torn, suffused with blood, and in a state of hæmoptoic infarctus to a greater or less extent.

The favorite seats of these cavities are the same as in hæmoptoic infarctus; it has happened in rare instances that, when seated near the surface of the lungs, they have opened into the pleural sacs through a laceration of the pleura, giving rise to effusion of blood into them, and pneumo-thorax.

The size of the cavities varies, but it rarely or ever exceeds that of hæmoptoic infarctus. Circumscribed gangrene of the lungs, however, at times gives origin to very large depositions of blood.

Simple hyperæmia and stasis easily retrograde to the normal state, especially when aided by judicious medical treatment; but it is well known that they leave great liability to subsequent relapses, and hence generally require continued prophylactic treatment.

But the questions arise, What alterations do hæmoptoic infarctus and apoplexy with laceration of the lungs undergo, in the course of time? And in what way do the natural endeavors at cure and their accomplishment evince themselves?

Experience very rarely presents us with the necessary, pure, and undoubted facts for the solution of these queries, especially with regard to the connecting links of the curative process; still, with the aid of the few materials which are at hand, and by a comparison with analogous processes in other organs, we propose the following:

The effusion in hæmoptoic infarctus either quickly becomes fluid, then takes on a blackish-brown, rust, or wine-lees color, and is partly absorbed, partly ejected through the air-passages, forming the peculiar sputæ of hæmoptysis; the parenchyma remains moist, soft, and fragile for some time, and is of a wine-lees color, but gradually returns to its normal state. Or the extravasation is only partly removed in this manner, and a coagulum remains, which either becomes blanched, dense, and fibrinous, or else soft, glutinous, and saturated with black pigment; the parenchyma around it contracts, and finally changes into a white, or blackish cellulo-fibrous tissue.

Apoplexy with laceration heals after the effusion has been absorbed, either by an immediate agglutination of the walls of the cavity, or the parenchyma contracts around a fibrinous coagulum, which finally becomes chalky, or a cellulo-fibrous capsule is formed around a glutinous coagulum, which consists for the most part of black pigment.*

* *Diagnosis of Congestion of the Lungs.*—In the milder forms there is sensation of pressure upon the chest, or only on one half of it, or upon a still more circumscribed place: this oppression is increased by motion and talking, and is a true dyspnœa, as the patient cannot take a deep breath, for, if he attempts it, increased difficulty of breathing and a short cough ensue, which effectually prevent a further progress of the experiment. If hemorrhage should take place, the patient feels as if warm water was suddenly poured into the chest, and quickly raises blood by coughing. This variety is most common in the phthisical. The severer form generally occurs in young and plethoric persons, from the sudden suppression of some natural or acquired discharge; the breathing quickly becomes much hurried and very short; the face is swollen and deeply flushed; there is excessive restlessness, short rattling respiration, the mouth may be filled every now and then with frothy, sanguinolent saliva; imminent danger of suffocation ensues, and the pulse from being full and large, becomes small, resisting, and hard, and finally very feeble.

Pulmonary Apoplexy.—The milder form resembles the severer variety of congestion of the lungs in its symptoms: according to Laennec, there is great oppression of the chest, with cough, attended by much irritation of the larynx, and sometimes by very acute pain in the chest; expectoration of bright and frothy, or of black and clotted blood, either quite pure, or mixed with saliva or mucus; the pulse is frequent and full, with a peculiar kind of vibration, even when soft and weak, as it frequently is after a day or two; the heat

d. Anæmia of the Lungs.

Bloodlessness of the lungs arises from various causes:

a. After exhausting hemorrhages.

b. From consumption of the blood, in the course of various acute and chronic affections.

c. From inspissation of the blood, in consequence of sudden and excessive losses of its serum, for the thickened mass then cannot penetrate the capillaries of the lungs; hence, among others, the anæmic condition of the lungs in Asiatic cholera.

d. Finally, it attends atrophy, emphysema, and high degrees of compression of the lungs.

e. Œdema of the Lungs

Is a very frequent and important disease. Its essential and primary characteristic is an infiltration of the pulmonary parenchyma with a serous fluid; this is evident even from external inspection, but becomes more so when an incision into the organ is made, as this is followed by a profuse gush of serum from the

of skin is natural or nearly so; the heart and arteries frequently yield a marked bellows sound. The diagnosis of the disease is obscure, for Laennec states that of all the symptoms the spitting of blood is the most constant, commonly copious, returning by fits, with cough, oppression, anxiety, intense redness or extreme paleness of face, and coldness of the limbs. But every pathologist knows that circumscribed pulmonary apoplexy is frequently found in the cadaver, though there had been no hæmoptysis during life; again, the almost invariable dependence of hæmoptysis, if at all considerable, upon tuberculization of the lungs, is an admitted fact. As about two-thirds of all cases of pulmonary apoplexy depend upon disease of the heart, generally hypertrophy of the right ventricle, it is evident that if the above train of symptoms set in, and there be no evidences of tubercular disease, but marked signs of disease of the heart, we may at once conclude upon pulmonary apoplexy, the more especially if copious hæmoptysis be also present. Laennec placed great stress upon the presence of dulness of percussion over the seat of the hæmoptoic infarctus, with absence of respiratory murmur there, but presence of crepitant rattle around the dull part; these signs, however, are only found when the hæmoptoic infarctus is seated on the very surface of the lungs, which is not often the case.

Severe apoplexy of the lungs is a more frequent cause of very sudden death in aged persons, than even cerebral apoplexy. It may

cut surface. Still, not only does the quantity of the serum, i. e., the degree of the œdema, vary, but this fluid also offers many differences in its properties.

In order to comprehend the importance of pulmonary œdema, under all circumstances, it will be necessary for us to premise what observation at the bed-side and careful examination and experiments on the cadaver have taught us concerning the seat of the serous effusion. According to all these it is effused into the cavities of the air-cells, and is accumulated there, according to circumstances, either with or without the admixture of different quantities of air. From thence more or less of it flows into the bronchi, forming *bronchial-foam* when it is mixed with air and frothy, or else a limpid, unareated fluid. The walls of the air-cells and the interstitial tissue are also saturated with serum, and infiltrated ; but the actual seat of the fluid, which often gushes forth in such astonishing quantities from the cut surface of an œdematous lung, is in the air-cells and bronchial canals.

Pulmonary œdema occurs either as an *acute or chronic* affection, and between both there are many minutely shaded transition stages. In *acute œdema* the lung appears bloated, does not

commence with oppression of the chest, difficulty of breathing, great lividity of the face and coldness of the limbs; at other times the first symptoms are, a sense of extreme weakness and oppression; the patient grows pale and totters, or perhaps falls down, yet he generally retains his consciousness, and may even be able to tell the bystanders that his distress is in his chest; the face is blue, as in one strangled, the eyes project from their sockets, froth and blood at times collect before the mouth, and slight gushes of black blood may occasionally follow; the breathing is short and unequal; there is rattling in the trachea and bronchi ; the pulse, from being full and strong, rapidly becomes small, weak and fluttering; the limbs are cold and in a quarter or half an hour a cold, damp sweat breaks out upon the skin. It is more dangerous than cerebral apoplexy ; life is more speedily extinguished, and remedial measures are altogether less efficacious; the latter generally lasts twelve or more hours; this frequently kills in a half or one hour. The appearance of the corpse is often sufficient to indicate the disease: there is great lividity over a large surface of the body; the eyes are open and projecting, as in drowned persons; frothy, sanguineous mucus oozes from the mouth in greater or less quantity, according as the head is elevated or not; the body retains its heat for a very long time, especially in the epigastrium.—[Leveille.]

collapse, feels distended, and when pressed upon with the finger we recognize the presence of a fluid which changes its position with a crackling noise; it has lost but little of its elasticity, so that only an almost imperceptible pit remains after pressure; it is of a pale-reddish color, but is quite pale when coupled with anæmia, and reddish and congested when attended with hyperæmia; the serum which flows from the cut surface is mixed with much air, hence frothy, and generally of a pale-reddish color, but in œdema from long continued stasis, with simultaneous decomposition of the blood, it is red, discolored, and has an icteric hue. The parenchyma is then soft, very moist, and easily torn.

If the œdema last for a *longer time*, the parenchyma gradually loses its elasticity, pits more readily under pressure, becomes paler, takes on a tawny, dirty-gray color, and becomes opaque and dull; the air is gradually pressed out of it, it crepitates less when cut into, and the serum becomes less and less frothy, loses its reddish hue, and finally is clear and colorless. The tissues gradually become more and more infiltrated with serum, the walls of the cells and the interstitial tissue swell, and hence the whole becomes denser and more firm.

Finally, in well marked cases of *chronic œdema*, especially in those which are such from the very commencement, we find the lungs pale, dirty-gray in color, bloodless, not swollen, but heavy, dense, and firm; they pit under pressure, but no longer crepitate, and a grayish or slightly greenish serum flows from the cut surface, without frothing. It is almost always associated with dropsical accumulations in the pleural sacs.

Œdema of the lungs, like acute œdema glottidis, often arises *very rapidly* from active hyperæmia, or passive or mechanical stasis, quickly reaches a high degree of development, and spreads over both lungs simultaneously, causing sudden death from suffocation. It very frequently is the cause of those so-called pure asphyxias of adults and new-born children, which are combined with hyperæmia and serous effusions within the cavity of the cranium. The cadaver usually presents the same external appearances as those described when treating of pulmonary apoplexy; the lungs in particular exhibit signs of œdema, a frothy serosity is accumulated in the air-tubes, and frequently forces its way into and outside of the nose and mouth upon the face, when it appears as a thick, whitish-red, or white froth. It may also follow in the train of acute or chronic bronchial catarrh,

or of exudative processes (croup) of the tracheal or bronchial tubes; it is a constant attendant upon the acute deposition of tubercles, acute decompositions of the blood in general, and upon the suppression of erysipelas, scarlatina, variola, rheumatism, miliaria, &c. In the form of acute œdema, it is developed in a greater or less degree in all the stages of pneumonia, attends the various metastases to the lungs, and often combines itself with hæmoptoic infarctus, tubercles, and cancer of the lungs. It finally follows in the train of diseases of the brain, general anæmia and tabes, and occurs towards the end of almost all chronic diseases.

Chronic œdema often connects itself with general dropsy, with dropsy of the larger serous cavities, chronic diseases of the heart and great vessels, &c. It but rarely is an idiopathic and independent affection.

The *extent* of œdema varies; thus in the very acute and rapidly fatal variety it generally attacks both lungs in an almost equal degree; in other cases it is limited to single portions of them. That which attends pneumonia commonly arises about the circumference of the inflamed parts; that which arises in the train of chronic diseases, is most marked in the posterior and inferior, or most dependent parts of the lungs, &c.*

f. INFLAMMATIONS OF THE LUNGS.

Pneumoniæ.

Usually but one, and that the most frequent variety of pneumonia, is recognized by authors; but we cannot even agree with

* *Diagnosis.*—According to Schoenlein, the credit of first calling attention to œdema of the lungs, belongs to Peter Frank; in many cases in which he had diagnosed hydrothorax, he found no trace of water in the chest, but œdematous infiltration of the lungs. In the acute variety the patient has extreme difficulty of breathing and violent dyspnœa, which is more intense from the very commencement than in hydrothorax; the signs of obstruction to the venous circulation are also strongly marked, the lips, tongue, and cheeks are blue; one of the most characteristic symptoms is, that no position or change of position affords any relief, while in hydrothorax the upright sitting position, with the feet hanging down, is the easiest: in œdema of the lungs the patient is equally uncomfortable in all positions; whether sitting or standing, lying on one side or the other,

all the prevalent opinions concerning this. We remark here quite briefly, that its croupous nature will be the more evident, the more rapid the course of the disease and the greater its severity; hence we will treat of this most common form under the name of

1. *Croupous Pneumonia.*

It, as is well known, runs its course in three stages, the first of which has been termed *inflammatory engorgement* (engouement); the second, *hepatization* (ramollisement of Andral); the third, *purulent infiltration.* We confine our remarks at present exclusively to those cases in which a whole lung, or at least a whole lobe, is inflamed.

The first stage, i. e., *inflammatory engorgement*, is always preceded by the above described state of simple stasis and splenization of the parenchyma; but this, on the other hand, need not necessarily increase to inflammatory stasis or engorgement. This may suffice at present as an expression of our opinion of the dispute concerning the inflammatory nature of simple stasis, and of its significance as the first stage of inflammation. A very careful examination is necessary to discover the distinctive marks between inflammatory engorgement and simple stasis; in the former the lung is generally of a darkish-red color, heavy and firm; it retains a pit after pressure, and we notice that it contains little or no air, its place being supplied by a fluid. When cut into we find its substance denser, owing to the swelling of its tissues, and to its being infiltrated with a sero-sanguineous fluid: according to the degree of this density, it may or may not crepi-

or upon the back, it is all the same; hence the patient tosses restlessly from one position to another, as he finds but little relief in any. The bronchi are filled with thin mucus and water; hence the patient coughs much, and often expectorates as much as one or two pounds of frothy, sero-albuminous mucus in twenty-four hours; this expectoration is not always present; when it is, it is pathognomonic. The general signs of dropsy are also added, viz., coldness of the limbs, scanty secretion from the skin and kidneys; anasarca may commence at the feet and progress up the legs.

According to Skoda, if all the air be not pressed out of the affected l ung the percussion remains normal, or even becomes somewhat tympanitic; hissing and whistling may be heard in the bronchi, but the most characteristic sign is a sharp, almost hissing vesicular

tate, and will or will not swim upon water; it is easily torn, very moist, and effuses a moderately or not at all frothy sero-sanguineous fluid from its cut or torn surface.

As has already been remarked, it bears the greatest similarity to, and relationship with, simple stasis, especially when combined with acute œdema.

We will endeavor to point out the characteristic signs by which inflammatory engorgement may be distinguished from the above-mentioned similar conditions. Among these belong the *brownish-red color*, and the *moisture* of the parenchyma, which will of themselves suffice to distinguish the inflammatory from the simple stasis, and even from that complicated with œdema; for they depend upon the *infiltration* of the tissues with blood which has undergone an inflammatory transformation, i. e., with a brown or brick-red colored, thin but viscid fluid, intermixed with many blackish crumb-like flocculi. As soon as it commences its transition towards the second stage, the *secretion* of a very tenacious, tough, reddish-brown fluid takes place, a portion of which may be expectorated and form the characteristic rusty sputa of pneumonia, as may be easily proved by an examination of the cadaver. Finally, true *exudation* sets in, and with it appears—

respiration, intermingled with a fine sub-crepitant rattle, which is not so dry as that which marks the first stage of pneumonia, for the bubbles seem larger, and give the ear a more evident impression of moisture.

The symptoms of chronic œdema of the lungs are very equivocal: there is cough, dyspnœa, and more or less of a watery expectoration; if the larger portion of the air is forced out of the affected part of the lung, and this is fairly soaked with serum, then the respiratory murmur is very feeble, and accompanied with the liquid variety of sub-crepitant rhonchus, the long continuance of which, and the absence of the general symptoms of pneumonia, and the presence of marked signs of excessive venosity, will always enable us to distinguish it from the first stage of pneumonia. The percussion sound is rather duller than normal, and slight bronchophony may be heard, as the parenchyma of the lung is somewhat compressed.

It frequently arises from Bright's disease of the kidneys, and is very frequently the immediate cause of dissolution, or of the increased distress towards the approaching termination of life in this disease. In one hundred deaths from albuminous nephritis, œdema of the lungs was present thirty-one times. Laennec says that it is very apt to occur during a temporary convalescence from pneumonia.

The second stage, that of *hepatization*, in which the lung presents a dark brownish-red color, both internally and externally; it is hard, but fragile; does not crepitate at all, and sinks in water. Upon the cut surface we either notice the dark brownish-red coloring equally distributed, or else deposited in irregularly shaped spots surrounded by black pulmonary pigment; the whole acquires a variegated or marbled appearance, from the visible ramifications of the pale-reddish interlobular tissue, and of the whitish bronchi and blood-vessels. Further, we detect a quite characteristic alteration of texture upon the cut or fractured surface, especially when the light falls obliquely upon it, viz., a well marked *granulation*, and it is this in particular, coupled with the firmness, fragility, and brownish-red color of the hepatized lung, which gives it its similarity to the liver and its texture, and has led to the adoption of the now universally understood term *hepatization*. The granulations are equal in size and roundish. Scarcely any fluid exudes spontaneously from the cut surface, and it is only by using some pressure, or by scraping it with a scalpel, that we obtain a small quantity of turbid, brownish-red, sero-sanguineous fluid, intermixed with blackish-brown, and some grayish-red flocculi.

The volume of a hepatized lung does not in general exceed that of a healthy lung, when in a state of full inspiration; hence its surface is always smooth, never indented by the ribs, and the thorax not at all dilated. Still we occasionally find single lobules projecting higher above the surface than others, owing to an irregularity in the progress and degree of the exudation; the granulation of these is generally larger and coarser, from the deposition in these places of a larger quantity of the products of inflammation.

This variety of hepatization is styled the *red*, in order to distinguish it from another, in which the granular texture remains, but has changed its color.

It is of the greatest importance to decide the manner in which the granular texture is formed. We almost universally find it attributed to such a degree of swelling of the walls of the air-cells that they become obliterated; in this view, every granulation is an air-cell, which has become converted into a solid body. We can by no means subscribe to this generally received opinion, but are convinced that the granulations arise from the deposition of the products of inflammation within the cavities of the air-cells, but we defer bringing forward our proofs here, as

the perfect solution of this question involves a decision of the seat and nature of the pneumonic process, of which we propose to treat more fully hereafter. We content ourselves with saying, at present, that each granulation is a hardish, fragile, and dark-reddish plug, which adheres so closely to the swollen walls of the air-cells, that it is extremely difficult to separate it from them and lift it out.

From the state of red hepatization, pneumonia passes through several, not well marked transition stages, into the *third stage proper*. The alterations consist almost exclusively in modifications of consistence, but particularly of color; for the red hepatized lung gradually becomes paler, assumes first a brownish-red, then a grayish-red and gray color, which finally takes on a yellowish tinge, when the whole forms a condition which has been aptly called *gray hepatization*. We recognize this color, even by external inspection, but in a far more marked degree upon the cut surface; in many instances the coloring is not uniform, but we find a grayish-red, or gray ground, tinged with yellow in some places, traversed by the white and prominent severed vessels, and sprinkled with more or less black pulmonary pigment, so that the whole presents an appearance similar to the yellowish-gray German granite.

The granular texture is still evident, and even becomes more distinct at the commencement of the third stage, especially when the disease has run a rapid course. The consistence diminishes the more the nearer the hepatization approaches to the third stage, and keeps pace with the increasing loss of color; the lung, it is true, feels pretty firm, but gradually retains a pit after pressure; it is mellow, easily torn, and a grayish-red, very turbid, flocculent, viscid fluid exudes from its cut or torn surface.

If we examine into the state of the granulations during these transition stages, we find that they have become more marked, and isolated, and larger; they are more easily separated and lifted out, as they adhere but slightly to the walls of the air-cells, through the intervention of a glutinous substance.

Such is the description of the transition stages from the red to the *third stage proper*, that of *purulent infiltration*, at the commencement of which the yellowish tinge becomes more equally diffused, and the granular texture disappears more or less rapidly, its place being supplied by a purulent infiltration of the parenchyma. The lung is then heavy; pressure readily forms a pit, which remains for a long time; the cut surface is of a straw-

yellow color, spotted with black pigment, and a large quantity of yellowish and viscid, purulent, nauseous fluid flows from it. The parenchyma is extremely soft, and tears from the slightest pressure, so that the least rude handling may cause artificial cavities, which closely resemble newly formed abscesses, and may be easily mistaken for them. The granular texture has entirely disappeared, and, if the pus be removed from the lungs by means of careful pressure and washing, we find that its parenchyma has again assumed its spongy and cellular arrangement.

The bronchi, especially their terminal branches, exhibit various appearances: in the first stage their mucous membrane is reddened and swollen; at a later period they become paler, and almost always contain, at first a reddish, and then a whitish, purulent, dissolving exudation. The blood-vessels are frequently obstructed with similar coagulations.

These are the three stages which the so-called acute pneumonia passes through in well marked cases; the third at the same time forms a natural and common mode of termination, which may be, and often is, a fatal one, but by no means necessarily so; for, on the contrary, the lung may return to its normal state, after the partial expectoration and partial absorption of the pus. There is no other, and, in particular, no earlier stage than that of the above-described inflammatory engorgement. That which is regarded by Stokes as the first stage of pneumonia, is any thing else rather than an inflammatory affection; for the bright red appearance of the lungs, or portions of them, which he attributes to arterial injection, and regards as in the earliest stage of inflammation, is

a. Always owing to anæmia, and frequently to a high degree of it.

b. The affected parts are puffed up, but without turgor and firmness, owing to deficient injection of their capillaries; they collapse readily, and not a trace of swelling of their tissues is present.

c. It always occurs whenever, in consequence of paralysis of the heart, or of excessive inspissation of the blood, an injection of the capillaries of the lungs cannot take place, for then the little blood which remains acquires a bright-red color from repeated exposure to the influence of the atmospheric oxygen in the hasty and repeated inspirations which attend the agony of death. Hence we see this condition present either in large sections or in small circumscribed portions of otherwise healthy lungs; or in

the neighborhood of hyperæmia and stasis, in many cases of asphyxia in adults and new-born children, in the train of rapidly exhausting diarrhœas, such as Asiatic cholera, and after extensive burns of the common integuments.

Before proceeding farther, it will be proper for us to add to the above sketch of pneumonia the conclusions which exact pathologico-anatomical investigations upon the cadaver, and physical examination during life, justify us in drawing, concerning the seat and nature of the pneumonic process. We think this the more important, as the conclusions will not only exert a marked influence upon what is to follow, but will also receive fresh corroboration therefrom.

As regards the seat of pneumonia we have already asserted, when speaking of the origin of the granular texture of hepatized lungs, that this is within the walls of the air-cells. The *exudation* which forms the granulations is preceded by the *secretion* of a viscid, tenacious, reddish-brown fluid in the cavities of the air-cells, which gives rise to the well-known crepitant rattle; this has disappeared when hepatization is fully formed, and its place is supplied by a plastic exudation. The granulations are roundish, and at first of a dark-red color, hardish, fragile; they seem to have coalesced with the swollen, dark-red walls of the air-cells, so that their isolation and removal can only be effected with great difficulty. Next the inflammatory turgor and redness diminishes; the granulations become paler, grayish-red, and finally grayish-yellow, softer, and increase somewhat in size. Soon the secretion of a glutinous mucus sets in around them, and breaks up their close adhesion to the walls of the air-cells, whence they and their swelling become more evident; as the walls of the cells which surround them are light-reddish in color, the granulations become more perceptible the paler they are. Finally they break down into a purulent fluid, which becomes muco-purulent from admixture with the above-mentioned glutinous mucus. Hence the *seat* of pneumonia is in the pulmonary mucous membrane, i. e., that which clothes the cavities of the air-cells, and its products are deposited within their walls; from the period which this takes place, i. e., from the stage of red hepatization onwards, the pneumonic process consists in a breaking down and transformation of the exudation, under the influence of an inflammatory process which has sunken beneath its acme. These conclusions are still farther strengthened by the following reflections:

a. If the granulations be regarded as air-cells, which have

become obliterated by the swelling of their walls, they could neither exhibit the pathologico-anatomical appearances which we have described, nor undergo the changes which we see they do.

b. Even the most considerable swelling of the air-cells could not produce the increased volume of hepatized lungs, while our theory readily explains this.

c. If the purulent infiltration, in the third stage, were an actual suppuration of the interstitial tissue, then a recovery from this could not possibly occur without the formation of abscess and solution of continuity, which it is well known is extremely rare after pneumonia; again, a lung when in a state of purulent infiltration presents a perfectly uncorroded, spongy, and cellular texture, and a resolution of it readily takes place by the partial absorption, and partial expectoration of the dissolved exudation which forms it, without the least ulcerous destruction of the lungs.

d. The same inflammatory process is generally present simultaneously in the terminal branches of the bronchi of an inflamed lung.

From all that has been said, it seems to us that even at this early period we are justified in calling pneumonia *a croupous process upon the pulmonary mucous membrane*, i. e., a *parenchymatous croup.* It exhibits even within the circumscribed limits of its pathologico-anatomical elements, a perfect resemblance to the croupous processes upon other mucous membranes; but we will develope this theory from higher and various points of view.

We frequently find all three stages of pneumonia coexisting simultaneously in one lung, and in fact in the various transition stages from one to the other; thus we generally find purulent infiltration and gray hepatization in the middle and toward the deeper seated portions of the lungs; above these, grayish-red and red hepatization, and inflammatory engorgement around the whole; finally, simple stasis and various grades of acute œdema are present in the adjacent parts.

Pneumonia may prove fatal in either of these stages, and may also retrograde from each to the normal condition. Besides the above described termination in purulent infiltration, it may, in rare instances, form abscesses, or pass over into induration, or into other conditions, of which it will be more proper to treat at a later period.

If the disease has not only reached the third stage, but also

commenced to retrograde towards the normal state, we notice the following phenomena: The purulent infiltration gradually lessens, and an exhalation of serum commences from the mucous membrane of the air-cells; from admixture with this, the still remaining pus is rendered thinner and thinner, and finally is changed into a turbid and flocculent serosity, which quickly becomes frothy, as the air commences to penetrate the air-cells almost simultaneously. The parenchyma at the same time becomes paler, more grayish and yellowish, and retains this color for a long time; it crepitates less than in the normal state; it is softer and moister; also œdematous to a certain degree, and easily torn.

The lung may also retrograde from the second stage, that of red hepatization, to the normal state, without passing through that of purulent solution of the exudation. This process is doubtless one of the most difficult undertakings which the curative powers of nature succeed in accomplishing, for it always takes place slowly, and in fact the more so, the more plastic the exudation is on the one hand, and the greater the exhaustion of the patient on the other, owing either to the severity of the disease, or to the effects of active medical treatment, or to both. The granulations then, together with the parenchyma, gradually become paler, and the former are melted down, layer by layer, by a serous fluid which is secreted from the walls of the cells. The lung still retains its granular texture, but the granulations become smaller, of a pale-reddish, or reddish-gray color, and are bathed in a fluid which is intermixed with rather consistent, pale-reddish, or whitish flocculi, and which gradually becomes frothy from admixture with air. When the granulations are thus finally melted down, the parenchyma remains in a state of serous infiltration; it is also somewhat redder, firmer, and denser, owing doubtless to a still existing partial infarctus of the walls of the air-cells, and of the interstitial cellular tissue. This retrograde process does not always progress with equal rapidity in all parts, and we often find more or less densely hepatized patches seated in the midst of tissue which has made some advance towards resolution. The diagnosis is then easy.

Finally, pneumonia may also retrograde from the first stage, that of inflammatory engorgement; and in fact very frequently does so, especially when aided by judicious medical treatment. The inflammatory stasis, after having deposited a moderate infiltration of turbid serous fluid, changes to simple stasis, after the resolution of which the lung recovers its normal condition, but

may readily become the seat of simple hyperæmia, which may as readily relapse into inflammatory stasis.

Pulmonary abscess. We have already become acquainted with the termination of pneumonia in purulent infiltration, i. e., in purulent solution of the exudation, which we have seen occurs without the least ulcerous destruction of the parenchyma. The opposite of this takes place when abscesses are formed in the lungs. This transition is extremely rare, but this infrequency will excite as little surprise if we retain our views of the nature and seat of pneumonia, as it will require a great deal if we adopt the explanations of Laennec. The circumstances under which it arises, and the manner in which it takes place, are but little known. Of all theories advanced, that is most in accordance with the nature of the pneumonic process, which attributes it to a peculiar quality of the exudation, under the influence of which the pulmonary mucous membrane (which has already lost its epithelial covering) and the other component tissues of the parenchyma break down in a process of suppuration. This supposition gathers fresh support from the occurrence of analogous processes in the course of true croup, but still more frequently in that of other exudative processes upon mucous membranes.

A newly formed, fresh pulmonary abscess is a cavern of irregular form, filled with pus, and surrounded by a parenchyma which is softened by and infiltrated with purulent matter; the walls are shaggy, and shreds of them hang down into the cavity of the abscess. They resemble very closely those artificial solutions of continuity produced by careless handling of the lungs when in a state of purulent infiltration, or by rude attempts to tear them loose from pleuritic adhesions: we have already cautioned against their being mistaken for true pulmonary abscesses.

The abscess either enlarges in the same way that it originated, i. e. by the continued purulent solution of the exudation, and of the parenchyma about its walls, or else by the confluence of several smaller abscesses. As a rule, the suppuration extends over the whole of the inflamed portion of the lung, hence the abscesses which follow lobar pneumonia are always of considerable size; one or more bronchial tubes may open into them with oblique or transverse mouths, and their tissues also become the seat of purulent solution. These abscesses form the true, but extremely rare, *ulcerous pulmonary phthisis*, consequent to simple inflammation.

It proves fatal, either from the addition of fresh pneumonia in its neighborhood, or of pleurisy, or of hectic fever, from the absorption of pus into the blood. In rare cases it perforates the pleura, and leads to suppuration of the adjacent parts, after having excited pleurisy and adhesion of the lungs to the walls of the chest. Still more rarely does it open into the cavity of the pleura before pleurisy and adhesion have been effected, and is then followed by general or circumscribed pleurisy. If one or more bronchi open into the abscess, pneumo-thorax will be added, and the pleuritic effusion may be ejected through the air-passages; an occurrence, however, which takes place more frequently from a reverse succession of events, viz., from primitive pleurisy, followed by consecutive corrosion and suppuration of the pleura. [See Pleurisy.) Finally, pulmonary gangrene may arise in the neighborhood of abscesses, and the purulent solution of the tissues may be changed into gangrenous ichoration.

If an abscess has existed for a long time, its inner wall will be found smooth, its form nearly roundish, and a secondary interstitial inflammation will have taken place, and transformed the surrounding parenchyma into a cellulo-fibrous tissue, which encloses the cavity and isolates it from the rest of the lungs. The perfect closure of an abscess is the more difficult the greater its size, but it ultimately takes place by adhesion of its walls; during this process the bronchi which opened into it become obliterated; if it were large, the walls of the chest sink in over it, and if seated near the surface of the lungs, this latter becomes puckered and contracted over the cicatrix.

Pulmonary abscesses may be mistaken for tuberculous vomicæ; also for certain accumulations of pus which arise from secondary inflammation of the capillary vessels of the lungs, of which we will treat more fully subsequently; or even for saccular dilatation of the bronchi. The diagnosis may easily be made by a comparison of the positive characteristics which attend each of these affections.

Induration. Under certain conditions hepatization does not pass into purulent infiltration, or abscess, but into induration; then the red products of inflammation become grayish-red and finally gray, and instead of breaking down they become more compact and indurated, and form the so-called *indurated hepatization*, which has occasionally, although not justly, been regarded as the result of chronic pneumonia. In it the lung is firm, but fragile and pale; it has lost somewhat of the increased size

which it attained when in the state of red hepatization, but it still retains its granular texture, which even becomes still more evident, as the granules, although somewhat smaller, are more compact, and hence more marked.

This condition may last for a long time, and then is always followed by a cachectic condition, but by dropsy in particular, and often proves fatal; or the induration may be gradually resolved, or pass over into obliteration of the air-cells, and consequent atrophy of the lungs.

The retrograde curative process from indurated hepatization is nearest similar to that of resolution from the second stage of pneumonia, for an exhalation of serous fluid takes place from the inner walls of the air-cells, and forms a menstruum by which the indurated granulations are corroded and consumed. As the granulations become smaller, we find this fluid growing turbid and flocculent, and as soon as air again penetrates the air-cells it becomes frothy.

In other cases, the air-cells contract over the granulations, coalesce with them around their whole circumference, and thus become obliterated; their tissues then become transformed into a cellulo-fibrous texture, in which organization the granulations doubtless also take part. Finally, a sinking in of part of the chest may follow this termination, unless a serous effusion fill up the empty space, or bronchial dilatation arise, or both simultaneously. It appears, however, to be less frequently a consequence of the above described croupous pneumonia, than of a creeping inflammation of the interstitial tissue, i. e., of *interstitial pneumonia.*

Such is a sketch of croupous pneumonia in general, the materials for which were taken from the appearances which it presents, when a *primitive* affection; but it frequently occurs as a *secondary* process. It generally runs an *acute* course, as it usually passes through its different stages in from fourteen days to three weeks; although in very rare and rapid cases it does the same in three or four days. At times, however, it is a *chronic* affection, and then advances with equal slowness in all its stages, or else persists for a long time in some one of them; but even then it presents no essential differences from the description which we have already given, for it, like the acute form, generally ends in purulent infiltration—rarely in abscess, or induration. It is very different, however, from that affection which we generally find treated of in manuals of pathology under the name of

chronic inflammation; with this we will become better acquainted, when we come to treat of *inflammation of the interstitial tissue.*

We notice various differences in the original *extension* of pneumonia, which are of importance on account of their connection with the nature of this disease. Thus lobar pneumonia is generally a primitive affection, while the lobular and vesicular varieties are generally secondary processes. We have already treated of lobar pneumonia: we have only to add that it always attacks a whole lobe, or at least a large portion of one; that it often attacks a whole lung, and extends to the other, and often does not prove fatal until the last remaining portion of the latter becomes inflamed. Under such circumstances it is generally a primitive affection; its special seat is in the lower lobes, and the right lung is most frequently inflamed; but both these rules are subject to many exceptions.

It is called *lobular pneumonia* when it attacks smaller sections of the lungs, i. e., varying numbers of single lobules, or single aggregations of lobules, between which we find the parenchyma in a relatively normal condition. This variety must be carefully distinguished from those lobular hepatizations which arise in consequence of an inequality in the course of lobar pneumonia, the remaining parenchyma being still in a state of inflammatory engorgement.

Finally, pneumonia may be confined to single air-cells, and is then styled *vesicular pneumonia.* In this variety the disease runs through its stages of inflammatory engorgement, hepatization, and purulent infiltration, in a single air-cell, or may pass over into induration and obliteration of it. This indurated hepatization of single air-cells is doubtless that condition which writers on pathological anatomy treat of under the name of *Bayle's pulmonary granulations,* and about which there has been so much unnecessary disputation. It undoubtedly is the result of inflammation, and Andral is justified on the one hand in upholding this view; but as the products of inflammation may under certain circumstances take on a tuberculous nature, Laennec and Louis are also right, on the other hand, when they declare it to be tuberculous. It represents, as will be proved subsequently, the tuberculous infiltration of single air-cells.

Lobular and vesicular pneumonias, as we have before remarked, are generally secondary processes.

It is of the greatest importance for us to examine into the differences which the products of inflammation offer with regard

to their plasticity, for these depend most intimately upon the condition of the blood, and hence upon the nature of the general affection. In place of a plastic, hepatizing exudation, we may according to circumstances find either a serous, flocculent, and turbid effusion, or a gelatinous and glutinous infiltration, or a sero-purulent, or even an ichorous one. Neither of these can ever lead to a granular hepatization of the parenchyma, as they are all deficient in coagulable matter, but render the lung dense and spleen-like; besides other marks of inflammatory stasis, it is discolored, and somewhat firm to the touch, but a close examination shows it to be softened and easily torn. Primitive acute pneumonia usually deposits a plastic hepatizing exudation, which passes through the various alterations which have already been described; but the last mentioned effusions and infiltrations are commonly the result either of slow, asthenic, and hypostatic inflammations, or still more frequently of secondary pneumonic processes; they represent secondary exudative processes, which often degenerate into circumscribed gangrene.

One of these infiltrations, viz., the *gelatinous*, requires especial examination. It differs widely from that condition which Laennec describes as gelatinous tubercular infiltration, and Andral sets down as a result of inflammation, without further comment, but which we regard as the product of inflammation of the interstitial cellular tissue (see page 114). In place of the usual plastic hepatizing exudation we find a gelatinous, viscid, at times almost frog-spawn-like fluid effused into the air-cells; it is either grayish, grayish-yellow, grayish-red, or brownish-red in color; either clear and transparent, or else flocculent and turbid; the parenchyma is pale-reddish, or more frequently reddish-brown, and easily torn. The pneumonia which deposits these unplastic effusions generally arises around pulmonary tubercles or tubercular infiltrations, or hepatizations which are undergoing a transformation into tubercular infiltration; it is commonly developed towards the end of pulmonary phthisis, and frequently involves all the parenchyma which is not occupied by miliary tubercles or tubercular infiltrations. But we occasionally find it about the circumference of extensive hepatizations, especially in the emphysematous edges of hepatized lobes, which are incapable of admitting a thicker effusion. Finally, it occurs whenever, in consequence of an original vice in the constitution, or of previous profuse exudations, plastic materials for the formation of a coagulable hepatizing product are not at hand.

We must also allude to certain transformations which the plastic, fibrinous, hepatizing products of inflammation undergo, in consequence of the existence of a general constitutional dyscrasia. We refer to the very frequent alteration of the same into tubercle, in the form of *tubercular infiltration;* and to the very rare transformation, or perhaps organization of them into a medullary cancerous tissue, in the form of *cancerous infiltration*, or *infiltrated cancer of the lungs.* We will treat of these two states more particularly when we come to the consideration of tubercle and cancer of the lungs.

The so-called *hypostatic pneumonia of Piorry* is a peculiar variety, which developes itself out of that passive stasis, or pulmonary hypostasis, of which we have already treated. It bears in a marked degree the stamp of an asthenic inflammation, for it is generally slow in its course, persists for a long time in the stage of stasis, with dark and livid coloring of the parenchyma, from which there gradually is formed either a general or partial, lax, soft, livid-brown hepatization, which commences in single and isolated spots, to which it may be confined, while the remaining portions, and even the whole at times, may become the seat of a sero- or gelatino-purulent infiltration, without ever having presented a trace of hepatization. It is the substratum of most of the so-called latent inflammations of the lungs.

Primitive pneumonia generally attacks robust adult persons; still delicate individuals are frequently enough subjected to it, and may even at times possess a remarkable predisposition for it. Up to extreme old age it is generally lobar, as it commonly attacks at least the whole of one lobe, and deposits a plastic, hepatizing product; it also occurs in young persons, and even in new-born children, and then presents various peculiarities, for the granular texture is commonly but very faintly evident, owing doubtless to the density of their lungs, and the smallness of their air-cells. In children, the termination in abscess is relatively more frequent than in adults, and lobular pneumonia is more common, although the simple catarrhal variety is often mistaken for the croupous. Primitive croupous pneumonia arises under the influence of a peculiar constitution of the atmosphere, which predisposes to inflammation in general, followed by profuse plastic exudations, and then may be induced by various well-known and often trivial exciting causes. Hence it is important for us to mention the combinations into which it enters, as they all proceed from a common proximate cause, viz., a peculiar spontaneous

affection of the blood; thus, a constant phenomenon is a sympathetic affection of the pulmonary pleura of the inflamed lobe, marked by the exudation of a thin, but plastic coating.

The condition of the blood, in which croupous pneumonia participates in company with the other primitive exudative inflammations, claims particular attention here, as it occurs in its most marked form and extreme degree. In consequence of it we always find fibrinous coagulæ in the cavities of the heart and in the large vessels, but in particular in those branches of the pulmonary arteries which supply the inflamed lobe; they are characterized by their yellowish-green color, their firmness, by a more or less evident purulent transformation in their centres, by their similarity to the exudations upon membranous expansions, by their being interwoven between the columnæ carneæ of the heart, and their partial adhesion to the endo-cardium and lining membrane of the great vessels; all of which, together with the inflammatory irritation which is found about their points of attachment, tend to prove that they were formed, at least in part, during life.

Pneumonia, if we except the constant coexistence of pleurisy, may occur as an isolated and independent affection, yet it is frequently enough combined with similar exudative processes upon other tissues; among these belong the very frequent croupous affection of the terminal branches of the bronchi, which varies in degree, but is marked by the presence of cream-like, purulent, dissolving coagulæ in them. In children is often associated with tracheal croup, with croupous affection of other mucous, and of serous membranes, viz., with pleuritis, pericarditis, meningitis, &c.

The combination of pneumonia with secondary inflammation of the lining membrane of the blood-vessels, is a point of great interest; it arises in consequence of a spontaneous coagulation of the fibrine, in extreme degrees of the inflammatory affection of the blood (hæmitis), and of its breaking down into pus, and is particularly apt to occur in the form of inflammation of the spleen, followed by its natural termination in ulcerous splenic phthisis.

Primitive pneumonia proves fatal from paralysis of the lungs; by the supervention of pulmonary œdema, or of the other combinations into which it enters; from the extreme degree of the affection of the blood, and its spontaneous coagulation in the heart and great vessels; by the occurrence of acute softening of the stomach and œsophagus.

Secondary pneumonias frequently arise in the train of inflammations of other organs, when the blood has taken on a consecutive affection similar to the spontaneous one already described. They frequently accompany those specific processes which are allied to the exudative, and hence occur with great frequency during the course of acute exanthematic affections. In both the above instances, the pneumonias are generally *lobar*. On the other hand, these secondary pulmonary inflammations may occur as so-called metastases, in the train of various acute dyscrasias of the blood, which degenerate into croupous affections, and among these, again, belong many exanthematic processes, also the typhous, and tuberculous; they are generally *lobular*, and even *vesicular*. Among these we may also arrange many of the so-called latent, or symptomatic, and, as already remarked, metastatic inflammations. They combine themselves, especially under the last named conditions, with exudative, croupous processes upon other mucous and serous membranes, which deposit products of varying degrees of plasticity.

From all that has been said, the croupous nature of pneumonia becomes distinctly evident; it is always based either upon a primary and spontaneous, or upon a secondary affection of the blood, which is similar in secondary pneumonia to what it is in those other metastatic croupous processes which are frequently combined with it, and also perhaps in its essential features both in primitive pneumonia and in the other primitive croupous processes upon the mucous membranes of the mouth and throat, but in particular of the respiratory organs. But as the peculiarities of constitution and age vary, and also the external influences under which buccal, pharyngeal, tracheal, bronchial croup, and also croupous pneumonia arise, so may the affection of the blood differ in all these, although perhaps only slightly. If we regard the degree of plasticity of the exudation, we must consider pneumonia as a pulmonary croup in its most exquisite form. It usurps the place, in adults, of the pharyngeal and tracheal croups which occur so frequently in the earlier periods of life; while bronchial croup forms the transition disease between the two. From croupous we proceed to the consideration of the *typhous pneumonia*, as the pathologico-anatomical characteristics of the two diseases are very similar.*

* *Diagnosis.—Acute primary pneumonia* is much more common in males than females: of 540 cases collected by Grisolle, Hughes,

TYPHOUS PNEUMONIA.

(*Pneumo-Typhus.*)

The pneumonic process is frequently associated with the typhous; but its relation to the latter, in particular to the

and others, 404 were in males, and only 136 in females. The lower lobes are more frequently inflamed than the upper: Andral and Bouillaud make the proportion of three to two; Grisolle, four to three; Briquet, two to one. In 189 cases collected by Andral and Hughes the lower lobes were inflamed as often as 109 times, the upper only 35 times; in infancy and extreme old age, however, the upper lobes seem to be equally, or even more frequently inflamed than the lower. The right lung is also more frequently inflamed than the left: this fact was known by Morgagni, Baglivi, and many others in former times; of 2402 cases collected by Hughes, Grisolle, Chomel, Andral, Bouillaud, Barth, Forbes, Lombard, Hourmann, Dechambre, Valleix, Vernois, Berg, and Pelletan, 1372 were on the right side, and only 696 on the left; this agrees very nearly with Grisolle's proportion, viz., eleven on the right to six on the left; Forbes makes the proportion five to three. Hence, in the majority of cases, we look for the signs of pneumonia at the base of the right lung, behind. Double pneumonia occurs, according to Grisolle and Barth, only once in 16 cases; but Hughes found it 19 times out of 101; Chomel, 16 times out of 59 cases; in 1640 other cases it was present 287 times; and Forbes calculates that it occurs as often as one case in five.

Chomel regards well marked shivering as infinitely more common a precursor of pneumonia than of any other inflammation; hence, whenever he meets with it, he at once turns his attention to the lungs. Andral agrees that more or less violent shivering ordinarily precedes the fever, pain, and cough; still the chill may be entirely absent, and fever, with slight cough and hurried breathing, which the patient may not notice, may be the only symptoms.

In those obscure cases in which cough and expectoration are almost or totally wanting, Bright and Addison regard a peculiar pungent heat of skin as the most constant and conclusive of all the symptoms of pneumonia, in a diagnostic point of view; by this symptom alone they have repeatedly pronounced pneumonia to exist, before asking a single question, or making the slightest examination of the chest.

In the first stage the patient has a deep red flush upon the cheeks, most marked on the affected side. The cough is short and suppressed, instead of loud and deep, as in bronchitis; there is little or

local typhous process upon the intestinal mucous membrane, is not always the same, and hence its importance varies.

In all cases of typhus, however, even in the most marked cases of ileo-typhus, we find the well-known hypostasis in the inferior lobes of the lungs, which often increases to pneumonia, depositing a gelatinous, glutinous, soft product, similar to the

no expectoration for the first day or two, except some bronchial mucus, mixed with saliva. When the pleura is but slightly affected, there is a sense of oppression and uneasiness in the chest, or a more or less painful weight, and an annoying and deep-seated sense of heat in the side, but generally no real pain. The most common character of the pulse is its frequency and largeness; but when the inflammation is very intense it is often remarkably small, soft, and frequent, but feeble, so similar in fact to the pulse of weakness, that the uninitiated may regard it as such; but it is the pulse of oppression, and if the patient be made to cough, or take a deep inspiration, it instantly becomes full and hard—it also rises after a free bleeding. The hurried respiration and dyspnœa are generally proportionate to the extent and severity of the inflammation, except when it is located in the upper lobes, for then the dyspnœa is much greater than in an equally severe affection of the base of the lungs. If the dyspnœa be great, the face often becomes livid; the patient, however, is often unaware of any difficulty of breathing, but the frequency of respiration will always strike the attention of the observant physician. The disturbance of the breathing is always at its maximum in the first stage, and cannot be increased afterwards except by an extension of the inflammation, or the supervention of a pleuritic effusion.

The percussion sound does not commence to be obscured before the second or third day; rapid and extensive dulness rather points to pleurisy with effusion. Crepitant rattle is characteristic when present, but in nearly one-fourth of all cases of pneumonia it is obscured by bronchial rales; as long as the normal, breezy, vesicular murmur is heard through and louder than the crepitant rattle, the inflammation is not far advanced; when it supplies the place of the vesicular murmur entirely, then the first stage is fully formed. As soon as the crepitant rattle becomes well marked, the expectoration begins to be characteristic; usually by the second or third day it assumes some shade of red, particularly that of iron-rust; smaller quantities of blood in the expectoration give it a sea-green, tawny, orange, saffron, yellowish, or dull greenish tinge; these colors are frequently intermixed in stripes or spots in the same spit-cup. At the same time the sputa become more tenacious and viscid, but not sufficiently so to adhere to the vessel, and are mixed with minute, equal-sized bubbles of air. If there be only a few viscid and tawny sputa amid

typhous bronchial and intestinal secretion, and in accordance with the general nature of the typhous dyscrasia. It is the result of an adynamic state, and commonly has no other more intimate

a mass of mucous expectoration, with unequal-sized, large and small bubbles, there is much bronchitis present.

When semi-transparent red sputa unite to form a gelatinous and trembling mass, which is so viscid and tenacious that the vessel containing it may be turned upside down without its flowing out, the second stage is fully formed; the peculiar, whiffing, bronchial respiration takes the place of the crepitant rattle, and the chest is completely dull on percussion, and offers a sense of great resistance to the percussing finger; the voice resounds in the chest as if it were formed directly under the ear of the auscultator (bronchophony). When red hepatization is well advanced the expectoration becomes slight and variable in quantity, and generally consists of a small proportion of pituitous sputa, more or less viscid and vitriform, or of a whitish or yellowish mucus, for the bulk of the plastic effusion is now firmly plugged into the air-cells, and cannot be expectorated until resolution ensues. Owing to the obstruction to the free circulation through the lungs, the blood now becomes more venous in its character, and is apt to produce a narcotic effect upon the brain, marked by more or less delirium or coma; as the inferior vena cava cannot empty itself freely, more or less passive congestion of the liver ensues, and the whole of the skin may take on a yellowish tinge, which may increase to actual jaundice.

It is commonly supposed that resolution generally follows the third stage, viz., that of purulent infiltration, but when pneumonia progresses thus far, it is almost always fatal; the majority of the deaths take place just at the commencement of it. The expectoration then usually becomes difficult and scanty, or is altogether suppressed; their secretion still goes on, but they cannot be ejected; hence they accumulate in the air-passages, and produce, according to Fournet, a mucous rattle with large bubbles, heard only during inspiration. Or the expectoration may become grayish, inodorous, and truly purulent, flowing into one soft mass; in other cases they lose their jelly-like appearance, great viscidity, and red color, and consist of a liquid of the consistence of gum-water, of a more or less deep brownish-red color, sometimes even black, and bearing considerable resemblance to prune or liquorice juice. When present, this expectoration is characteristic of purulent infiltration; a peculiar sallowness of the skin, like that which attends cancerous affections, is often so well marked as to serve better than any other sign in diagnosing purulent infiltration.

But the more frequent termination is resolution. According to Andral, there is no disease in which the occurrence of critical sweats

or metastatic connection with the typhous affection of the intestinal mucous membrane.

Pneumo-typhus, however, assumes such a connection when

seems more perfectly demonstrated than in this. But we should distrust them unless attended with a lateritious sediment in the urine. The urine, from being of a deep red color in the acute stage, becomes turbid, and then minute, crystalline, reddish, glittering particles are precipitated, and the water assumes its natural, clear, amber color; if the urine still remains dark-colored or turbid a relapse is apt to occur, and if the peculiar sediment is wanting, the probability is that the lungs are tuberculous. [SCHOENLEIN.] About the time of convalescence, the urine also becomes precipitable by nitric acid; in fatal cases no precipitate ensues. [GRISOLLE.] The expectoration, too, is often peculiar; the cough becomes moist, and opaque, yellowish, slightly greenish, or almost white, woolly or cottony globular masses are expectorated with ease; they do not flow together, but remain distinct and float in water. If resolution takes place from the first stage, crepitant rattle gradually gives way to weak but normal vesicular murmur; the expectoration becomes easy, and is no longer marked by the presence of minute, equal-sized bubbles, but the large and small unequal-sized bubbles, peculiar to the bronchitic sputa, are noticed.

Delirium is an unfavorable symptom, except when it only occurs at night, or in nervous or hysterical persons. Coma, especially in old people, is still more unfavorable. Jaundice is an accident of but little moment. Recovery seldom takes place if the pulse runs over 130 or 140; but if the pulse becomes slow, while the respiration increases in frequency and difficulty, the termination is almost invariably a fatal one. Dyspnœa modifies the prognosis more than any other sign; if considerable, it is always an alarming sign. Inflammation of the upper lobes is more dangerous than that of the lower. If the urine, after having been turbid, becomes clear before the fourth day, a fatal result may be anticipated. Pneumonias not attended with expectoration do not seem more dangerous than others.

The termination in abscess is rare; there is not much hectic fever; there is a dull, muddy sallowness of the complexion; the expectoration is of a brownish, or greenish-yellow color, has an intense putrid odor, and resembles rotten eggs in appearance and smell, so that it has become familiar in the profession that this variety of expectoration holds out a more favorable prospect of recovery to the patient, than the ordinary purulent expectoration which attends phthisis. The percussion-sound over the abscess becomes fuller and less dull, and if the abscess be large, empty, and near the surface of the lungs, it becomes tympanitic, but empty. In rare instances, the cracked-pot sound may be produced, but it is only very large ab-

the typhous process localizes itself originally upon the pulmonary mucous membrane, to the total or partial exclusion of other tissues, especially those of the intestines, to which it usually exhibits a marked preference. It then is distinctly a *primitive pneumo-typhus*. The same holds good when it appears in its genuine form upon the lungs on account of a great severity and

scesses which give forth a metallic resonance. These abscesses can rarely be diagnosed before their walls become organized, for it is then only that cavernous and mucous rattles, pectoriloquy, cavernous cough, and respiration and other sounds are transmitted to the walls of the chest.

Acute secondary pneumonia is a much more serious disease than the primary form: according to Louis, the latter rarely proves fatal when it occurs in a previously healthy subject; while one-fourth, or even one-third, of all cases of the former terminate fatally. This is not surprising, for the patient is not only enfeebled by previous disease, but both lungs become inflamed in nearly one-half of the whole number of cases.

Senile pneumonia is one of the most frequent and fatal diseases of aged persons. It is frequently latent; there may be no chill or pain, and the patient only feels ill at ease and altogether more feeble; there may be no cough or disturbance of breathing, even when the patient is subject to asthma, or chronic cough; the patient may get up well in the morning, but feels fatigued in the course of the day, lies down and dies after a short struggle. The pain in the side is seldom sharp; even when the pleura is affected, there is often a mere sense of soreness and uneasiness over the affected part; and when stitch in the side is present, it rarely lasts more than two or three days, and then is replaced by a diffused, dull pain. When the upper lobes are inflamed, which is often the case, the breathing is almost always much distressed, and constant orthopnœa is present. True crepitant rattle is rarely heard; mucous and sonorous rales are more frequent, and at times an unusually dry resonance, like that produced by the crackling of parchment is heard. Bronchial respiration is common. In the greater number of cases the patient, without being positively irrational, still seems to be lost; they do not understand what is said to them, and their answers may have no reference to the question, or they remain taciturn and take no notice of any thing. During the progress of the disease, delirium sets in and lapses more or less speedily into coma, especially when the suppurative stage commences. The expectoration is rarely characteristic; the signs from the tongue, skin, bowels and urine are frequently too deceptive to be relied upon; and the pulse, too, is apt to misguide us. [HOURMANN and DECHAMBRE.]

extension of the disease, or of an imperfect localization of it upon the intestinal mucous membrane. Under the latter circumstances it is a *secondary pneumo-typhus.*

Primitive pneumo-typhus is a croupous, lobar pneumonia, characterized by the livid and violet color of the pulmonary parenchyma during its first stage, and by a dirty brownish-red, or chocolate-colored, and soft hepatization during its second, which breaks down rapidly, when the affection of the blood and the want of plasticity of the exudation is extreme. It is perhaps always combined with bronchial-typhus, and the bronchial glands exhibit the alterations peculiar to this latter variety. It may exist either without or with only a slight secondary affection of the intestinal mucous membrane, and it and bronchial-typhus doubtless form the substratum, if not of all, at least of the most, of those cases of typhus which run their course without any local affection of the intestines. This is true especially of exanthematic typhus fever. Like genuine pneumonia, it combines itself with pleurisy.

The *genuine secondary pneumo-typhus* occurs in connection with an imperfectly developed ileo-typhus, and presents the same pathologico-anatomical characters as the primitive, but does not attain to the same degree of intensity and extent, except in those cases where obstructions arise to the further development of the original intestinal affection. It also enters in the same combinations, but especially with genuine secondary laryngo-typhus.

But secondary pneumo-typhus occurs far more frequently in a *degenerate* form, as a local expression of the degeneration of the collective typhous affection, and in fact in the form of a lobular or vesicular pneumonia, which deposits a purulent and dissolving product, and associates itself with great frequency with a laryngo-typhus which has changed into a degenerate croupous affection ; or it appears in the form of purulent and dissolving depositions in the interstitial tissue, coupled with inflammation of the capillary vessels, and circumscribed depositions of pus in the lungs ; or, finally, in that of gangrene of the lungs, especially in the form of circumscribed gangrene, or gangrenous eschar of the lungs.*

* *Diagnosis.*—In typhoid pneumonia the local symptoms are by no means prominent : although there may be pain, cough, and very imperfect breathing, still the patient's intellect may be so obtuse that he is not aware of them, or at least rarely complains of his chest,

2. *Catarrhal Pneumonia.*

This variety has not been described by former authors, on account of the infrequency of its occurrence; or else has been mistaken for croupous pneumonia in children, in whom it occurs more frequently, because of the slight degree of development of the granular texture in hepatization of the lungs of young persons. It has been remarked that it is a rare disease, especially in adults, while it occurs with greater frequency in children. It is the earliest and most frequent form of catarrh in children; at a later period of life, bronchial and finally tracheal catarrhs become more frequent than it. Thus, it will be seen that it runs an opposite course to that of croupous affections of the respiratory mucous membrane; for pharyngeal, laryngeal and tracheal croups are most common in young persons, while bronchial and pulmonary croup is most common in young adult persons. Catarrhal pneumonia is always lobular, and always coexists in connection with a catarrhal affection of the bronchi, which supply the diseased

even though extensive and fatal disease may exist. The pulse is small, quick, and weak; the skin is harsh, dry, and partially hot, or covered with a clammy sweat, and sometimes spotted with petechiæ; the face has a dusky-red hue; the tongue is furred, brown, and dry; stools dark-colored; the urine scanty, turbid, and ammoniacal. According to Stokes, this is one of the diseases in which the practical utility of auscultation and percussion is most frequently seen; again and again the stethescopist will detect inflammation of the lung which has occurred without cough, pain, dyspnœa, or expectoration; even where pleuritis is also present, the patient may feel no pain. It is important to know that the crepitant rale is very often absent in the first stage; in some cases, we only find a degree of dulness on percussion at the base of the lungs behind, coupled with feebleness of respiration there; the hepatization is so soft that even bronchial respiration may not be heard in the second stage, which is only marked by dulness on percussion and absence of respiratory murmur, whence the case may easily be mistaken for pleurisy with effusion. It is often insidious and deceitful in its career; for the patient frequently appears better immediately before its fatal termination, but the breathing suddenly becomes laborious, the action of the chest more irregular, rattling in the throat comes on, the limbs become cold, and death ensues. The presence of rusty expectoration is not unfavorable, but when dark grumous blood is found in the sputa the case will prove a severe one.

lobules; it is frequently found in the various catarrhal affections of children, especially in those who have suffered and died from catarrhus suffocativus and hooping-cough. Its seat is particularly in those lobuli which are near the surface of the lungs; numbers of these are often affected simultaneously, and are usually found bluish-red in color, dense and firm. The walls of the air-cells are often swollen to such an extent as to close their cavities, which in lesser degrees of tumefaction contain a watery, mucous, and only slightly frothy secretion; there is no trace of a granular texture. As the portions of the lungs immediately adjacent to the inflamed lobules are generally emphysematous and paler in color, the latter are depressed somewhat below the surface of the lungs; this, together with their darker color, renders them easily recognized.

This disease frequently proves fatal from the superaddition of pulmonary œdema and paralysis of the lungs, or from obstruction to the circulation, especially in the heart, owing to the emphysema.*

* *Diagnosis.*—The pneumonia of children is a most difficult disease to diagnose, as it is frequently unaccompanied by any well-marked symptoms, or marked by those which are considered characteristic of other diseases. The complication with cerebral symptoms, viz., pain in the head, delirium, coma, or convulsions, is by no means unusual, and always of a serious nature; it often becomes developed and makes great advances unnoticed, while the attention of the physician is directed to the brain; in these cases it only betrays itself by a slight cough, and even this may be wanting; the physician too often directs his treatment to the cerebral affection, and, while congratulating himself on his success, he has the mortification of losing his patient by a disease which he had not suspected. Violent convulsions sometimes occur, accompanied with twitchings or paralysis of some of the limbs; at other times the symptoms so closely resemble those of ordinary hydrocephalus, that, without having recourse to auscultation, the most acute practitioner may be deceived, and after death finds the brain quite natural, or in a state of scarcely appreciable congestion. Convulsions and coma are most common in the third stage of pneumonia.

In other instances, the degree of emaciation to which it reduces the patient, and the slowness of its advance, leads one to mistake it for phthisis; the child wastes daily, its skin hangs in wrinkles about its attenuated limbs, the abdomen becomes tumid and tender, the tongue grows red and dry, or is covered with aphthæ; the cough

3. *Inflammation of the Interstitial Cellular Tissue of the Lungs.*

INTERSTITIAL PNEUMONIA.

This disease as a rule is not correctly described in the works on pathological anatomy, for it is commonly regarded as a chronic form of croupous pneumonia, without any accurate determination of its seat in a particular tissue, which is the interstitial cellular, although the walls of the air-cells at times become affected by an extension of the inflammation, which then occasionally puts on a croupous character.

Its course is almost always chronic, and we very rarely have opportunities of studying it, except in its ultimate effects. From the few opportunities which we have had of witnessing it, we

now perhaps attracts notice, and the physician believes that he has failed to afford relief because the disease was in itself irremediable; at last the child dies, and great is the surprise to find no tubercle in any part of the body, but pneumonia with purulent infiltration of both lungs; a disease which ought to have been detected, and which probably might have been cured. [West.]

In some cases intense puerile respiration is the first physical sign. True crepitant ronchus is present in less than one-half of all the cases, while the sub-crepitant is heard in nearly four-fifths; in some cases the latter is succeeded directly by bronchial respiration, in others crepitant ronchus is interposed between the two; frequently sub-crepitant ronchus only will be present until the child begins to cry, when, at the moment of the deep inspiration which follows a fit of crying, distinct, true crepitation will be heard. When the true crepitant or sub-crepitant rattles have attained their greatest extent, the frequency of respiration is generally at its maximum, and diminishes when bronchial respiration and dulness on percussion indicate that the lung is hepatized, but the breathing almost always becomes irregular, for one or two slow inspirations are succeeded by three or four very hurried; then, if we raise the child from the recumbent posture to the sitting position, the great acceleration of the breathing at once shows that the diminished frequency of respiration is not the result of any favorable change. But whenever, with a diminution in the frequency of the respiration, the pulse sinks also, we have a sign of amendment on which the most implicit reliance may be placed. Bronchial respiration is only heard in about two-fifths of the cases, and when present it is a very grave sign, as nearly one-half die. [West.] The results of percussion are still less valuable than those of auscultation, for both lungs are generally affected, and we have no points of comparison except between the upper and lower parts of

judge that it commences originally in the cellular tissue situated in the interstices between the lobules and the smaller groups of the air-cells, for, if too great a quantity of pulmonary pigment be not present, this will appear of a pale-reddish color, and is swollen from the presence of an albuminous infiltration, while the air-cells will be found pale, and more or less compressed, in proportion to the swelling of the interstitial cellular tissue; but if the inflammation have extended to them, they will be reddened and occasionally granulated, although but slightly.

In the course of time the albuminous infiltration becomes organized, and coalesces with the interstitial cellular tissue so as to form a dense cellulo-fibrous substance, which obliterates the air-cells by the compression which it exerts upon them, and finally converts them into a similar cellular substance. We then

the chest; the dulness is generally appreciable when bronchial respiration sets in; and even if it should be necessary to percuss so gently as scarcely to elicit a distinct sound, yet the resistance of the walls of the chest makes the finger sensible of the presence of solid lung beneath. [WEST.]

In children from two to four years old, it generally sets in suddenly; the child who has gone to bed well, wakes up towards morning in a state of alarm, and refuses to be pacified; its face is flushed, skin burning hot, breathing hurried, and cough short; this storm usually subsides without treatment in twenty-four hours, and the disease passes into the second stage, while the physician is still delighted with the apparent success of his treatment.

In infants at the breast, there is feverishness for one or two days, increased towards evening, with pain in the head, great restlessness at night, and short hacking cough, which seems to cause the child no uneasiness, and is so slight as scarcely to be noticed by the mother. The thirst is considerable, tongue and lips are of a florid red and dry, bowels constipated, and vomiting not infrequent. The infant seizes the breast eagerly, nurses for a few moments with eagerness, then suddenly stops, and often begins to cry. In the healthy infant the tongue is applied to the roof of the mouth while sleeping, and breathing is performed through the nose; but as soon as the lungs become affected he breathes through his mouth, which is partly open. As the disease advances these peculiarities in nursing and breathing become more striking, for respiration becomes greatly hurried by the effort of nursing, so that he drops the nipple panting, and has not breath sufficient to bring the flow of milk. [WEST.]

In the second and third stages, either the rational or physical signs will be sufficiently marked to detect the disease in the great majority of instances.

find either firm whitish stripes, or misshapen masses of such substance, interwoven in the parenchyma of the lungs. They often creak under the knife which essays to divide them.

This is the usual alteration which portions of the lungs undergo in consequence of the so-called chronic pneumonia; in rare instances, however, a form of suppuration may ensue, which burrows around and dissects out the lobules; and it is possible that some pulmonary abscesses arise in this way.

Not unfrequently it is an independent and spontaneous disease, which creeps from one lobule to another, until many are affected; its most common seat is in the apices of the superior lobes, and, as we may infer from the frequent presence of corresponding pleuritic adhesions, it is usually combined with circumscribed pleurisy. The affected parts contract and draw in the surrounding healthy tissue after them, so as to present the appearance of cicatrices, which may be mistaken for such as arise after the closing of tubercular abscesses. Sinking in the walls of the chest at corresponding points, or dilatation of the bronchia, may also take place.

More frequently, however, it is a consecutive disease, arising out of the reactive inflammation which sets in around apoplectic effusions into the lungs, lacerations, pulmonic abscesses, tuberculous cavities, gangrenous destruction, &c.; it then leads to the formation of cicatrices, cysts, &c.

At times the fibro-cellular tissue which it forms contains a considerable quantity of black pulmonary pigment, and is then blackish-gray in spots or stripes, or else is equally stained of a bluish-black color.*

* *Diagnosis.*—Chronic pneumonia is a singularly rare disease; Chomel met with but two cases in sixteen years, and Laennec only five or six cases in his whole lifetime. The diagnosis, of course, is still incomplete.

As it is primarily an affection of the cellular substance of the lungs, the physical signs at first are merely some dulness on percussion, and feebleness of the respiratory murmur. The rational signs are not more conclusive: there is usually little or no fever, but the pulse is somewhat tense; there is a dull aching and a sense of heaviness in one or the other side of the chest, and some cough; the affected side is somewhat depressed, and moves but little when the patient breathes; the voice and cough resound so as to form imperfect bronchophony.

When the air-cells become diseased, there is irregular sub-

g. DEPOSITS IN THE LUNGS.

Metastatic Processes.

In consequence of the absorption of various matters into the circulation, or more rarely from a spontaneous disease of the blood itself, various affections arise, which will be treated of more fully under the head of "Diseases of the Blood," but to which we must turn some attention here, as they affect the lungs more frequently than any other organ. They occasion the deposition of various pseudo-plastic products, most frequently of pus, generally in several circumscribed portions of the lungs simultaneously. At other times fibrinous deposits in the lungs are found, with coagulæ in the capillary vessels, owing to a *capillary phlebitis*. All these deposits undergo alterations similar to those which the substances absorbed into the circulation are subject to.

As deleterious substances are most frequently formed in the veins, or at least are most often absorbed by them, and as the whole of the venous blood circulates through the lungs, we can readily understand why these deposits take place more frequently in the pulmonary organs than in any other.

It is a peculiar characteristic of these deposits that they take place in the superficial or peripheric portions of all parenchymatous organs; this is particularly the case in the lungs. We find mustard-seed, lentil, pea, bean, or even nut-sized deposits scattered through the lungs, and separated from one another by large patches of healthy tissue: the smaller they are, the more they resemble roundish granulations; while the greater their size, the more they depart from the roundish form and take on that of misshapen, angular, and branched masses. When large deposits are seated near the very surface of the lungs and verge upon the pulmonary pleura, they usually have a wedge shape, for they are thicker externally and gradually grow thinner towards their apices,

crepitant rattle, which slowly gives way to permanent bronchial respiration, bronchophony, and bronchial cough; there is increased dulness on percussion, and a sense of great resistance to the percussing finger; the sounds of the heart are transmitted with undue intensity. It is almost needless to add, that it is next to impossible to distinguish it from tubercular, or cancerous infiltration of the lung, or contraction of the chest after pleurisy, &c., except by watching the case carefully for a length of time.

which point towards the roots of the lungs. The same holds true of those found in the spleen. At first they are of a brownish-red or black color; they are firm, though fragile, and are readily distinguished from the healthy parenchyma, from which they are sharply circumscribed, by their apparently homogeneous structure. In the beginning the surrounding parenchyma is either in the normal state, or else is merely the seat of hyperæmia and œdematous infiltration; at a later period, however, when the deposits have commenced to undergo their progressive alterations, the lung is drawn into a state of reactive inflammation and hepatization, which extends to various distances, but generally in a degree proportionate to the size of the deposit.

The deposition then becomes of a lighter color, and forthwith enters into one of two metamorphoses; one of which, however, is very rare.

In the one case, and in fact most frequently, the deposit breaks down, under various degrees of discoloration, into a cream-like and purulent, or into an ichorous fluid which corrodes the adjacent tissues. This process generally commences in the centre of the deposit, and we then find one or the other of the above described fluids situated within and enclosed by the peripheric remains of the deposit, which in their turn are surrounded by lung-tissue in the state of reactive inflammation. Finally, the remains of the deposit and the adjacent inflamed portions of the lung also melt down into pus, and in fact the more extensively, the more the products of the reactive inflammation contain the germs of dissolution. This process also frequently depends upon the absorption into the circulation of gangrenous ichor, or, on the other hand, it may degenerate into gangrene, and then spachelus or gangrenous eschar of the surrounding tissues may ensue.

These deposits are frequently combined from the very beginning with a secondary croupous pleurisy, which may also be a consecutive affection, arising from an extension to the pulmonary pleura of the reactive inflammation which sets in around the purulent or gangrenous abscesses. If the reactive inflammation have also passed over into suppuration, then we will notice the abscesses shining through the pleura, in the form of roundish, nodulated, furuncle-like, and yellowish prominences; but if it have changed into spachelus, then dirty-greenish or brownish collapsed spots will be visible through the pleura, which will itself become engaged in a destructive process, either suppuration or gangrene, with or without perforation, and a diffused general pleurisy may arise.

In other, but extremely rare cases, the deposit may pass immediately from its crude state into that of obsolescence, i. e., conversion into a callous, grayish nodule, which becomes enclosed in a cellulo-fibrous capsule, and which in the course of time may be converted into an osseous concretion. Doubtless, many of these concretions have been mistaken for chalky tubercles. A more complicated retrograde-process, which occasionally takes place in the spleen or kidnies, may also at times occur in the lungs: we allude to a conversion of the deposits into a cheesy matter, and a final change of them into a chalky substance, but from a large number of observations we cannot recollect a single instance in which this has occurred.

In the first volume we have spoken of the infrequency in which these deposits became obsolete, and have also explained the reasons of this.

These deposits combine themselves with pleurisy in the way already described; and also with similar depositions in other organs, especially the spleen, kidnies, liver, brain, and thymus gland; also in the substance of the mucous membranes, particularly the intestinal; in the skin, sub-cutaneous cellular-tissue, in all interstitial cellular stratæ, and in the muscles; finally, they may be associated with exudative processes upon mucous and serous membranes, or with metastases of pus in the joints.

They should be carefully distinguished from lobular pneumonia, for which they have frequently been mistaken.*

h. GANGRENE OF THE LUNGS.

Spachelus, Mortificatio-pulmonis.

This is a disease of pretty frequent occurrence, which, as Laennec truly remarks, must not be regarded as the result of an excessively intense degree of inflammation. This does not include the impossibility of its occurrence in an inflamed lung, for

* *Diagnosis.*—Deposits of pus in the lungs are almost always secondary to phlebitis of the uterus, inferior vena cava, or vena porta, or to abscesses in various parts, especially in the liver or spleen, when followed by phlebitis of the splenic or hepatic veins. In addition to the typhoid fever, and the irregular, frequent, and severe chills, which attend the formation or absorption of pus in the veins, there will be cough, difficulty of breathing, pain in the chest, &c., when

we must admit that hepatized parts are, under certain circumstances, most frequently attacked by it.

We will first point out its pathologico-anatomical characteristics, and then proceed to the consideration of the conditions which are necessary to its production.

The consideration of the former have induced anatomists to admit and insist upon two varieties, viz., *diffused* gangrene, and *circumscribed* or *gangrenous eschar*.

In *diffuse* gangrene we find a larger portion of the lungs discolored, greenish, or brownish, and filled with a like-colored, moderately frothy, flocculent, turbid serosity, which renders the affected parts soft, rotten, and easily converted into a pulpy, shreddy mass. The whole gives forth the peculiar gangrenous odor. Toward the circumference of the gangrenous part, the discoloration, infiltration, and diminution in the firmness of the lung, gradually decreases, and imperceptibly passes over into tissue which exhibits nothing abnormal except simple colorless œdema and anemia. It corresponds to diffuse gangrene of the bronchi, and is almost always associated with it; it is rare, upon the whole, but always attains a considerable degree of extension, as it commonly inculpates the whole, or at least the greater part, of a lobe. It is most frequently found in the upper lobes, under circumstances which have led to the formation of emphysema and anemia in them, and to passive stasis in the lower lobes. It may be regarded as so much the less an essentially independent affection, as it is almost always associated with gangrenous eschar of the lungs, and hence may be readily induced by the exhalation of gas or ichorous fluid from it upon the bronchial and pulmonary mucous membranes; or it may frequently arise from a similar affection of a bronchus. The above description of gangrene of the superior lobes will answer in all respects when any other portion of the lungs is affected. It is distinguished in particular by the absence of all demarcation by means of a surrounding re-active inflammation.

the pus reaches the lungs. As the deposits are most frequent near the surface of the lungs, it is generally not difficult to detect crepitant rattle in numerous circumscribed spots, and limited dulness on percussion in two or three, or a dozen or more small places. The physical signs are in fact precisely similar to those of *lobular* pneumonia.

As has already been remarked, it should be carefully distinguished from *softening of the lungs*.

Circumscribed or *partial* gangrene of the lungs occurs in the form of gangrenous eschar, and is, beyond all comparison, far more frequent than the former variety. In any one part of the lungs, we may find a larger or smaller portion of the parenchyma converted into a blackish or brownish-green, hardish, but moist and tough eschar, which adheres to the adjacent tissues, and gives forth the peculiar gangrenous odor in a very marked degree; it is, as Laennec truly remarks, very similar in appearance to the eschar produced by the action of lunar caustic upon the skin. It is sharply circumscribed, and the surrounding tissues may be in various conditions.

The eschar gradually separates from the adjacent parts, and is then found seated in an excavation which corresponds to it in form and size; its circumference and edges are soft, shreddy, pulpy, and bathed in an ichorous fluid; its centre is a hard, firm, blackish-green plug. More frequently, however, the whole eschar breaks down into a greenish, brownish, extremely fetid, ichorous fluid, in which are intermixed many rotten, shreddy remnants of parenchyma; no trace of a plug is left, and the whole is contained within a cavity, to the walls of which a shreddy tissue infiltrated with ichor adheres.

The original size of a gangrenous eschar and its cavern varies from that of a bean to that of a hen's egg; but usually the latter is not larger than a hazel or English walnut; its shape is, upon the whole, irregular, but generally somewhat roundish; its seat is more frequently in the superficial than in the deeper parts of the lungs, and more frequently in the lower lobes than in the upper.

These eschars either occur singly, or else several are present simultaneously.

Gangrenous destruction attacks more and larger bronchi, the larger the gangrenous cavern originally was; they form the passages through which the horribly fetid effluvia and sputa are exhaled and ejected. It attacks the pleura the more quickly, the nearer the eschar was originally situated toward the surface of the lung. If the eschar should then separate from the lungs, it will, provided no adhesions prevent it, fall free into the cavity of the pleura; if it have already broken down into a thick ichorous fluid, then this will flow into the pleural sac, and pleurisy, with ichorous exudation, and pneumo-thorax will ensue; either the

fetid gas which is accumulated in the gangrenous cavern will alone form the pneumo-thorax, or, if the cavern communicate with the air-passages, then both gas and atmospheric air will be effused into the chest. Such superficial gangrenous caverns may be recognized at a glance, after opening the thorax, for the pleura above them is either converted into a blackish-green eschar, the internal surface of which is shrunken and hardish, or, if the eschar have already broken down without perforating the pleura, then this latter will appear of a blackish-green color, be moist, rotten, and puffed up by the gas in the cavern; finally, if the pleura be lacerated in one or several places, or be perforated, or perfectly destroyed by a spontaneous dissolution of its tissue, we will find the cavern either partially, wholly, or not all concealed and covered by the remnants of the pleura, and either partially or wholly emptied.

The original gangrenous abscess should be distinguished, if possible, from one which has undergone a consecutive enlargement; very large caverns are rarely of primitive formation, but have arisen from the extension of circumscribed gangrene; they, as will subsequently be shown, are not circumscribed in the same manner as primitive abscesses.

The pulmonary parenchyma surrounding a gangrenous abscess is at times in a normal condition, with the exception of being the seat of a serous or sanguineo-serous infiltration; when melting down of the eschar takes place, diffuse gangrene may ensue in this to various distances. More frequently, however, we find it in a state of reactive inflammation of various degrees of intensity and character. Very frequently simple asthenic stasis is formed and gradually changes into inflammatory congestion, in which it remains for a long time, and then slowly passes over into imperfect hepatization. In consequence of a want of energy in this reactive inflammation, the original gangrenous destruction may extend into it in various directions and distances, so as often to attain to the size of a man's fist or child's head. In such cases the adjacent tissues become discolored more or less rapidly without the successive extensions being marked by any distinct limitation, and break down into a gangrenous, ichorous pulp. In this way the gangrene may reach the pleura, and there occasion all the consequences which have been alluded to; and if the pulmonary pleura be adherent to the costal, this also may be involved in the destructive process.

Frequently we find the surrounding tissues forced into a higher

degree of inflammation, viz., that of evident hepatization, which is times so extensive as to include the whole of the lobe in which the gangrene is located. It often happens that the disease proves fatal, mostly in consequence of the severity of this reactive inflammation.

The most important process however, takes place in the tissues immediately surrounding the cavern, and must be regarded as a natural curative endeavor. The reaction here appears as an inflammation of the interstitial cellular tissue, which together with the walls of the cavern passes over into suppuration, and thus effects the separation and ejection of the spachelated parts.

At the commencement of this process, the gangrene still progresses in single parts, and we find the pus which is secreted from the walls of the forming purulent abscess still mixed with gangrenous shreds of tissue, and with ichor. In the course of time the suppuration gains the upper hand, and after the spachelated parts are ejected through the bronchi, the cavern is changed into a simple suppurating abscess. The internal parts of the walls are infiltrated with pus; beyond this for three, four, or six lines, the parenchyma is grayish-red and firm, and in case the inflammation of the interstitial tissue is coupled with croupous exudation into the air-cells, we find a scarcely perceptible, very minute granular texture. If the suppurative process in the internal layer of the capsule now abate, a cavity will be left, with whitish, cellulo-fibrous, callous walls, which sooner or later approximate each other and coalesce in the manner described when treating of tuberculous vomicæ, so that nothing but a cicatrix will remain. This is the manner in which circumscribed pulmonary gangrene heals in single rare instances.

If the softening of the eschar progress very rapidly, and none or only very slight reactive inflammation be developed in the adjacent parts, or if the primitive cavern enlarge itself very quickly, then the gangrenous destruction may involve large blood-vessels, which have not yet become obliterated, and exhausting hemorrhages ensue into the cavern, bronchi, and, when the abscess has opened into the pleural sac, even into this latter.

Partial gangrene often arises in healthy lungs, under the influence of general depressing causes, especially in weak, decrepit, and dyscratic subjects; it then developes itself out of circumscribed passive stasis. Besides, it associates itself, when aided by similar exciting causes, with pneumonia, in all its stages; also to pulmonic abscesses, tubercles, tuberculous vomicæ, and to bron-

chitis, especially that which arises in the course of various exanthems. Finally, it appears in the train of typhus fever, as a local expression of a spontaneous degeneration of it into putrescency; or it may be excited by the absorption into the blood of gangrenous ichor from distant abscesses, and then shows itself in the lungs in the form of gangrenous and dissolving deposits, or as septic capillary phlebitis.*

* *Diagnosis.*—Gangrene of the lungs occurs most frequently in debilitated and half-starved persons: according to Grisolle, in five cases only out of seventy did it supervene upon acute inflammation of the lungs. The rational signs are much more important than the physical, which only serve to point out the locality of the disease, and not its nature: in fact it can scarcely be diagnosed, if the peculiarly and intolerably fetid odor of the breath of the patient and equally fetid expectoration be absent.

The invasion of the disease is often extremely insidious; nothing but great debility may strike the attention of the physician, and no signs may be present to announce a severe affection of the chest. The face is usually remarkably pale, wan, and leaden; there are blue rings about the eyes, and the features are sunken and much altered; there is great lassitude and prostration; the pulse is small, weak, and frequent; the respiration oppressed and rapid; the cough is dry at first, but often in the course of twenty-four hours peculiarly fetid, ichorous, brownish, and greenish matters, are coughed up in considerable quantities, or almost vomited up; the breath of the patient is intolerably offensive, and the atmosphere for several paces around him is loaded with putrid and gangrenous exhalations. At first the skin is hot and dry, but the limbs soon become cold, and the face is covered with a clammy sweat; the tongue is dry, and the thirst is great. Profuse bleeding from the lungs may suddenly set in and carry off the patient; or the gangrenous abscess may burst into the cavity of the pleura, and the signs of severe pleurisy and pneumothorax will be speedily added.

Circumscribed gangrene of the lungs often runs a slower and more chronic course; there is constant hectic fever, considerable heat of skin, and rapid emaciation, so that the disease might readily be mistaken for phthisis, if it were not for the fetor of the breath and expectoration.

The physical signs are almost the same as those of abscess in the lungs, viz., large mucous rattle, followed by cavernous rattle and cough, &c., when the abscess is empty. When reactive inflammation set in around the gangrenous spot, crepitant rattle, &c., may be heard.

i. SOFTENING OF THE LUNGS.

This is a very rare affection ; it has nothing in common with Andral's red and gray softening or hepatization, hence we hold ourselves absolved from all further discussion on this point. Like softening of the stomach, it is a peculiar spontaneous process, and arises from the same exciting causes that the various softenings of the stomach do ; in further proof of the identity of the nature of the two affections, we add that it is almost always found in combination with softening of the stomach.

We may find it in any portion of the lungs, and the softened part is not at all sharply circumscribed ; according to the quantity of blood which is present in the affected part at the commencement of the softening, we find it discolored and dirty-brownish or blackish-brown ; it is soft and moist, so that it breaks down under the slightest pressure into a pulp which is thinned by the addition of a serous fluid intermixed with many black flocculi, owing to a charring of the blood. The bronchial mucous membrane is found in the same condition at various distances from the diseased part.

Owing to the quantity of blood which is contained in the lungs, softening of them always presents some similarity to that variety of black softening of the stomach which arises out of a disease of the blood itself; it may be very easily mistaken for gangrene of the lungs. In extreme cases we can only distinguish it from this, by the absence of all gangrenous fetor, and by the lesser degree of discoloration.

k. HETEROLOGOUS FORMATIONS.

1. *Cysts.*

These are among the very rarest occurrences in the lungs, which in this respect present a very marked contrast with the frequency with which they occur in some other organs.

Doubtless *simple serous cysts* may at times be formed in the lungs, but the so-called *acephalocysts* are more commonly met with. The infrequency of the occurrence of these in the lungs also contrasts strongly with their frequency in the liver ; on the other hand, tubercles are as common in the lungs as they are uncommon in the liver, and these two facts form most important objections to the hydatid theory of the origin of tubercles.

As yet single acephalocysts only have been found in the lungs, varying from the size of a pigeon's egg to that of a man's fist, and situated either in the upper or lower lobes.

They are doubtless formed in the interstitial tissue, and crowd upon the parenchyma of the lungs in proportion to their size, so that portions of it, from the compression exerted, are changed into fibro-cellular tissue. The parent sac is found surrounded by and adherent to such tissue, and contains within its walls various numbers of acephalocysts, which vary in size, and either float free in a serous fluid or else are attached to its internal surface.

It is important to know that in rare cases the mother-sac may be destroyed by inflammation and consequent suppuration, and a communication formed with the air-passages, through which the smaller acephalocysts may be ejected. It is the more necessary to be aware of this fact as hepatic acephalocysts still more frequently find their way into the lungs, and are ejected in this complicated manner.

Occasionally we find an acephalocyst seated in the lungs, communicating with one in the liver.

Cysts containing other matters, such as cholesterine or hair, &c., are much more rare than serous cysts.*

2. *Anomalous Fibrous, and Fibro-cartilaginous Tissue.*

These occur in the form of *callous condensation*, owing to chronic inflammation of the interstitial tissue; besides this, it is also found in the neighborhood of old abscesses, tuberculous vomicæ, apoplectic deposits, &c., and forms the so-called tissue of cicatrices.

* *Diagnosis.*—The most important, and, in fact, only diagnostic sign of hydatids of the lungs, is the expectoration, either of entire hydatids, or only of *debris* of them, which resemble small pieces of skin rolled upon each other. The next most important symptom is extreme dyspnœa: in all of Andral's seven cases it was present, and in some amounted to decided orthopnœa; the same difficulty of breathing was also present in Cruvelhier's four cases, in two of which it now and then became quite suffocative. None of the other symptoms were alike in any two cases; some seemed to have tubercles, others emphysema, or chronic pneumonia, or pleurisy. Not one of the eleven cases were diagnosed during life. The patient generally dies from suffocative dyspnœa.

b. Fibrous tumours are rarer beyond all comparison. They also attain only a very inconsiderable size, as they are rarely found larger than a bean or hazel-nut. They are either bluish-white, firm, but elastic, very dense and flat bodies, or, and in fact far more frequently, they are pale-yellow, or dirty-white, lax, soft, and puckered, presenting much similarity to the structure of the bronchial or salivary glands.

3. *Anomalous Osseous Substance.*

Besides ossifications of the bronchi and bronchial glands, bony deposits occur in the lungs under various circumstances, and in fact as ossification of anomalous fibrous tissue, or else as transformation of unorganized deposits into chalky concretions. To the first belong various flat, or roundish, knobby, yellow, and generally very compact bodies, which are developed in and out of all the varieties of anomalous fibrous tissues, but particularly in the callous stripes, capsules, and cicatrices, so frequently found in the lungs. To the latter belong the chalky, whitish, or grayish, nodulated, fragile, and even friable masses, into which tubercles and tubercular pus are converted under certain circumstances.

4. *Black Pulmonary Pigment.*

No organ, except perhaps the alimentary canal, is so frequently and extensively the seat of depositions of black pigment, as the lungs and bronchial glands. It is found, with rare exceptions, in the lungs of all adult persons, and increases in quantity with the advance of years. Hence it can only be regarded as a pathological appearance when it occurs at an unusually early period of life, or in excessive quantity.

It is either deposited *free* into the interstitial cellular tissue, or into the walls of the air-cells, or else it is *connected* with some other deposit, or diseased alteration, or new formation. In the *first* case, it is found, according to the degree of its accumulation, either as blackish-gray, blackish-blue, or ink-black points, or in spots as if daubed on with a brush. If accumulated in greater quantities, large branched portions of the interstitial tissue which extend in insular patches under the pleura, will be found equiformly infiltrated and blackened, and rendered dense and firm by it. This thickening of the cellular tissue is of importance, as it prevents the expansion of the air-cells on the one hand, and

causes obliteration of some of the blood-vessels on the other, so that atrophy of a portion of the lungs is finally induced. We must make particular mention of the frequent accumulation of this pigment in the apices of the superior lobes, such as often occurs without being the consequence of any particular form of disease: we there find the parenchyma uniformly blackened and dense from the deposition of masses of pigment; its structure is either uniformly firm, or else it has become converted into an irregular net-work, in consequence of atrophy of portions of it. *Atrophia senilis* of the lungs is doubtless partially induced at times by an excessive accumulation of pigment in the interstitial tissue. The deposit often takes place in the whole lung equally; still it is generally most abundant towards the surface of the lungs, and in the upper third of the superior lobes. It is often a consequence of slight irritation, and transient congestions; it may be conveyed by absorption to the bronchial glands, and thus deposited in them. It is still a matter of doubt whether the large quantities of pigment which are so often found in the lungs of those who work in coal and coal-dust, are owing to the actual reception of the latter into the parenchyma of the lungs, or whether it be a consequence of the continued irritation to which the pulmonary mucous membrane is subjected.

In the *second case*, the pigment is infiltrated in various quantities into an indurated and callous parenchyma; this happens in the neighborhood of tubercles, especially of hemorrhagic tubercles; also in cancerous deposits, in the so-called cancer melanodes, &c.

5. *Pulmonary Tubercles,*

Forms one of the most frequent and at the same time most fatal diseases of the lungs. It is also the most frequent of all the tuberculoses.

We refer our readers to what has been laid down in the first volume concerning tubercles and tuberculosis in general, for those remarks hold good here, but require a fuller elucidation on some points.

Tubercles do not occur under the great variety of forms formerly described; we distinguish *two* only, which differ in their origin and seat. We term the one the *interstitial tubercular granulation* and the other the *infiltrated tubercle,* or *tubercular infiltration.*

a. Interstitial tubercular granulations form the well known

barley-corn, mustard, or hemp-seed sized, grayish, semi-transparent, and resistent bodies, which are so frequently found in the lungs, either discrete, or in groups, or large confluent masses. Careful dissections and analogy tend to prove that their seat is in the interstitial cellular-tissue between the smaller lobules and the air-cells, and on the walls of the air-cells themselves. They are absolutely situated without the walls of the air-cells, and only form a prominence on their internal surface by pushing a part of the cell before them; if they be of large size, they compress some of the cells, so that every group or infiltration of tubercles encloses compressed and more or less obliterated air-cells. These tubercular granulations result from a *chronic or acute tuberculous process*, which deposits its products under the signs of local congestion or hyperæmia. Thus much at present: subsequently, whenever we speak of *tubercles*, or *tubercular granulations*, we refer to this variety; we will treat of tubercular infiltration separately, and whenever we allude to it we will carefully prefix its peculiar name.

b. The *latter*, i. e., *the infiltrated tubercle*, unlike the *interstitial*, is actually deposited within the walls of the air-cells. It results from a croupous pneumonia of various extent, which deposits its products, and these, under the influence of the tubercular dyscrasia, become discolored and changed into yellow tubercle, instead of being absorbed or converted into pus. Hence tubercular infiltration is a *hepatization* formed by a tuberculous exudation; the pneumonic, originally red, and granular structure of which gradually becomes paler, and grayish-red, with a tinge of yellow; it is then dry and fragile, but finally it becomes yellow, moister, of fatty-cheesy consistence, soft, and breaks down sooner or later into tubercular pus. The granular texture gradually disappears while these changes are going on, for the walls of the air-cells also become tuberculous, and the affected portions of the lungs actually seem to be converted into a homogeneous, cheesy-fatty tubercular mass. *Lobstein* has described this, but mistaken it for a conversion of the lungs into fat.

This variety of tuberculosis may, in proportion to the extent of the pneumonic process which lies at the foundation of it, either attack a *whole lobe* equiformly, or even a *whole lung;* much more often, however, it is confined to one, or several larger or smaller separated sections, and very frequently we find a *lobular tubercular infiltration;* in both instances the affected parts are commonly sharply circumscribed; finally, it may occur as a *vesic-*

ular tubercular infiltration, and then is one and the same with the so-called *Bayle's pulmonary granulations*, about which there has been so much discussion.

It frequently attacks the superficial portions of the lungs in the form of lobar, or lobular infiltration, and then may be recognized at once, from an external examination of the lungs, by the firmness and peculiar color of the affected part.

It is always the result of a high degree of tuberculous dyscrasia, and hence *rarely* appears as a primitive affection, but *usually* associates itself with an advanced degree of interstitial tubercle. It causes a consumption, which runs a rapid course, and is attended with tumultuous, acutely inflammatory, and painful affections of the lungs.

It occurs with especial frequency in young persons and children, and corresponds to bronchial tuberculosis, to one variety of tubercular affection of the intestinal mucous membrane, and to the tuberculous transformation of exudations upon serous membranes, &c.; it is very often combined with the intestinal affection, and always with a high degree of tuberculosis of the bronchial glands.

These are the two fundamental varieties of pulmonary tuberculosis; all other forms, viz., those which have been admitted on account of some peculiarities in their physical properties, although of much importance individually, still do not differ from the above in their locality, to which we have thus far confined our remarks; they either depend upon various modifications in the general disease, or merely upon alterations which the tubercles themselves undergo.

Tubercles are found in no other organs, except the spleen and serous membranes, in such great numbers as in the lungs. They appear either as *discrete granulations*, or else several of them are united together in *one group*. In the first case, each granulation stands singly, so that they are separated from one another, in proportion to their numbers, by a larger or smaller quantity of more or less healthy parenchyma. This takes place either in a certain uniform, or else irregular manner; in the latter case we find a greater number of tubercles aggregated in smaller portions of parenchyma, while in other places we find a smaller number of these deposited in a greater quantity of the substance of the lungs. If many be present, they are crowded, and even run together in irregular masses, as may be noticed in the superior lobes, which are generally more diseased than the other parts of

the lungs. In many of the more common cases we find a uniform increase in the number of the tubercles, and also an equally proportioned approximation of them towards one another, the more we advance in our examination from the lower lobes towards the upper.

This accumulation of the tubercles into irregular masses, such as is noticed in their usual starting point, viz., the apices of the superior lobes, or occasionally in other parts, should be carefully distinguished from the *primitive deposition of them in nearly regular groups.* Under certain not yet well understood local and general conditions, tubercles are deposited originally in roundish groups of the size of a pea, bean, or hazel-nut, while around them we find discrete tubercles scattered in greater or smaller numbers, or at times none at all. In extreme cases of this kind, they are seated around a central nucleus of pulmonary substance, from which processes of parenchyma are found running in various directions so as to divide the groups of tubercles into several laminæ.

Pulmonary tubercles either appear originally as the well known gray, imperfectly transparent, mustard or hemp-seed sized granulations; or, in many cases of acute tuberculosis, as still smaller, scarcely barley-grain sized, clear, transparent, and hence vesicular-like bodies; or, finally, in very high degrees of tubercular disease, they are excreted with rapidity from the blood as yellow, cheesy tubercles. At first sight they seem to be of a round or almost roundish form, but by a closer examination we discover that the outlines of their shape are not sharply marked, and that delicate prolongations extend from their circumference into the parenchyma of the lungs. According to their size they may enclose one or several air-cells, which generally become obliterated, although they are frequently found dilated.

By the confluence of several tubercles which were originally deposited in one group, or by the subsequent addition of fresh tubercles in the intermediate spaces, it often happens that large, either regularly roundish, or misshapen branched and laminated tubercular masses are formed; the parenchyma, which is enclosed by them, gradually wastes away, so that nothing but the pigment which may have been deposited in it remains; the air-cells, together with the terminal ends of the bronchi, become obliterated. Such tubercular masses should be distinguished, if possible, from primitive tubercular infiltration. It often happens that the whole of the superior lobes are so thickly strewed with tubercles, as to appear converted into a firm, dense, and homogeneous tubercular mass.

In the common course of the disease, the principal seat of the tubercles is in the upper third, or apices of the superior lobes, i. e., they are first deposited there and in the greatest quantity, and hence the first traces of softening are also noticed in the same locality; therefore the apices of the lungs must be regarded as the starting point of tubercular disease, which then gradually extends downward towards the lower lobes. Still there are some exceptions to this rule, for occasionally we find some tubercles in the tops of the lungs, and then others far distant from them in the very lowest parts; or rarely we may find some in the latter situation, and none in the former. The general truth of the above rule has led to the supposition of an antagonism between tuberculosis and pneumonia, which certainly holds good in the majority of instances, for pneumonia is certainly most frequent in the lower lobes, and tubercles in the upper, and in fact in their very topmost parts.

The proximate cause of this preference which tubercles exhibit for the upper lobes is still involved in obscurity, although many more or less satisfactory attempts at explanation have been advanced; in fact, they are either based upon mere conjecture, or else the effect has been mistaken for the cause. We admit our ignorance upon this point, and regard the question as analogous to those mysterious preferences which certain eruptions and exanthems exhibit for particular sections and parts of the general integuments.

Pulmonary tubercles pass through various stages or metamorphoses:

1st. And very frequently they *soften*, and thus cause *tuberculous suppuration* of the lungs, or *tuberculous ulcers*, or *tuberculous abscesses*, (vomica pulmonis tuberculosa, caverna tuberculosa,) and finally *tubercular phthisis*. The softening of the discrete, gray tubercular granulations commences in their centres, which become turbid, opaque, yellowish, and cheesy, and finally break down into pus. A group of tubercles breaks down at several points simultaneously, corresponding in number to that of the tubercles which form it; hence we have, first, several small *primitive tubercular ulcers*, and after the melting down of the whole mass, they coalesce to form one larger one. The following remarks apply to the advances which this latter makes.

It is of great importance to understand the manner in which primitive tubercular ulcers *enlarge*, and how they effect their peculiar and extensive ravages in the lungs. This is explained by

the process which goes on in the tissues surrounding the softened tubercle. The breaking down of tubercles is always followed by a secondary deposition of them in the surrounding parenchyma, which is abundant in proportion to the degree of intensity of the constitutional disease. The secondary tubercles and the tissue in which they are seated also break down with a rapidity proportionate to the intensity of the general affection, and thus the tubercular ulcer becomes enlarged. If this process proceed so rapidly and tumultuously as quickly to exhaust both the reactive powers of the constitution and the supply of plastic materials, then the ulcer will usually be found to have extended itself unequally in various directions, so as to form an irregular, multisaccular, and apparently lacerated cavern, the walls of which are composed of parenchyma loaded with softened tubercles. Its internal surface presents a corroded or gnawed appearance, without exhibiting any trace of a lining membrane, except a coating of tubercular pus; the small quantity of parenchyma which is found between the tubercles is in a state of compression and dirty-brownish discoloration, i. e., in a state of *carnification*; that which is farther removed offers no trace of inflammatory reaction, except a certain degree of hyperæmia. If this process take place, as it usually does, in several points simultaneously, then two or more caverns will be formed in close proximity, and will finally open into each other; we then find a number of caverns communicating together, by means of fistulous passages of various widths, which run either straight or tortuous courses, so that the whole represents a large, irregular, and multi-saccular cavern. Bridges or ridges of rotten pulmonary parenchyma traverse the abscess in various directions, and shreds of the same hang down into it.

This variety of pulmonary phthisis is analogous to that acute form of tubercular intestinal ulceration which runs its course without any inflammatory reaction.

In other more common and less rapid cases, an inflammatory affection arises in the parenchyma adjacent to the softened tubercles or the primitive tubercular ulcer, and near to the secondary tubercular deposits; it is seated in the interstitial tissue, and must certainly be regarded as a curative process. It throws out an albuminous, grayish-white, or reddish and viscid exudation, which Laennec mistook for a peculiar variety of tubercular infiltration, his so-called *infiltration tuberculeuse gelatiniforme*. (See Interstitial Pneumonia.) It induces a closure, and finally atrophy

of the air-cells. During this process, the internal surface of the cavern already becomes more regular and even, and we frequently find it covered with a thin, grayish, or grayish-yellow, soft and somewhat thin lining membrane. This coating, as Andral remarks, may at times be composed of the more solid portions only of the pus contained in the cavern; but in the majority of instances this certainly is not the case, but, as Laennec already supposed, it is a true exudation from the walls of the cavern, similar to that which is thrown out from cut or ulcerated surfaces. This lining membrane is undoubtedly thrown off very many times in succession, for if the tuberculous softening still go on, neither the membrane itself, and much less the adjacent walls of the cavern, can become organized; hence it melts or breaks down into pus, which mixes with that already in the cavern, and its place is quickly supplied by another; this process is repeated as long as the tuberculous affection on the one hand, and the reactive inflammation in the parenchyma persist in certain antagonistic proportions. The cavern still enlarges itself in the manner already alluded to, viz., by the softening down of the secondary tubercular depositions in its walls, and by the confluence of several neighboring caverns into one. The bridge-like portions of parenchyma which traverse the caverns are also coated with exudation, their substance is in a state of gelatinous infiltration, and tubercles may be found in them.

In further consequence of this process, the parenchyma which forms the walls of the cavern becomes atrophied, and converted into a bluish-grey or blackish-blue, dense, and tough substance of varying thickness, and containing more or less black pigment; the layer which looks towards the cavern is mostly composed of whitish cellular tissue, and forms its inner surface; to this the above described exudation adheres, and through both we distinguish faintly the bluish and atrophied parenchyma beyond, also the naked and obliterated vessels, which look like yellowish-white, arborescent streaks; and scattered crude, or softening yellow tubercles. These latter gradually soften, and thus lead, on the one hand, to a slow enlargement of the cavern, and prevent, on the other, a perfect consolidation of the cellular lining membrane, as they perforate and partially destroy it. The internal surface of such a cavern is even, and pretty smooth, with the exception of the newly-formed small tubercular excavations.

The vomicæ, when in this condition, resemble the tuberculous ulcers of the intestines which have undergone a gelatino-fatty condensation of their bases, i. e., of their sub-mucous cellular tissue.

The caverns themselves naturally present the most manifold differences in their size and number. The instances are not rare in which abscesses are found capable of containing a duck's egg, or a man's fist, or even as large as a whole lobe of a lung. The larger they are, the greater the probability that they were formed by the confluence of several smaller abscesses. The largest caverns are, with rare exceptions, usually found in the upper lobes, as might be expected from the preference which tubercles show for this locality.

It is a point of much interest to determine in what manner the other component tissues of the lungs take part in the above described ulceration; above all, how the *bronchi* and *blood-vessels* are affected, and what takes place when the cavern reaches the *pulmonary* pleura.

The *capillary bronchi* undergo the same softening as the parenchyma, for their walls also are filled with tubercles, and their mucous membrane, as is also the case with the larger bronchi when seated near large tubercular abscesses, becomes infiltrated with tuberculous matter at the same time that the previously deposited tubercles commence to soften. The capillary tubes are thus converted into tuberculous matter, and they, together with the cellular tissue which surrounds them, are exposed to the same process of softening. If the abscess should now enlarge, the destruction of the bronchial tubes advances in the same proportion as that of the surrounding parenchyma, and when this latter has attained to a certain degree, the tubes become perforated, and a communication is established between the bronchi and the abscess. We only find bronchial tubes of a certain larger size opening into abscesses, for the smaller ones are compressed by the tubercles deposited in their walls and neighhorhood, and by the products thrown out by the interstitial inflammation; further, they become completely obliterated by the catarrhal swelling and tuberculous infiltration of their mucous membrane. In such places, however, where they cannot become thus compressed and obliterated, on account of the greater thickness and firmness of their walls, and their larger calibre, they open freely into the abscesses. In proportion to the size of the cavern, one or several bronchi are found opening into it. They always open with a roundish, or else oval or fissure-like mouth, according as they have been ulcerated transversely, obliquely, or only on one side. When this has happened recently, the bronchial opening presents an ulcerated appearance; at a later period, however, when the

abscess has received a dense and callous lining membrane, the opening is fringed by a shreddy muco-membranous edge, which is in a state of gelatinous infiltration. This edge is similar to the serrated, shreddy, gelatinously-infiltrated muco-membranous edge that surrounds the callous intestinal tubercular ulcerations. The bronchial opening never projects above the internal surface of the cavern, but always lies on the same level with it.

The *blood-vessels* are generally in an opposite condition; they are usually obliterated, the bronchial vessels especially, and are thrust to one side, so that they traverse the walls of the cavern as ligamentous, prominent, yellowish-white, and branched stripes: the arteries withstand ulceration for a long time. We also generally find partially or wholly obliterated blood-vessels in the centre of the bridges of parenchyma which run through the cavity of the abscess. They are usually covered with atrophied parenchyma; still it happens frequently enough that they are laid bare on one or the other side before they have had time to obliterate; their cellular sheath is soon eaten through, and then both the internal coats give way, and form the source of the well known and often fatal *pulmonary hemorrhages* which occur in the course of consumption.

The abscesses may reach the pleura in two different ways: they are either formed originally in the neighborhood of the pleura, or else deep seated abscesses reach it after having attained a considerable size. The first very rarely takes place from the softening of miliary tubercles, but far more frequently from the melting down of tubercular infiltration. In both instances the pleura may become diseased, and in fact, in different ways, and with different consequences. If no pleuritic adhesions exist at the point where an abscess reaches the pleura, this will be laid bare and converted into a yellowish-white eschar of varying size, which eschar either becomes lacerated, or else detached around its whole circumference, and falls out entire; through the opening thus made, tubercular pus and atmospheric air find their way into the cavity of the pleura; the consequences are, pleurisy with pneumo-thorax, and generally a speedy death. Mere cellular adhesions cannot prevent this termination; they are partially torn asunder by the weight and force of the effusion from the abscess, and they also take part in the pleuritic inflammation and break down into exudation. But if thick adhesions be present, i. e., if the lungs be adherent by means of dense, thick, callous, cellulo-fibrous, or fibro-cartilaginous pseudo-mem-

branes, such as are formed after pleurisy, especially about the apices of the lungs, then the pulmonary pleura which has coalesced with these, may be laid bare even to a great extent, yet a perforation and its consequences will be permanently prevented. But although it usually happens that the tuberculous destruction is arrested in this direction when it reaches such callous swathes, still cases have occurred in which these also have been perforated. They are first thrown into a state of irritation, under which they soften, become tuberculous, and finally suppurate by layers; thus they are perforated, and the tuberculous process attacks the walls of the thorax, progresses slowly in the ribs and soft parts, and ultimately reaches the skin, in the form of a fistulous passage, which may open at various parts of the chest, or even of the neck. [Cruvelhier.] This fistula is tuberculous, and is often complicated with caries of the ribs, sternum, or even vertebræ.

Tuberculous infiltration, when present in connection with interstitial tubercles, generally softens down with great rapidity; it is this which hastens the course of consumption, and forms the basis of the so-called "galloping" or florid phthisis. It causes the most disgusting form of destruction of the lungs, viz., large caverns of irregular shape, surrounded by a half-rotten, tuberculously-infiltrated, purulent, dissolving, and corroded parenchyma. Tuberculous infiltration is generally deposited in the superficial portions of the chest, and hence the abscesses which arise from its softening are those which most frequently open into the cavity of the pleura. This may take place in various ways:

a. The pulmonary pleura may be puffed up by the air which flows into the abscess, and be forcibly peeled off from the lungs to various distances beyond the extent of the cavern, so as to form a flattish, roundish air-bladder, which finally bursts.

b. The pleura may, as was before remarked, be converted into a yellowish-white eschar, which either gives way, or falls out entire into the pleural sac.

c. The pleura, together with the infiltrated parenchyma which surrounds the abscess, may be attacked by gangrene, and then both become changed into a dirty-brownish, greenish, pulpy, shreddy, offensive mass.

This latter termination is the more worthy of notice, as it may take place not only near the surface, but also in the deeper seated parts of the tubercular infiltration, especially about the circumference of an abscess already formed in it. Besides, it

may happen, in consequence of the frequent combination of tubercular infiltration with a high degree of tuberculosis of the bronchial glands, that such an intercurrent gangrene may form the medium of communication between a pulmonary-cavern and a glandular abscess.

The *contents* of tuberculous caverns present various differences: at times, and in fact at the commencement of the softening of miliary tubercles, and of tubercular infiltration, they contain a thick, yellowish pus; more frequently, however, we find a fluid (tubercular ichor) composed of a thin, whey-like portion, intermixed with numerous grayish and yellowish, fragile, cheesy-purulent flocculi, and similar crumb-like particles, the quantity of which does not, however, alone suffice to account for the profuse expectoration which attends consumption. Frequently this fluid is of a grayish-red, reddish-brown, or chocolate color, from admixture with blood; or of an ash, or blackish-grey color, from admixture with black pulmonary pigment. Besides, we occasionally find larger or smaller fragments of the lungs, which are similar in composition to the parenchyma which forms the walls of the caverns; also chalky concretions in the abscesses. In other cases, they contain fluid or coagulated blood in various states of discoloration.

The above-described two-fold alterations which pulmonary tubercles undergo, occasion, as has already been hinted, the so-called *tuberculous pulmonary consumption.* If we now turn our attention, in a pure case of this disease, to the condition of the parenchyma around the tubercles and their abscesses, and from thence to that of the other organs and systems, we will obtain the following facts in addition to those already set forth:

In the upper third of the superior lobes we generally find a large cavern, surrounded inferiorly by several of a smaller size, some of which communicate with the former; between these we find yellow tubercles, which are on the point of softening; a little lower down, gray tubercles, just becoming opaque and discolored; in the lower portions of the upper, and in the inferior lobes, a proportionately far smaller number of gray, crude tubercular granulations.

The pulmonary parenchyma between the tubercles is found in various conditions, according to the stage of the consumption. It is either normal, but generally in a state of slight vicarious emphysema of its superficial parts, with hyperæmia and œdema of its deeper seated. But it is more important to notice the

atrophy of the parenchyma, induced in part by the interstitial inflammation, partly by the obliteration of the bronchi and air-cells from the pressure exerted upon them by the agglomerated tubercles, and in part by the obstruction of the bronchi by the blenorrhoic mucous secretion. On the other hand, croupous pneumonia may have attacked the greater part of the parenchyma which is free from tubercles; it appears partly as brownish-red hepatization, partly as grayish-red, which are quickly converted into yellow tubercular infiltration, and break down into abscesses in various places. At other times, the pneumonia is followed by an exudation of the gelatino-glutinous product already described. [See page 133.] In the best marked cases of this kind the lungs are very voluminous, and are coated with a grayish-yellow, generally thin, pleuritic exudation, through which and the pulmonary pleura we can see superficial tubercular infiltrations, surrounded by emphysematous patches.

In the *larynx* we find tuberculous ulcers in various numbers and of different sizes; also aphthous erosions upon the tracheal, and at times upon the pharyngeal mucous membrane.

The mucous membrane of the bronchi which go to the abscesses is in a state of tuberculous infiltration to some distance, and the tubes are filled with tuberculous matter; besides this, they are always in a state of catarrh, with reddening and softening of their mucous membrane, which secretes a muco-purulent matter, forming by far the greater proportion of the sputa which are expectorated in the course of consumption. The *bronchial glands* are enlarged and tuberculous in various degrees.

Externally upon the lungs we find *pleurisies* of various extents, and, with the most different exudations, their organizations and consequences. They occasion, at times, those very acute pains in the chest, which so frequently annoy phthisical patients. Except when they arise from superficial pneumonia, they are generally developed during the softening down of tubercles and the formation of the abscesses, and are complicated with inflammatory reaction in the adjacent interstitial cellular tissue. Among these, the pleurisies around the apices of the lungs and the body of the upper lobes are remarkable for the constancy of their occurrence; they correspond to the favorite seat of tubercles, and deposit an exudation, which either becomes organized into fibro-cellular bands, or into thick, compact, fibrous swathes, which cover the upper lobes, from their apices downwards, in the form of a cap; they are thickest superiorly, and gradually grow thinner from

thence downward ; they bring about an intimate adhesion of the lungs to the costal walls, and thus afford an effectual protection against perforation of the pleura by large abscesses.

With rare exceptions we find pulmonary phthisis complicated with *tuberculous intestinal phthisis*, which, although almost always a secondary affection and dependent upon the pulmonary tuberculosis, may run its course far more rapidly than this, and quickly occasion great and exhausting ravages. As a rule the lower third of the ileum is attacked ; from thence the ulcers progress upward along this intestine, and downward over the colon ; in the first direction they may even reach the stomach. Frequently, however, and especially at certain times, the colon is not only most severely affected, but even forms the almost exclusive seat of the intestinal tuberculosis ; at other times, we at least find that the ulcers, which are simultaneously present in the ileum, are of more recent date than those in the colon. The *mesenteric glands*, which correspond to the ulcerated parts of the intestine, are tuberculous in various degrees.

The mucous membrane of the alimentary canal, especially that of the stomach and colon, is in a state of more or less developed blenorrhœa. *Acute softening* of the mucous membrane of the greater *cul de sac* of the stomach not unfrequently sets in towards the end of phthisis.

The *liver* is found with unusual frequency in that state which has received the name of Nutmeg liver, consisting in an abnormal separation of the yellow and red substances, with predominance of the yellow and fatty infiltration of the same, forming the true fatty liver. It is not, as is usually supposed, peculiar to phthisis, i. e., to the softening of tubercles and tuberculous ulceration of the lungs, but belongs to the tubercular dyscrasia in general.

The *spleen* exhibits no constant alteration which stands in essential connection with tuberculous ulceration of the lungs.

The right side of the *heart* is sometimes dilated, in consequence of the obstruction to the passage of blood from it, owing to impermeability of portions of the lungs, from the presence of tubercles and the consecutive disease they occasion ; but far more frequently we find it remarkably small, pale, and void of fat, in consequence of the general anæmia which keeps pace with the progress of consumption. In the first instance, we find stagnation and accumulation of blood in the right side of the heart, and thence in the whole venous system ; in the second, we find general bloodlessness coupled with narrowness of the aorta.

The *central organs of the nervous system* exhibit no essential alterations; still we find frequently enough, especially in the train of acute consumption, hyperæmia of the brain and its membranes, recent serous effusions into the ventricles, coupled with white, i. e., hydrocephalic softening of the substance of the brain.

The *muscles* are all emaciated in an extreme degree; the fat of the body is in most instances almost perfectly consumed; the cellular tissue, especially that of the limbs, is often infiltrated with serum.

Tubercular pulmonary consumption is doubtless *curable*, as may be inferred from not unfrequent appearances in the dead bodies of those who formerly had more or less suspicious affections of the chest, from which they recovered. We can only expect to arrive at a truly rational and certain method of treatment, by an investigation of the circumstances under which such spontaneous cures have taken place; and the consequences of such treatment will be the more beneficial, as it will be directed not merely against the abscesses in the lungs, but against the tuberculous dyscrasia also. Pulmonary consumption and tuberculous abscesses may only be cured after the general disease, and hence that process which lies at the foundation of its local effects, viz., the tubercles and abscesses, has been eradicated. Under such conditions it has been proven, by numerous incontrovertible facts, that tuberculous abscesses may heal, and in fact in several different ways.

a. The reactive inflammation in the interstitial tissue around the caverns, deposits, as has already been remarked, a gelatinous infiltration, which obliterates the air-cells. This henceforth occasions a conversion of the collective adjacent parenchyma into a dense, fibro-cellular tissue of varying thickness. While this is taking place, the exudation which is deposited on the walls of the cavern by the same inflammatory process, changes into fibro-cellular tissue, and from thence becomes organized into a smooth serous membrane, which unites intimately with the former. The whole tuberculous abscess has now become transformed into a *cellulo-serous cavity*, from the internal surface of which a serous, sticky, synovia-like fluid is secreted. The bronchi which open into these cavities present a peculiar appearance, for the serous membrane which lines the sac, and the fibro-cellular tissue below it, become attached to the external sheath of the tubes, while the mucous membrane of the latter projects with a shreddy and somewhat inverted free edge into the cavity of the sac.

But more commonly we find the abscesses lined with a *villous, cellulo-vascular*, more or less dark red, muco-membranous-like layer, which is intimately adherent to the subjacent tissues. It appears to be in a constant state of irritation, and as it is usually found in caverns which communicate with large bronchial tubes, it would seem that its constant exposure to the influence of the air prevents its being converted into a smooth serous membrane. Doubtless an already formed serous lining may be again broken down by the irritation thus induced, into such a cellulo-vascular, muco-membranous-like tissue. It secretes a turbid, muco-serous fluid, and not unfrequently we find it covered with fresh exudations in consequence of higher degrees of irritation. In all probability these irritations, coupled with other causes, effect a gradual diminution and final closure of the caverns, in a manner subsequently to be described. In such caverns the appearance of the bronchial opening is different from that already described, for the mucous membrane of the bronchus coalesces with the analogous tissue which lines the cavern, and both run into each other without visible lines of demarcation.

At times plates of bone are developed beneath the serous lining of the caverns, in like manner as they are formed in the cellular tissue beneath healthy serous membranes.

In such caverns an accident not unfrequently takes place, the like of which had not previously occurred, and which very often proves fatal at the outset. We allude to the *hemorrhages* which take place from abscesses thus constructed. They always spring from large permeable branches of the pulmonary arteries which traverse their walls, and are laid bare to some extent. These blood-vessels may open in two different ways:

a. They either undergo an aneurismal dilatation, in consequence of an absence of support on the side turned towards the abscess, and finally give way at this place, without their tissues having undergone any other alteration of texture.

b. Or the delicate cellular sheath of the vessel participates in the irritation which is going on in the adjacent lining membrane of the abscess; this extends to the fibrous coat, which becomes loosened, softened, and infiltrated with gelatine; then the vessel gives way, either with or without having suffered a previous dilatation of its coats.

An occurrence deserving of attention sometimes attends these hemorrhages. The extravasation of blood into the cavern at times coagulates into a fibrous mass, which fills it completely,

and is connected with a neck-like prolongation which penetrates into the lacerated opening of the vessel, and is continuous upward and downward, with a cylindrical coagulum in the latter. The abscess may certainly close around this mass of fibrine, when the latter has become contracted and chalky ; but as the abscess, in its previously described state, must be regarded as a simple non-malignant cavity in the lungs, which may heal in a safer and simpler way, this method of cure must be looked upon as an uncertain and dangerous one, except in cases where the abscess does not communicate with a bronchial tube. Hence, it is only so far of actual benefit as it affords a support to the vessels seated in the walls of the cavern, and thus prevents subsequent hemorrhages, which might occur before the abscess is healed by safer and more common processes.

The already described cavern must be regarded as a healed tuberculous abscess, but the cure may progress farther, viz., to perfect cicatrization, i. e.:

b. If the abscess be not too large, it may close by a gradual approximation of its walls, which finally touch and unite with each other. We then find a cellulo-fibrous stripe in the place of the former cavern ; the bronchi terminate in it in a blind cul de sac. This process takes place most frequently in the apices of the lungs, in which the presence of open abscesses, together with obsolete and chalky tubercles, sufficiently mark the nature of the process which has been going on. The obliteration of the cavern always occasions a degree of sinking in of the chest, proportionate to its size, and also a cicatrix-like puckering and contraction of the pleura ; this is observed more plainly and frequently after the closure of the often quite superficial abscesses situated in the tops of the lungs. The thorax also sinks in, in a degree proportionate to the size and number of the closing abscesses, as may be inferred from the frequency of flattening or pit-like indentations in the sub-clavicular regions.

The approximation and agglutination of the walls of the abscess may be essentially aided by various occurrences, among which belong the sinking in of the walls of the chest, the compression of the lungs by the diaphragm, from enlargement of the abdomen or some of its organs, the emphysematous dilatation of the parenchyma adjacent to the abscess, bronchial dilatation, &c. It has been proposed and attempted to produce one or the other of these conditions in artificial and sometimes forcible ways ; we have given our opinion of the propriety of such attempts at

cure in the first volume, and have pointed out the effects which they may produce.

If the curative process sets in and proceeds very rapidly, the cicatrix may enclose chalky concretions of various sizes, formed by the inspissation of a remnant of tubercular pus.

c. The abscess may not cicatrize in the above described manner, but is filled up by a roundish or irregularly branched mass of fibro-cartilaginous tissue, in which the bronchi terminate, formed by the conversion of its cellulo-fibrous walls into a fibro-cartilaginous callus, which progressively becomes thicker and thicker. In such cases a slight contraction only of the parenchyma ensues.

This fibro-cartilaginous mass may be transformed, sooner or later, into a very compact bony concretion of corresponding size and shape.

2. The second metamorphosis which pulmonary tubercles undergo, under favorable circumstances, is the very opposite of softening, viz., the *chalky transformation.* After the softening of them has commenced, or even been perfected, they gradually become thickened into a yellowish-white, or grayish, or blackish-gray, smeary, chalky paste, which finally changes into a chalky concretion of much smaller size than the original tubercular mass. This concretion is seated, according to the intensity and extent of the reactive process in the neighborhood of the softening tubercles, either in obliterated pulmonary tissue, or in a fibro-cellular, or callous fibro-cartilaginous capsule. In this case, also, cicatrix-like contractions of the parenchyma take place in the direction of the chalky tubercles.

Tubercular infiltration doubtless may also undergo the same chalky transformation, for we not unfrequently find pasty masses of chalk, especially in the apices of the lungs, and aside of chalky tubercular granulations. In size and form these masses correspond to the lobules of the lungs ; they are surrounded by a quite delicate sero-cellular capsule, composed of condensed interlobular cellular tissue, and in all probability are chalky *lobular* tubercular infiltrations.

3. Finally, the crude, gray, tubercular granulations may become *obsolete*, shrivel together, and wither away. They are then found changed into opake, bluish-gray, cartilaginous, resistent tubercles, or nodules, which are subject to no further metamorphosis. This abortion or dying away of tubercles, either affects them wholly, or else only partially, and is then combined with chalky

transformation, so that we find their central portions converted into chalky concretions, which are incapsuled in a layer of obsolete tubercular matter.

In accordance with what has been above laid down, it is evident that pulmonary tuberculosis may be cured by the intervention of phthisis and the ejection of the tubercles; but the two last described metamorphoses represent more direct curative processes. Either may take place under like favorable circumstances, and as a rule we find them all in one and the same individual, for we may see cellulo-fibrous cavities, their cicatrices, and chalky and obsolete tubercles in the neighborhood of each other. All of these are commonly impacted in obsolete parenchyma, which is saturated with black pigment.

Tuberculosis is either an *acute* or *chronic* affection. In the first case it attacks both lungs simultaneously, under peculiar symptoms, resembling those of typhus fever; frequently other parenchymatous and membranous organs become tuberculous simultaneously. Acute tubercles are the product of a very high degree of tuberculous dyscrasia of the blood; they are either deposited at one time, or else at several, which follow close upon one another, and are marked by a paroxysmal exacerbation of all the symptoms; they are very minute, scarcely millet-seed sized, vesicle-like, and transparent, or else barley-seed sized, miliary, gray, crude granulations; they are always very numerous, discrete, and equiformly scattered through the substance of the lungs; very rarely are they found heaped together and confluent, and then only in single places; all are in the same stage, viz., that of crudity. The lung itself is in a state of hyperæmia, œdema, and partial emphysema; the former may pass here and there into pneumonia and hepatization.

In the majority of cases, acute tuberculosis only attacks the lungs after tubercular disease has already shown itself in its favorite seat, viz., the apices of the lungs, and existed for a longer or shorter period of time in the form of a more or less circumscribed, slow tuberculosis, which has already entered upon its stages of softening and ulceration. A pre-existing chronic and circumscribed pulmonary tuberculosis hence generally forms the point of departure and attraction for acute deposition of tubercles. It proves fatal in consequence of the attendant hyperæmia and consequent œdematous infiltration, also from the forcible development of emphysema, and final paralysis of the lungs.

Chronic tuberculosis either deposits its products silently and

imperceptibly, or else as crises of a mild general disease, attended with moderate vascular excitement, and re-occurring at different intervals of time. In accordance with this, we always find tubercles of various dates and stages, viz., in the upper lobes, which is their favorite and primary seat, we have abscesses; in the lower lobes, for which they have the least predilection, and hence attack last, we have recent crude tubercular granulations; between these extreme points, we find softened tubercles nearest the abscesses, and lower down, we discover such as are just on the point of breaking down.

It either proves fatal from phthisis with exhaustion and tabes, or else from some of the already described accidents which may occur during the course of consumption, viz., from the superaddition of pneumonia, followed by hepatization, which tends towards tubercular infiltration; or of hyperæmia, or œdema of the lungs; or of hyperæmia of the brain, and serous effusions into the ventricles, causing hydrocephalus and serous apoplexy; or of tubercular meningitis, exudative inflammation of neighboring muco-membranous canals, as of the trachea, or œsophagus; or from absorption of pus, or the superaddition of acute pulmonary or general tuberculosis.

The external peculiarities of persons predisposed to pulmonary tuberculosis is similar to those which characterize the tuberculous dyscrasia in general, and agree in many respects with those of the so-called irritable scrofulous constitution; the torpid scrofulous is more frequently associated with tuberculosis of the bronchial glands. The well-known and so-called phthisical conformation of the chest is not always present; and what peculiar relation it bears to tubercular disease is still unknown; the assumption that it depends on smallness of the lungs is unwarranted and hypothetical. Tubercles, as is well known, are often developed in persons who have never been exposed to injurious and predisposing exciting causes, and then form the so-called *constitutional pulmonary tuberculosis and phthisis.* On the other hand, they may arise without any such hereditary constitutional taint, in consequence of demonstrable exciting causes which either lead to a pure tuberculous dyscrasia of the fluids, or a more or less modified one; they then form the *acquired*, either *pure*, or more or less *complicated or modified* tuberculosis, such as occurs in the train of exanthems, impetigo, gonorrhœa, syphilis, irregular gout, or from the abuse of ardent spirits, after the suppression of natural or habitual evacuations, as of the menses, after the cure of inveterate ulcers, &c.

These inveterate forms of dyscrasia deposit different varieties of tubercles, which, however, have not as yet been accurately described, except the hæmorrhagic tubercle. The varieties are distinguished from the more common, by their selecting unusual seats, or attacking different portions of the lungs unequally, by their being agglomerated into circumscribed or grape-like bunches, by their unusual size, their dirty greyish or lead color, and greenish tinge. (See Vol. I.)

They should carefully be distinguished from that form of cancer of the lungs in which the cancer is deposited in the form of tubercles.

Pulmonary tuberculosis, like general tuberculosis, is incompatible with all the diseases enumerated in the first volume; more particularly, however, with those affections of the lungs which are attended by atrophy of the parenchyma, by emphysema, bronchial dilatation, excessive density of the tissue of the lungs, compression of the same, or by obsolescence and obliteration of it, &c.*

* *Diagnosis.*—Phthisis, unlike pneumonia, is most frequent in females: of 204,993 cases collected from all sources, 109,588 were in females, to 95,405 in males, or an excess of about 7 per cent.; Louis makes it 12 per cent., and Farr calculates it at 14 per cent. Again, no pathological fact is so well established as that tubercles are most frequently deposited in the anterior portions of the apices of the superior lobes; hence it is there that we look for the first physical signs of phthisis. Stark first made the observation that the left lung is more frequently tuberculous than the right; Carmichael Smith corroborates this from an examination of the cases collected by Bonetus and Morgagni; of 337 cases collected by Morton and Hughes, the left lung was most diseased 167 times, while the right was most affected in 117 times only; Louis makes the proportion as three to two; and in 48 cases of pneumo-thorax, from the opening of tubercular abscesses into the cavity of the pleura, 34 were on the left side, to 14 on the right. Laennec, Lombard, Mackintosh, Home, Hasse, and others, however, insist that the right is most frequently tuberculous.

Cough is generally the earliest symptom: at first it is so slight as not to attract much attention, as there are only one or two imperfect efforts to cough, first noticed in the morning on getting out of bed; then occasionally during the day, after unusual exertion, and at night on getting into bed; it is dry at first, but gradually a semi-transparent, ropy, saliva-like fluid is raised by the morning cough, and in it we often notice a crowd of small, dull-white, or slightly yellowish, crumb-like particles, which Bayle compared to grains of boiled rice, and Hippocrates to grains of hail. Gradually the semi-transparent, saliva-like fluid and the crumb-like particles are found

o. CANCER OF THE LUNGS,

Occurs in the form of *carcinoma medullare*, and of carc. fasciculatum, or hyalinum. The latter is extremely rare; the former is more frequent, and to it most of the following remarks apply.

intermixed with grayish and semi-transparent, or yellowish, or greenish, and completely opaque mucus, so that the whole of the sputa presents a variegated appearance.

With the cough there is slight breathlessness, or quickness of breathing on going up stairs or exerting one's self; also some tightness of the chest, and fleeting pains shooting from one or both clavicles to the scapulæ, or apparently occupying the shoulder-joint, so as often to be mistaken for rheumatism, or, if on the right side, for an indication of liver complaint, or of heart-disease, if located on the left. These pains when present are extremely significant.

The pulse becomes quicker, especially after meals and towards evening, when a slight chilliness is often experienced, followed by heat in the palms of the hands and soles of the feet, which continues during the night. In five cases out of six, the frequency of the pulse in incipient phthisis exceeds the highest frequency observed in health, and it is remarkable that change of posture from standing to sitting or lying produces very little alteration of it; the mean difference in the frequency of the pulse in health from such change of posture is six times greater than the mean difference, and three times as great as the maximum in phthisis, so that the slight effect on the pulse, produced by change of posture, often forms one of the most certain signs of incipient phthisis.

After some time, slight morning perspirations begin to follow the evening fever; the face of the patient is paler and expressive of languor in the morning; in the evening the cheeks are slightly flushed, there is an unusual brilliancy of the eyes, and the patient is often unusually gay and cheerful. A slight degree of emaciation and some loss of strength ensues almost imperceptibly. This state may last from nine months to two years or more.

But nothing is more various than the commencement of phthisis: the cough, instead of being trifling at first, may return in frequently repeated single coughs, which are exceedingly loud, ringing, and metallic. The pulse and breathing may remain tranquil.

In a certain number of cases the patient may always have enjoyed good health, be apparently of strong constitution, and have no cough, when suddenly, in the midst of apparently perfect health, he may be taken with a more or less profuse blood-spitting, after which all the symptoms of confirmed consumption may rapidly develope themselves.

a. It is found most commonly in the form of *roundish, separate masses*, which vary in size from that of a hemp-seed to that of the fist, and more; they are enclosed in very delicate cellular capsules, and are composed of a gelatino-fatty, or fatty-marrowy, or truly encephaloid substance; they vary in consistence, and

In other cases dyspnœa may precede the cough and all other symptoms for a length of time.

Auscultation is generally available at an earlier period than percussion. Feeble respiration is one of the most common of the earlier signs; it is most valuable when heard principally on the right side, and generally corresponds with dulness under one, or the other, or both clavicles. In many cases we hear prolonged, rude, blowing inspiration and expiration, and then often find that the percussion sound is clear; this sign is most valuable when heard on the left side. When dulness of percussion corresponds with rude, blowing respiration, the tubercles are frequently situated more posteriorly, and proportionately greater dulness will be found above the ridges of the scapulæ than below the clavicles. In still other cases we only hear interrupted respiration, in which the inspiration, instead of being a soft, continuous, gradually-swelling, and breezy murmur, is broken into a succession of jerks or efforts. These alterations of the respiratory murmur are often attended from an early period by various minute sounds, such as the *pulmonary crumpling sound*, which is as fine as that produced by crumpling tissue-paper or a silk handkerchief in the hand; it is heard in about one-eighth of all cases of incipient phthisis, but only during a limited period; it may be mistaken for dry crepitant rattle, or slight pleuritic friction sound. In other cases, a succession of small sounds are heard, each forming a minute click or crack; when dry, they indicate crude tubercles, and are mostly heard during inspiration; when moist, they point to the softening of the tubercles, and are heard equally during inspiration and expiration. In the progress of the disease, these single clicks, which at first may only be audible at the termination of a deep inspiration, are replaced by a small, thin, mucous rattle, resulting from the admixture of air with the glairy fluid which is present in the minute bronchial tubes. All these sounds may arise from capillary bronchitis in the apices of the lungs, and hence do not necessarily presuppose the existence of tubercles there, but they are pathognomonic when several or more of the following signs are also found: a difference in the contour of the two infra-clavicular regions, one being full and rounded, the other comparatively flat and depressed; when this is the case, if the patient take a deep inspiration, one infra-clavicular region will be elevated as in health, while the other remains comparatively motionless; at times this difference

are generally whitish, although occasionally of a grayish-red or dirty yellowish-gray color. They are generally scattered through the lungs in considerable numbers, and are seated as much to-

may be better appreciated by the hand than the eye: the sounds of the heart may also be heard more distinctly under the clavicles than in health; if more distinctly audible on the right side than the left, this sign acquires double importance: a trembling vibration of the voice, or a slight bronchophony in the same localities, completes the category. [Hughes.] Particular attention should be paid to the quality and duration of the inspiratory and expiratory murmurs; according to Fournet, when a few crude tubercles are seated near the surface of the lungs, the inspiration is dry, rough, about one-sixth louder than normal, and nearly one-fifth shorter; while the expiration is also dry and rough, but nearly four times louder and longer than natural. When groups of crude tubercles are present in the same locality, the pulmonary crumpling sound is often heard, but if pleuritic pains are felt about the clavicles it may be masked by slight pleuritic friction sound; the former is continuous, and is almost exclusively audible during inspiration, while the latter is composed of little shocks or jerks, and the ear detects a sensation of the displacement of something upward and downward, both during inspiration and expiration; the inspiratory murmur is louder and shorter, and the expiratory is five or six times louder and longer, often rude and blowing, and occasionally almost bronchial. When the tubercles soften, moist, crackling ronchus supplies the place of the dry, crackling, or crumpling sounds; inspiration is one-half or two-thirds louder, its duration almost one-half shorter than natural, and it is also slightly bronchial, while the expiration is from six to ten times louder and longer, it has a marked bronchial character, and bronchophony, or even imperfect pectoriloquy, may also be heard.

In the second stage the cough becomes more severe, unless it occurred from the commencement in frequent and harassing kinks, for then it becomes less troublesome, and very slight efforts often suffice to expel the sputa. The expectoration is composed of thick, flat, almost circular, opaque masses, of nearly equal size, which remain distinct from each other, and float on the surface of more or less turbid and semi-transparent serum, at the bottom of which we find large or smaller quantities of the already-described crumb-like and cheesy fragments of crude tubercles; or this so-called *nummular* sputa is entirely hidden in a mass of frothy, bronchitic expectoration; or, finally, we see the whole intermixed with dirty-grayish, or ashy, or reddish, fetid, sanious, porridge-like matter, which comes from the lax, soft, and semi-putrid walls of the caverns.

The hectic fever is now fully established, the evening chills are

wards the surface of the lungs as in its deeper seated portions; when they come in contact with the pulmonary pleura, they are either flattened or else receive a navel-like indentation. The

more severe, and slighter morning paroxysms are added; the fever is greater and more general; the night sweats are more abundant, and very regular in their appearance; during the accession of the hectic the patient now feels more oppressed, the cough is more frequent and painful, and in some cases the expectoration is only characteristic towards the end of the paroxysm. The pulse is frequent at all times, and the respiration hurried; the loss of flesh and strength is now very evident, and the feeling of languor and debility increases; the pains about the chest become more frequent and severe, and the attacks of hæmoptysis more common and copious; the transient hoarseness of the first stage becomes permanent in nearly two-thirds of all the cases. In three-fifths of all consumptive patients the ends of the fingers become fusiform, at least Pigeaux found them so 167 times out of 200 cases of phthisis, while in 183 non-tuberculous, but diseased subjects, he found this state of the nails in 17 only, all which were emaciated, and 13 had more or less dyspnœa; the curvature of the nails depends upon an œdematous infiltration of the pulp of the fingers; the thumb and forefinger are generally affected first; the swelling is greatest at the root of the nail, and tapers forward towards its end, and backward towards the joint; the nails are arched forward, and their ends bent downward: it is more frequent in women than men.

In this stage the results of auscultation and percussion are more positive, although it should always be held in mind that a patient may have obstinate cough, frequent hæmoptysis, short and hurried breathing, emaciation, quick pulse, hot skin, partial and even general night sweats, &c., and yet in rare cases we will find few or no physical signs of disease of the chest, for isolated tubercles do not alter the percussion sound very much, even when scattered through the lungs in considerable numbers; if they be deep-seated, they may even soften and form caverns, the physical signs of which may be completely masked by the normal respiratory murmur from the surrounding healthy parts. Generally, however, the percussion sound is evidently duller under one or both clavicles, while it is clearer than natural lower down the chest, and in the lateral regions. Caverns are almost always present in the lungs in this stage, but their signs are often indistinct, so that we invariably find more abscesses after death than were diagnosed during life. If we hear single but well-defined and rather large mucous bubbles, with either feeble or rude respiration and prolonged expiration around them, we may count on the presence of a cavern about the size of a hickory-nut; when a number of small bronchi open into a pretty large

affection of the surrounding parenchyma is limited to its being forced from its usual locality, and being in a state of compression. Very rarely does it break down into ichor, and form a cancerous, ichorous abscess, which opens into the bronchi. In general it proves fatal, in consequence of the exhaustion of the system induced by its excessive growth ; the more so as it is generally the result of a very high degree of the cancerous cachexia. Pulmonary œdema commonly is superadded, and hydrothorax, with or without simultaneous cancer of the pleura.

cavern we hear intense mucous rattle, instead of gurgling ; when considerable dulness of percussion and bronchial respiration give way within a short time to clearer percussion and gurgling, we may be sure that a large tubercular mass has softened down rapidly, but generally the transition from single to numerous mucous clicks, and from thence to mucous rattle, with small and then large bubbles, and finally to gurgling, is very gradual; sometimes when auscultation does not discover a cavern to us, if the tips of the fingers be applied over the suspected spot, at each word uttered by the patient, a sensation *(fremissement)* will be felt extending along the fingers, and at times even to the palm of the hand, as if a vibrating metallic wire had been touched—at times it is almost painful ; if the cavern be pretty large and near the surface of the lung, percussion produces a tympanitic sound ; when the cavern is empty we also hear cavernous breathing, i. e., a sound is heard at each inspiration, as if air were forced into a large bottle—also cavernous cough and pectoriloquy ; when it is full we hear large mucous rattle or gurgling, and the commotion of the fluid within may be so great as to be felt by the hand applied to the chest, and by the patient ; the resonance of the voice and cough may be so strong that the patient is enabled to point out the exact seat of the abscess ; if located in the midst of a mass of tubercular infiltration, caverns give forth an evident tympanitic sound, even when deep-seated ; if covered by much healthy tissue, the only sound which can be produced is the cracked-pot sound, although this is most frequently heard over large superficial abscesses ; moderate sized caverns with walls several lines thick afford bronchophony, bronchial respiration, and various rattles which consonate with the resonance of the voice and cough ; caverns with very thick and rigid walls receive and eject very little air during inspiration and expiration, and hence these acts afford no sounds, but distant noises may resound in them in a peculiar murmur ; caverns which afford metallic tinkling and resonance are at least as large as a man's fist ; if indurated parenchyma be interposed between a cavern and the heart, we sometimes hear a single, but oft-repeated tick, like that of a watch or small clock, which corresponds with the motions of

Pulmonary cancer is very rarely a primitive disease, i. e., it is not often the first in order of the successively developed cancers, but almost always exists in company with other, and in fact numerous cancerous deposits of older date in other organs; it often is developed with great celerity after the extirpation of cancer of the breast. It is most frequently combined with cancer of the pleura, with which it is commonly developed simultaneously; or with cancer of the mediastinum, mammary glands, liver, kidneys, bones, &c.

the heart, so that we can count the pulse by it; at times a similar ticking attends each inspiration; if numerous small caverns of the size of a pea or bean are situated in one lobe, we frequently hear dry, large, bubbling, crepitant rattle, which, however, is often masked by the bronchial rales which always attend it, &c. [Skoda.]

The signs of the third stage of phthisis are too well known to require repetition here.

Latent phthisis. We have already described the laryngeal variety: chronic diarrhœa, with emaciation, more or less, and even double hectic fever and night sweats, are at times the first symptoms of a consumption in which the patient neither has, nor ever had, any cough, or difficulty of breathing; but after a time slight cough sets in, the breathing becomes oppressed, hæmoptysis ensues, &c. In some cases irritation of the stomach acts as a revulsive upon phthisis, the cough becomes rarer, the expectoration less copious, the breathing freer, the sweats cease, &c., but the affection of the lungs is still progressing silently. In other cases, emaciation and loss of strength are the only symptoms. Louis calculates that nearly one-fifteenth of all cases of phthisis are latent, and only to be detected by means of physical exploration of the chest.

Acute phthisis is a most severe and obscure disease, which may run its course in from twenty to sixty days. In the midst of health the patient is seized with rigors, and more or less violent trembling, which is renewed on the following days, and followed by persistent heat, as intense as that of typhoid fever; extreme thirst, frequency of respiration, marked oppression of the breathing, often amounting to complete orthopnœa, which is out of all proportion to the trivial signs of slight pulmonary catarrh detected by examining the chest; the lips and face may become livid, more or less severe palpitations and violent action of the heart ensue, owing to the obstruction to the circulation through the lungs, and other symptoms referable to disease of the heart may arise; if cough, slight or severe, with or without expectoration of blood, sets in, the case will become more clear, although it often has been and will be mistaken for the first stage of pneumonia, or severe bronchitis, or typhus fever, &c.

b. Much more rarely it is found in the lungs as *a peculiar variety of tubercles*, but never except in company with cancer of some other organ. They consist of tubercles or nodules, of the size of a barley-corn or hemp-seed, which, as far as is yet known, may be distinguished from true tubercles by their whitish or bluish-white color, their softer consistence, by their agglomeration into groups, and by the difference in their structure and composition. They are found at times aside of *genuine* but *retrograde pulmonary tubercles.*

c. It is excessively rare to find cancerous matter *infiltrated* or effused *into the air-cells.* This is the result of a pneumonia, which, under the influence of a dyscrasia that frequently has been aroused by the extirpation of an external cancer, deposits a product which assumes the characteristics and elementary structure of cancer; hence the lung seems to be hepatized by cancerous matter.

Medullary cancer is at times colored black of various shades and degrees, by a pigment which enters into its composition; then it is either punctuated, streaked, or thoroughly saturated with a brown, blackish-blue, violet, or black color, and hence has received the name of cancer melanodes. We have never found it except in connection with general, and in part very acute, medullary cancer.*

* *Diagnosis.*—Cancer of the lungs is an excessively rare disease, and even the most experienced physicians have observed single cases only; of 9118 cases of cancer in various organs, collected by Tanchon, there were only seven examples of cancer of the lungs. It seems to be more frequent in males than females, for of 42 cases, collected by Kleffens and Hasse, 30 were in males and only 12 in females. Hughes thinks that it occurs almost exclusively in the right lung, for in 11 cases reported by Sims, Taylor, Graves, Stokes, Walshe, and Hughes, all were on the right side; but in 19 of Kleffens' cases, 7 were on the left, 6 on the right, and 6 on both sides. It is most common between the twentieth and fortieth year of life. Isolated encephaloid tumors are rarely attended with distinct symptoms or physical signs. The proportion of double cases is thought to be much less than that derived from a comparison of Kleffens' cases, so that the absence of disease in one lung, while the other is affected to a considerable extent, has its influence in leading us to consider such a case as one of cancer and not of tubercles. The pain is at times characteristic, viz., severe and darting, as if electric sparks were passing through the chest. The peculiar straw-colored

tint of the skin of the patient may often lead us to suspect the existence of cancer. In nearly one-half of the cases, cancerous tubera are found in external parts, and this again removes much of the difficulty of the diagnosis. Hughes lays great stress upon the peculiarity of the sputa, which occasionally consist of blood so thoroughly incorporated with serous fluid as to resemble either red or black currant jelly. In many cases there are evidences of obstruction in the superficial veins of the affected side, evinced either by an enlargement of the vessels themselves, or by œdema of the parts situated just below the diseased organ; thus enlargement and extreme congestion of the jugular, axillary, mammary, and superior epigastric veins, has been noticed; also a notable difference in point of fulness between the two radial pulses; œdema of the affected side, corresponding arm, and side of the face; fulness of the neck. Prominence of the eye-balls has been noticed, which, combined with the condition of the neck and face, gives the patient the appearance of a strangled person, to a certain extent. The malignant growth is also apt to compress the œsophagus and cause dysphagia. [Walshe.] In infiltrated cancer of the lungs the chest is never dilated, but frequently contracted to about the same extent as in chronic pneumonia; in tuberous cancer there is a partial dilatation of the affected side. The intensity of the dull sound on percussion is greater than any other disease, and the resistance of the walls of the chest to the percussing finger is often so great that Walshe found it almost as painful to use the finger as a pleximeter. The bronchial tubes are very apt to become compressed and even obliterated, which is not the case in chronic pneumonia and tubercular infiltration; hence the respiration, which at first is strongly bronchial, gradually becomes weaker and weaker, and all respiratory sound may cease to be heard during the progress of the disease in some places, or even over a whole lung. It may readily be mistaken for chronic pleurisy, tubercular infiltration of the lungs, or chronic pneumonia; still the above signs will enable us to detect at least a small majority of cases.

APPENDIX.

1. DISEASES OF THE THYROID GLAND.

The thyroid gland is subjected, as a rule, to but few diseases; and, as its structure and functions are but little known, the same is for the most part the case with its diseases.

It, however, often presents alterations in *size*, and in particular it is very frequently and very considerably enlarged. *This increase of volume* is at times transient, and arises rapidly, from congestion or inflammation of the organ, and occasionally in the so-called lymphatic goitre; at other times it is permanent and increases gradually, as is observed in the latter stages of the above mentioned goitre. It either affects the whole of the gland equally, so that this retains its original shape, or else it attacks one lobe only, or only a small portion of it, whence the pressure which it naturally exerts upon the trachea and larynx is increased in various directions, and it may even encroach upon the pharynx and œsophagus, the bloodvessels and nerves of both sides of the neck, and even upon the trachea, bronchi, and some of the bloodvessels seated within the thorax. Those enlargements are less frequent, but the more important, in which the thyroid body grows around the œsophagus in the form of a ring, and those in which the isthmus of the gland enlarges so as to form a middle lobe which descends along the trachea into the thorax behind the manubrium sterni, at the semi-lunar notch of which it becomes contracted in size, but increases greatly immediately below. It causes the so-called *asthma thyreoideum.*

Decrease in volume or *atrophy* of the thyroid gland is an affection of but little interest.

Hyperæmia of this gland is of pretty frequent occurrence, especially from mechanical causes which prevent the emptying of the vena cava descendens and the right side of the heart. It may be either permanent or transient, and is characterized by the darker color of the gland, its abundance of blood, its softness

and swelling, forming the so-called hyperæmic turgescence of the thyroid body. Apoplexy of this organ is very rare, provided its tissues be in a normal state.

Primitive inflammation is one of the very rarest occurrences, at least as an object of pathologico-anatomical investigation. On the other hand we find *accumulations of pus* in it at times, but generally in combination with numerous similar deposits in other organs, arising in the train of puerperal uterine phlebitis. Thyroid abscesses may break into one or the other of the mediastinæ, or into the trachea, but most frequently they open into the left side of the œsophagus.

The most frequent disease of the thyroid body is that which is called *struma*, in the narrow sense of the word; its most striking feature is an increase in volume. In the more common and slighter degrees it consists in a quite simple alteration of texture, arising from a more evident development of the cellular structure of the organ. This either takes place equally throughout the whole gland, which then contains cells of equal size; or else we find one, or several, or very many discrete or agglomerated cells, which are converted into roundish or oblong cysts with delicate membranous walls. They contain a gum-elastic or glue-like yellowish, brownish, or greenish matter (*colloid*) in their cavities; if it has attained some consistence, the cut surface of the gland will present a bacon-like appearance, and has a peculiar waxy-doughy feel. The gland under such circumstances is pale and bloodless, and has increased more or less in size, without any proportionate alteration in form.

From unknown circumstances the secretions within the cavities undergo modifications at times, either from the very commencement or during the progress of the disease; at other times the walls of the cells are more evidently altered. In the former case we find gelatinous or albuminous substances, of whitish-gray or flesh-color, deposited in the form of concretions, which may readily be lifted out of the cells, or else they are seated in the meshes of an extremely delicate cellular net-work of new formation. In the latter case the walls of the cells increase in thickness, and become converted into a sero-fibrous cyst, which may enclose the most varied contents, besides those already described; they often attain an astonishing size. These alterations form those strumæ which have received the names of *st. lymphatica* and *st. cystica*.

Irritation doubtless lies at the foundation of these processes:

this is the more probable, as repeated attacks of inflammation take place in the walls of the dilated cells, and especially of the above described cysts, during the ordinary course of the disease, although they often pass unnoticed. These inflammations deposit the most various exudations here, in like manner as they do upon normal serous or sero-fibrous tissues; but the hemorrhagic varieties, as might be expected from the newness of the tissue, are the most frequent, and are followed by the separation of large masses of fibrin from the effused blood. These latter and the walls of the cysts undergo the same transformations as the exudations from, and the walls of normal serous sacs do; they even change into chalky concretions or become ossified. Under such circumstances the cysts not unfrequently become entirely obliterated, as they contract around the exudation, and we then find dense, more or less voluminous, knobby, cartilaginous, bony, or chalky concretions scattered through the gland.

At times we find true effusions of blood into the cavities of the dilated cells, and in the cysts.

The predisposition on the part of the parenchyma of the thyroid gland to the *formation of cysts*, extends itself to the neighboring cellular tissue in a remarkable manner, for nowhere do we find small or large cysts containing serous, gelatinous, or glue-like contents, so frequently as in the neighborhood of this body.

Other heterologous formations, except the above mentioned serous and fibrous cysts, and cartilaginous and bony concretions, occur but very rarely in the thyroid gland. *Tubercles* are scarcely or ever found in it, and *medullary cancer* only occasionally.

Note.—The diagnosis of the different varieties of goitre is of especial service in connection with the use of iodine; it is specific against *struma lymphatica;* and its external use will dissipate the goitre-like distention of the cellular tissue of the thyroid gland; while it is utterly useless in *struma cystica*, and comparatively so in *struma vasculosa and scirrhosa.* The two former are painless, but it frequently happens that, after the first inunction or internal use of iodine, they become harder and painful, instead of becoming softer, but in about eight days the skin over it becomes less tense and more wrinkled; the swelling softens and gradually decreases in size. Of 670 cases treated by Manson, Bayle, Bardsley, and Coindet, 422 were cured, 16 relieved, and 161 either very slightly or not at all improved. From the experience of others it is fair to infer that iodine was not adapted to the latter cases, and it is notorious that immense mischief occurred in some of them from the large doses used to force a cure.

2. DISEASES OF THE THYMUS GLAND.

These are still more rare than those of the thyroid body. The most important is a more or less considerable abnormal increase of its size in new-born children, and a deficient involution of it, whence it persists in retaining its original volume as late as the fifth, sixth, or seventh year of life, or even to and beyond the age of puberty. When hypertrophied, we almost always find a simultaneous predominance of the whole glandular system, or it occurs in connection with rhachitis, or hypertrophy of the brain. It then either presents two lateral, flattish-round, and thick lobes, which extend downward on both sides, into the posterior mediastinum; or else it consists of a single tongue-like mass, which projects downward upon the pericardium and right ventricle. More extended observation and careful examination is still requisite in order to decide whether the so-called *asthma thymicum* of delicate children really depends upon the pressure of an enlarged thymus gland upon the air-passages, or whether there be any essential connection between this disease and organ, or with hypertrophy of the brain. *Tubercles* are more common in the thymus gland than cancer; but both are rare.

Note.—The thymus gland acquires its greatest normal size during the first year of life, and then is about two and a half inches long, one and a half wide, about four lines thick, and weighs about two hundred and fifty grains. Peter Frank was the first to call attention to thymic asthma, but the matter was neglected until Kopp in 1829 endeavored to show that almost all fatal cases of asthma in children arose from the pressure of an enlarged thymus upon the trachea. Caspari and Pagenstecher were the first to prove that the so-called thymic asthma often occurred when the gland was even remarkably smaller than natural, and *vice versa* that very large thymus glands in many cases induce no attacks of asthma. Albers has laid much stress on the fact that an enlarged thymus is much more apt to cause sudden death by preventing the return of the blood into the heart, by the pressure which it often exerts upon the descending vena cava and right auricle. Still, cases have occurred in which hypertrophy of the thymus has produced sudden death under asthmatic sufferings, without there being any sufficient reason to attribute these symptoms to any other cause; but fatal asthma in children also arises from various affections of the organs of respiration and circulation, from the pressure of enlarged lymphatic glands, &c.; and at times it is a purely nervous and spasmodic affection.

Thymic asthma is more common during the first than any subse-

quent year of a child's life; the little patient, who perhaps a minute before had been in perfect health, is seized with a most painful difficulty of breathing, each act of which may be attended with a whistling or crowing sound, as if the air had to pass through a narrow metallic opening; this frightful state may last for a minute or two, when the respiration ceases, the face becomes livid, the limbs cold, and the pulse small, weak, and intermitting, or irregular; the body becomes convulsed, stiff, and bent backward: it is said to be characteristic, that the child stretches its tongue forward and lets it hang out over the lower lip. Sometimes the child recovers from it with a scream, and the breathing resumes its normal regularity: at other times it begins to breathe deeply, and, after a longer or shorter interval, to cry loudly, shrilly, and interruptedly; or the recovery is more gradual, and the little patient remains exhausted and drowsy for a considerable period; or, when the attack begins to yield, five or six shrill inspirations, not followed by expirations, are made, the body loses its spasmodic stiffness, and after a short fit of groaning or crying the child may seem almost well. These attacks are most apt to occur when the child is suddenly started up from sleep, or after a paroxysm of fretfulness or crying, or when it has nursed and become satiated. At first the attacks are rare, and occur at long, irregular intervals, but gradually become more frequent and severe, so that in extreme cases, ten or even twenty paroxysms have happened in the course of twenty-four hours. Often the child is suffocated in a minute or two, or, if it survive this period, convulsions are apt to ensue, and death occurs under symptoms of an apoplectic character; or the case is more chronic and lasts for a half or even two years, the attacks becoming more frequent and violent all the time. It is apt to run in families; boys are more subject to it than girls. [Schoenlein.] According to Wood, most cases of so-called chronic, spasmodic, or cerebral croup, are examples of thymic asthma; and enlargement of the thymus gland may be inferred to be present in all cases in which a child is cut off suddenly, without any previous complaint, after a fit of crying, or violent vexation, or irritation of any kind. When the thymus gland is very large, there will be dulness on percussion over the sternum, and absence of the respiratory murmur there, as the lungs are forced back into the posterior parts of the chest.

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