have applied this girdle many hundreds of times, and have very often seen rapid improvement ensue; nevertheless, it cannot be denied that the half of these children perish notwithstanding. If *cold* compresses are applied to the children, as recommended by some authors, a cry of fright is the consequence; the child is seized with a feeling of dread, the breathing is palpably accelerated, and does not subside until the cold water has become warm through the temperature of the skin. Hence it seems more rational to make the compresses warm at once, by using warm water, in order to avoid the temporary restlessness and discomfort to the child.

(3.) Acquired Atelectasis of the Lungs.—Congenital atelectasis has already been treated of (on page 54) in connection with the diseases which are regarded as the immediate effects of the delivery; it therefore only remains for us to speak of the acquired atelectasis. This affection has the most intimate connection with rachitis of the thorax, and therefore mostly occurs in children between the ages of six months and three years. In many cases the augmentation in the density of the pulmonary tissue and the final atelectasis are due to a marked curvature of the spine, to a distended pericardium, hypertrophied heart, to aneurisms or neoplasms. It is found most exquisitely marked in pleuritic exudations, where the lung is compressed to the thickness of a finger and correspondingly condensed.

Pathological Anatomy.—The degrees of atelectasis vary exceedingly. A mere increase in the density may occur, which is recognized by the augmented consistency, but the compression may also attain to such a high degree as to cause a total obliteration of the alveoli, and the disappearance of the capillary vessels. At first these compressed and atelectic places contain blood and have a great similarity to muscle, on account of which this condition has been called carnification; but, when it has existed for some time, they become bluish brown or gray, shrink up into a leathery rind, the pulmonary tissue cannot be recognized, and is converted into a fibro-cellular mass, which is gradually displaced by the slightly emphysematous surrounding parts, and ultimately disappears altogether. Such solitary atelectic places are very rarely found, at least, in older children and adults. Sometimes it is still possible to inflate such atelectic places, if they are of but recent formation; generally, however, this experiment proves fruitless, for the alveoli have actually disappeared, and been replaced by a fibro-cellular mass.

When the lesion is extensive, it will have a similar effect upon the circulation as pulmonary emphysema. The capillary circulation becomes so impeded here, that a stasis takes place in the trunk of the pulmonary artery, producing dilatation of the right side of the heart, and finally venous stagnation and cyanosis.

The cause of acquired atelectasis is therefore chiefly to be sought in the rachitic thorax; the latter, however, originates in the following manner: The inspiration is brought about by the contraction of the inspiratory muscles, and a dilatation of the pulmonary vesicles is thereby produced. A momentary rarefaction of the air within them results, which helps to overcome the atmospheric pressure which is becoming stronger and stronger upon the thorax, aided by the elastic pulmonary tissue, which drags inwardly at every inspiration. The combined effects of these forces is an inward curving of the intercostal spaces, and, in lean persons, of the clavicular region also. I was once able to see this condition most strikingly displayed in a child in whom a rib was broken in two places by the shaft of a wagon running against it. The fragment of the rib, one and a half inches in length, was kept in place by mere skin, and flapped in and out with every inspiration and expiration, like the valve of a bellows. If the bony ribs have lost their firmness by being deprived of some of the calcareous salts, they will also participate in the inward movement, which otherwise is only seen in the intercostal muscles, and thereby lose their external convex shape. Moreover, they also yield to the diaphragm, which, by the pressure of the abdominal viscera, drags upon them so as to retard their longitudinal growth (producing rachitic shortening of the bones). By these various forces is finally produced a distorted, contracted, and misshapen thorax, the contents of which necessarily must suffer, more especially as, in consequence of the curving and retarded growth of the spinal column, it is also lessened in perpendicular dimension.

Symptoms.—In consequence of the diminished number of pulmonary cells containing air, an acceleration of the respiration necessarily must result, if an interchange of gases corresponding to the bodily weight is to take place. The respirations, in fact, are quickened and executed with considerable exertion, the alæ nasi thereby participating. The application of the stethoscope to the rachitic thorax is attended by many difficulties, for the button-like sternal ends of the ribs, and the concavities in the region of the nipples, render a perfect adaptation of the instrument impossible. We almost always have to confine ourselves to an immediate auscultation of the back, and generally hear sonorous râles in all parts, because the bronchi leading to the atelectic portions are affected with catarrhal inflammation. Over the diseased places proper crepitating râles and bronchial breathing are heard, provided the sonorous râles do not drown all other sounds. But on the infantile thorax, and especially the rachitic, the vesicular, puerile breathing is so sharp, and the expiration so loud, that the distinction between puerile and bronchial breathing consists

only in a fine modification of the sounds, and the utmost skill is requisite to distinguish with certainty between the two.

By percussion it is but rarely possible to demonstrate the atelectic places, for, in most cases, they are too small in extent, and very frequently border on the liver, where, by the incarceration of the borders of the lungs between the upper surface of the liver and the inwardly-curved ribs, a condensation of the tissue is produced. Besides, we must always take into consideration the physiological dulness during the abdominal pressure, the rachitic condensation of the scapular portion, and the similar condition from the curvature of the spinal column that very frequently occurs, before we can ascribe a discovered dulness to atelectasis.

From what has been said hitherto, no difference will have been discovered between the symptoms of pneumonia and those of acquired atelectasis, and in reality there is but one symptom by which we are enabled at the very first sight to discriminate between these two conditions. In pneumonia a burning hot skin is always present; in atelectasis, on the contrary, it is absent. But when, in a rachitic child, with acquired atelectasis casually, or from dentition, or some other acute affection, fever becomes superadded, then no one is able to decide from one examination as to the correct diagnosis. Only the course of the accidental complication, the continuance of the dyspnœa and the respiratory modus after the fever has disappeared, can clear up the obscurity and aid us in the diagnosis. This diagnostic difficulty is an additional reason why pneumonia should not be treated instantly by leeches and antiphlogistics. In all cases such a treatment agrees very badly with rachitic children.

The progress of rachitic acquired atelectasis is always very gradual; the course is chronic, and may be prolonged for years. With increasing invigoration, and recommencing growth of the ribs, the respirations become slower, the strong inward curving of the fourth to the eighth ribs decreases with every inspiration, the pigeon-breast subsides, the auxiliary respiratory muscles of the neck and alæ nasi cease to participate actively.

But if no such consolidation of the thorax takes place after several months, and if the atelectasis progresses and implicates still larger portions of pulmonary tissue, then the portions that still remained normal will be unable to perform the extra amount of labor thus imposed upon them. A still more intense bronchitis is now liable to supervene, and the subjects die from paroxysms of suffocative cough, after having suffered for weeks, and even months, from the most violent dyspnœa. Œdema of the feet precedes death in these cases sometimes several weeks.

The prognosis depends upon the degree and the duration of the conditions. The more developed the pigeon-breast, the more extensive the solidification of the tissue, the greater the dyspnœa, the more imminent is the danger of the child's being carried off by a slight bronchial catarrh, or by hydræmia, in consequence of defective metamorphosis of the materials. And yet, even very decided disfigurements of the thorax, and the atelectasis resulting therefrom, are often completely recovered from.

Treatment.—The first question always is that of the nutrition, the second that of the residence. As the pigeon-breast only develops itself from the sixth to the ninth month, the children are usually already weaned and fed upon various kinds of broths and soups. However, it is not possible to maintain that any of these methods of nutrition are absolutely injurious, for upon all of them great numbers of children thrive as well as die; and it cannot even be decided which of these ward off the rachitis best, for it occurs in all kinds of diet and all manner of nutrition. The most important point in this relation is, that the food should be well borne and assimilated, and that no diarrhœa or other kinds of digestive disturbance be produced by it. Children with perfectly regular digestion very rarely become rachitic.

Living in damp houses materially promotes the production of rachitis, on account of which it is also much more frequent in winter than in summer, and among the poorer class of people than among the rich. Consequently, the treatment must be chiefly directed to the procurement of well-ventilated, dry rooms, and as long a residence in the country as possible. Where these conditions are unattainable, the termination will generally be unfavorable. Our efforts to eradicate the bronchitis, which invariably accompanies atelectasis, by the use of expectorants, narcotics, or any other class of remedies, will almost always be fruitless. This complication subsides spontaneously, as soon as the lungs have again acquired a more capacious and better condition. I confine my treatment to inunctions of fat upon the breast, several times daily, and internally give *ol. jecor.*, or the malate of iron, more precise indications for which will be given further on, in the treatment of rachitis.

(4.) Pulmonary Emphysema (τὸ ἐμφύσημα, to inflate).—The well-known blubber-like emphysema of the lungs, from which adult patients acquire a barrel-like thorax, and suffer from displacement of the heart and diaphragm, is scarcely ever seen in children; indeed, this kind of rarefaction of the pulmonary tissue seems to be altogether absent in the infantile organism. On the other hand, a vesicular and interstitial emphysema is often found under the following pathological conditions:

Pathological Anatomy.—Purely vesicular emphysema consists in a permanent dilatation of a large section of pulmonary alveoli, which, however, are not ruptured, but only distended to perhaps twice their normal size. This species of alteration of the pulmonary tissue is almost invariable in the vicinity of condensed portions; thus along with pneumonia, atelectasis, and tuberculosis, it is often found. Emphysematous lungs do not collapse on opening the thorax, have a peculiar feel, like a cushion filled with air, are grayish or yellowish gray, anæmic, and, when incised, collapse with a hissing, slightly-crepitating sound. When the condition is of long-standing and in progressive atrophy of the alveolar walls, *interlobular emphysema* invariably becomes superadded.

This condition consists in an accumulation of air in the cellular tissue connecting the different pulmonary lobules with each other, and can only be produced by the rupture of some of the pulmonary cells, and by the escape of air into the adjacent interlobular interstices. Larger or smaller transparent air-bubbles then appear on the surface of the lung, beneath the pleura, which may be displaced in the direction of the interstices, and also ramify into the deeper structures of the lung. Sometimes they circumscribe a pulmonary lobule, in the shape of an island, and, when the interlobular emphysema has developed itself between many neighboring lobules, form large air-bubbles, which may be pushed hither and thither over extensive portions of the pleural surface of the lung. The escape of air into the connective tissue surrounding the bronchi, into the mediastinum anticum, and thence out upon the neck and breast, is a very rare occurrence. These instances almost invariably terminate fatally.

In regard to the origin of the ordinary emphysema, many, and in part untenable, views still exist. It is certain that solidification of one portion of the pulmonary parenchyma will produce a vicarious vesicular emphysema of the rest of the tissue, and that, in the autopsies of atrophic children, principally as the effects of enteritis folliculosa and cholera infantum, interlobular emphysema is usually found. I have formed no positive conclusions upon the occurrence of emphysema from pertussis, as stated in so many text-books; on the whole, I am unable to recall a single instance of ever having met with it in the autopsy of a child who died from pertussis or any of its complications. *Rilliet* and *Barthez* also are opposed to the recognition of this complication; and it follows from this that, aside from the mechanical distention of the alveoli, which, in forced expiration, too, may be produced at the expense of the amount of blood in the lungs, still another special disturbance of the nutrition of the alveolar walls must be present, without which, notwithstanding all exciting causes, no emphysema

could be brought about. The inflation of air, in asphyxiated new-born children, has been suggested as an additional cause, but which is not very probable, in view of the fact that the lungs of the new-born child may be inflated, after death, with all the strength possible, without rupturing their air-vesicles. The lungs are so distensible, and, by forced inflation, may be enlarged to such a degree, that one lung will fill up the entire thoracic cavity, and yet, as soon as the air is allowed to escape, it collapses again, without leaving the least trace of emphysema behind.

Symptoms.—In children, the barrel-like shape of the thorax never develops itself, because, as it appears, they scarcely ever suffer from chronic, but always from acute emphysema, and, for that reason also, that depressed state of the diaphragm is not produced. Hence, we have no physical signs for it, and it is a very great question whether the acceleration of the respiration, mentioned in the text-books, had not better be imputed to the pulmonary affections producing the emphysema than to the emphysema *per se*. This condition, therefore, is only of anatomo-pathological importance. The prognosis and the therapeutics, in a disease in which the diagnosis is so uncertain, are, of course, altogether out of the question.

(5.) Œdema Pulmonum (*οιδημα*, a swelling).—In most of the diseases of the heart, of the large vessels, and of the lungs, a rapidly-fatal pulmonary œdema supervenes as the final pathological state. Then, of course, it has but little importance as a pathological condition, and is only to be regarded as the beginning of death. On the other hand, in measles, and more frequently in scarlatina, a rapidly-developed pulmonary œdema is met with. It does not, however, always lead to death, but disappears spontaneously or by proper remedies. It is to this latter form in particular that our attention is to be directed here.

Pathological Anatomy.—By pulmonary œdema we understand a transudation of serum into the pulmonary alveoli, the finest bronchi, and into the interstitial tissue. Neither the first nor the last alone can become infiltrated with serum without the participation of the others; and the disputes of some authors, whether the œdema has its site in the alveoli, or in the interstices, may therefore be decided in favor of both. Œdematous lungs do not collapse on opening the thorax, are of a grayish-blue or yellowish-gray color, according to the quantity of blood in the affected parts, are heavier than the healthy lung, swim in water, and crepitate strongly on pressure. The pressure of the finger leaves a pit behind, for the corresponding pleura is also œdematous. On section, the œdematous lung presents a smooth glistening surface, from which a large quantity of reddish or yellowish fine-frothy serum escapes on the least pressure. The escape of this

froth is also accompanied by a hissing or crepitating noise. Œdema of the lungs is never confined to small portions of the pulmonary tissues, but generally affects the lower lobes of both lungs, a proof that its cause is not a local, but a general one, and that it must be due to a disturbance of the circulation. Œdematous lungs may be inflated, and thus it is seen that not all the alveoli are filled with serum. The corresponding bronchi contain mucus, and in the bronchi of the higher order a similar frothy serum, like that which oozes out from the cut surfaces, is always found.

Symptoms.—The predominating symptom is a marked dyspnœa, which rapidly becomes aggravated to such a degree as to actually endanger life by suffocation, and may terminate fatally in a few hours. When the children are already large enough, and when their strength allows them, they will raise themselves and sit upright in bed, in order to acquire the utmost dilatation of the thorax possible. Small children while in the recumbent position are seized with severe fits of suffocation, rendering it necessary to raise them up immediately. The breathing is extremely rapid, gasping, and rattling, and the voice grows low and indistinct. The cough is loose; older children produce also a little white foam at the mouth. The pulse is very small, but, as regards the number of beats, stands in no relation to the frequency of the respiration.

In extensive œdema the physical investigation gives a less sonorous but never a completely dull percussion-sound. As œdema of the lungs is mostly bilateral, and the dulness not very intense, percussion therefore often furnishes no very satisfactory information concerning the existing alteration of the lungs. Auscultation is of greater importance. Extensively-diffused, moist, sibilant râles are heard over the œdematous places, which the practised ear readily distinguishes, by the coarser and less regular sound, from fine crepitation of pneumonia. They are often drowned by the large sonorous râles produced in the larger bronchi by the accumulation of mucus within them, but these, after a violent cough, momentarily disappear. If a hand is laid upon the chest, it will feel these rhonchi extremely strong, while crepitation usually is not perceived by palpation. It is very difficult to distinguish pneumonia from œdema of the lungs, especially in those acute cases of œdema where it is attended by active fever. The dyspnœa, if possible, is even greater in œdema than in inflammation of the lungs, but the physical examination supplies no characteristic differences; the only symptom that tends to make the existence of œdema tolerably certain is the bilateral appearance of crepitation, while lobar pneumonia very generally is only observed on one side.

The prognosis, if the cause of the condition is not due to cardiac

disease nor to a chronic disease of the lungs, is not so unfavorable as the first impression would lead one to suppose. Children attacked by nephritis and consecutive œdema of the lungs, after scarlatina, frequently suffer from the most intense dyspnœa, their faces are disfigured by swelling, and one supposes that a speedy end may be prognosticated with certainty; but after a while they rally somewhat, the albumen and casts in the urine diminish, and at the same time the urine is voided in increased quantities.

Treatment.—For nephritis after scarlatina as a cause of this pulmonary affection, the antiphlogistic treatment, with calomel, purgatives, and abstraction of blood, has proved itself to be decidedly injurious. The dyspnœa of older children may indeed be rapidly relieved by venesection, but it soon becomes as torturing as before, and, anæmia now having become superadded, the condition will be found to be vastly aggravated. A large number of dry cups applied to the back and breast mitigates the dyspnœa very considerably, and this remedy may be repeated once or twice daily without any harm or special annoyance. The utmost attention is to be paid to the state of the skin, which should be made to act energetically. The best means by which to accomplish this purpose is to wash it with a highly-diluted solution of lye. The secretion of urine, according to the observations which I have hitherto instituted, is not stimulated by any remedy so well as by the widely-known and popular roob * juniperi, of which half a teaspoonful may be given once or twice daily. It has also the advantage of having no unfavorable effect upon the appetite and stools, and that, mixed with syrup or honey, children are able to take it for a long time. The other diuretics, squills, digitalis, and acetate of potash, taste badly and their use is attended by numerous concomitant disagreeable effects, and therefore they are much less appropriate than *roob juniperi*. In the higher grade of dyspnœa an emetic of ipecacuanha and tartarized antimony often performs very efficient service.

(6.) Hæmorrhage from the Lungs (*Hæmorrhagia Pulmonum—Hæmoptysis*).—Three kinds of bleeding from the lungs are known to occur in the adult—either in the form of bloody sputa for a long time, or the blood suddenly bursts out from the mouth and nose in a stream, or the patient sinks down in a state of unconsciousness, and, after he has regained his faculties, is seized with coughing up of blood. In children, so far as I am aware, the second form only occurs, and is a complication of two very different conditions, whooping cough and tuberculosis. In some epidemics of pertussis, large quantities of blood are very frequently poured out from the mouth and nose, but the invariably favorable course, the absence of consecutive

* See note on page 223.

bloody sputa and all other bad effects, give rise to tolerably well-grounded doubts whether the blood does actually come from the lungs, or whether it is not merely the effect of the violent paroxysms of cough, and comes from some small lacerated vessels or capillaries in the larynx. The last of the two just-mentioned sources seems to me, in fact, to be the most probable.

Pulmonary hæmorrhage of tuberculous children is exceedingly rare. One may see hundreds of them perish from phthisis pulmonalis without meeting with a single instance of hæmoptysis, and where it does occur it is not always seen at the commencement of the tubercular process, but as a closing scene a few days before death. I have never yet observed it in infants, and only once in older children, and that was in a girl ten years old.

The treatment of hæmoptysis, as an effect of pertussis, may be found in the section devoted to that subject. That occurring in tuberculous children is only symptomatic, and consists entirely in the administration of small doses of narcotics to palliate the cough, and for the purpose of procuring euthanasia.

(7.) Hæmoptoic Pulmonary Infarction.—This morbid lesion of the lungs, first accurately described by *Laennec*, is not very unfrequently observed in the autopsies of children who have perished from purpura or pulmonary tuberculosis, and is even found in the newborn child, but in the latter it is generally complicated with pyæmia and the formation of emboli in the lungs.

Pathological Anatomy.—In one lung, and sometimes in both, reddish-black spots, of the size of a pea up to that of a walnut, are found, which are sharply defined from the rest of the pulmonary tissue, and of decidedly greater resistance. The cut surfaces are not dry and smooth, but slightly granular, and the portions of the lungs thus altered are almost as friable as the hepatic parenchyma. The cause of this darker color and augmented resistance is to be found in an extravasation of blood, which has filled up a large number of alveoli, and compressed the interstitial substance. The slightly-granular appearance of the cut surfaces is also explained by the circumstance that the coagulated blood represents a precise cast of the cluster-like arrangement of the alveoli. On scraping such a cut surface with the back of a scalpel, a bloody fluid, mixed with fine, granular blood-coagula, is obtained.

There is great danger of confounding this condition with croupous pneumonia. But, if due attention is given to the circumscribed form of the hæmoptoic infarction, its dark-red color and sharp boundaries, and the dark-red granular serum, which may be scraped off the cut surfaces, it will then hardly be possible to entertain any doubt in regard to the nature of the lesion. When these infarctions have become developed between dark-red hypostatic congested tissues, behind and below for instance, the distinction of color is then lost; still, the greater compactness and fragility, likewise the absence of air-bubbles, supply sufficient cardinal points. Hæmoptoic infarctions are oftener central than peripheral, and, in the latter case, glisten through the pleura. The bronchi leading to them, up to a certain grade, are filled with coagula; the blood, however, generally does not extend very far upward, and consequently no bloody sputa are expectorated.

This condition, according to *Rokitansky*, is often attended by active softening of the right side of the heart, and in severer forms may become complicated with rupture of the pulmonary tissue, when large cavities may be seen filled with blood and loose pulmonary substance. According to the same author, it is also possible for a retrograde development to take place, the infarction either becoming liquid, and assuming a blackish-brown, or rusty and wine-yeast-like color, and thus partly absorbed, partly expectorated by the bronchi, or the coagulated blood shrinks and is metamorphosed into an obsolete fibrous or brown amorphous tissue. In grave instances, the infarction may also become gangrenous, and then present the signs of a perfect gangrene of the lungs.

Symptoms.—Hæmoptoic infarction is never idiopathic, but always complicated with purpura, tuberculosis, and cardiac affections; in all cases the dyspnœa and fever become vastly aggravated by its appearance. Neither by physical examination, nor by any symptoms otherwise developed, are we able to distinguish this condition from lobular pneumonia. Most of the physical signs are devoid of importance, for the reason that the infarction usually occurs about the roots of the lungs, and not on their periphery. A special treatment in a malady so deficient in diagnostic symptoms as this, is, of course, impossible.

(8.) Gangrene of the Lungs (*Gangræna s. Mortificatio Pulmonum*).—Gangrene of the lungs is an exceedingly rare affection in children. It occurs after traumatic pneumonia, produced by foreign bodies, which, during a forced inspiration, have found their way into the lungs, and in the malignant course of acute exanthemata, in noma, in abdominal typhus fever, in pyæmia, and, lastly, as an unfavorable termination of hæmoptoic infarction.

Pathological Anatomy.—Since *Laennec's* time a *diffused* and a *circumscribed* gangrene of the lungs have been distinguished.

The characters of the diffused are: Spreading of a dirty-greenish or brownish-colored putrid slough over larger portions of a lung, over

one lobe, or an entire lung, or the tissues, having become totally liquefied, have a gangrenous odor, and are infiltrated with a flocculent, frothy, gangrenous-odored ichor. This kind of mortification is nowhere strongly defined, but gradually merges into healthy structures, larger or smaller streaks of œdematous tissue being interposed between the gangrenous and sound portions of the lung. It is very rarely observed alone, but generally associated with circumscribed gangrene.

Circumscribed gangrene is oftener met with than the preceding. In this process, a small portion of the tissues at one place becomes transformed into a greenish-black, moist, not easily-lacerable crust or slough, which is sharply defined. In the infant, the size of this slough rarely reaches that of a walnut. After a while this gangrenous plug becomes detached from its normal surroundings, and lies in an excavation of gangrenous pulmonary parenchyma, and bathed by a gangrenous ichor; or it soon dissolves into a sanious, ichorous fluid, and is surrounded by a sloughing excavation, with irregular, shaggy walls. Its site is oftener on the periphery, and in the lower lobes, than in the centre of the lung, and, after it becomes detached, it will drop into the pleural sac, if the pleura is not implicated and firmly united with the costal pleura. This accident results in an ichorous pleuritis, and pneumothorax becomes developed.

The pulmonary tissue surrounding the gangrenous plug is either only œdematous or pneumonic to variable extents; in both instances there is a disposition to assume a diffused mortification, and thus, if the children have lived long enough with this dreadful disease, an entire lobe may be found transformed into a pultaceous, sanious mass. If the arteries coursing through the affected places do not become completely occluded by thrombi, serious hæmorrhage may ensue, the blood escaping by the bronchi, while that which accumulates in the gangrenous cavities tends to increase the gangrenous material. No recovery from traumatic gangrene of the lung has been ever observed.

Symptoms.—The symptoms vary according to the cause of the gangrene. In typhus fever, in noma, and malignant measles, the general disease is so severe, and the susceptibility to the pain, at the same time, so diminished, that no subjective symptoms whatever, and only a few objective symptoms, become noticeable, while traumatic gangrene begins with the symptoms of pneumonia. I once met with such a case, in which a boy, fourteen years of age, had a grain of corn in his mouth, and, from some cause or another, suddenly commenced to laugh, during which the grain slipped into his larynx. For several days thereafter he was still tolerably well, and it was supposed that he was mistaken, and that he had swallowed the corn. But all the

symptoms of pneumonia at length suddenly came on, but did not run the regular course. The sputa became gangrenous, and, through violent paroxysms of coughing, the patient expectorated portions of the grain of corn, and large quantities of sloughing shreds, whose odor contaminated the atmosphere of the room to an unbearable degree. This expectoration continued for several weeks, and did not stop completely until after many months. The boy was reduced to a mere skeleton, and a cavity in the lungs remained, which gradually has diminished in size, and now, after six years, is barely traceable. Many years elapsed before he regained his former health and appearance. This case of gangræna pulmonum is the only one that I have seen terminate favorably.

In the other, non-traumatic, cases of gangrene of the lungs, the disease makes its appearance by a sudden aggravation of the general condition, in which the face, in particular, quickly becomes changed, assumes a leaden hue, and a distorted Hippocratic *facies*, and the pulse becomes extremely small and rapid. The temperature of the skin is not increased; the putrid odor from the mouth is always the most pathognomonic sign, which cannot be attributed to any morbid alteration in the mouth. The physical investigation may prove barren of results, if the process is central, or there be feebly circumscribed dulness, crepitating râles, bronchial breathing, and sibilant râles, or when perforation of the lungs occurs, and signs of pneumothorax appear. Generally, the sputa are bloody, the cough is intense and spasmodic. Colliquative sweats, hectic fever, and delirium, soon become superadded, after which death almost invariably closes the scene.

Treatment.—Where death appears to be inevitable, any rational treatment must be doubtful. The recoveries observed hitherto have been achieved by a treatment with quinine, mineral acids, acetate of lead, chlorine and its preparations, and, finally, with creosote.

(9.) Tuberculosis of the Lungs and Bronchial Glands.—Since we intend to subject the dyscrasiæ, as collective diseases, to a detailed discussion in a special section, it will be sufficient, for the sake of completeness, to speak here of the pathological anatomy and symptomatology of pulmonary tuberculosis, while the etiology and consideration of the general disease will be treated of along with the dyscrasiæ.

Pathological Anatomy.—All kinds of tuberculosis occur in the infantile lung. Thus there is (1), the discrete or miliary tubercle; (2), the aggregated; and (3), the large cheesy tubercular infarction. All the three varieties are often met with in one lung.

Miliary tubercle originates in the pouring out of a rich fibrous