Leaming (J. R.)

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PATHOLOGICAL PROCESSES.

By J. R. LEAMING, M.D.,

Special Consulting Physician in Chest Diseases for St. Luke's Hospital, New York.

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PHYSICAL SIGNS OF INTERPLEURAL PATHOLOGICAL PROCESSES.

In health the pleuræ are smooth, opposing surfaces, free in motion, and lubricated by their natural secretion. They cover the inner costal wall, the outer surface of the pericardium, nearly the entire upper surface of the diaphragm, and all the surfaces of the lungs. They help to form the mediastinum, and surround the origin of the great vessels and of the air-passages. In short, they line the great acoustic chamber of the chest, and cover the sound-producing organs which it contains. The constant motion of these organs gives voice to their action, and breathes into the ear of the auscultator an harmonious idyl of health, or whispers and mutters of the discordances of disease.

The acoustic properties of the normal chest are so perfect that the most delicate signs, such as true respiratory murmur or aortic regurgitation, are delivered through its walls to the ear without loss or change. The healthy pleuræ are no obstacle to the free passage of sound, and at the same time are no cause of sound in themselves. It is like looking into an open room filled with light.

But at the first trace of an inflammatory process they cease to be silent themselves, and modify or prevent sound passing through them. It is like looking into a room filled with cloud, through obscured glass; nothing is clearly seen.

The pleuræ are prone to diseased change. Mental depression, physical exhaustion, or sudden alternations of temperature, may cause hyperæmia, and plastic exudation is then likely to follow. It is a vital process, but indicates a diseased condition of organic life. It occurs in cellular tissue and on serous sur-

faces—very frequently on the pleural. Comparatively few autopsies are made without discovering more or less of interpleural thickening and adhesions.

"Physicians of old did not regard them as preternatural; nor do many at the present day consider them as necessarily connected with inflammation. This opinion is founded upon the fact of these adhesions being met with in individuals not known to have suffered from any inflammatory affection of the chest. But, until satisfactorily traced to some other cause, it would appear more proper to refer these exclusively to an inflammatory origin." (Hasse's *Path. Anat.*, p. 182.) The process may be summarized as follows:

First, local vital exhaustion, vaso-motor paresis, stasis, hyperæmia; then the white globules, "the wandering amœba," pass through the meshes of the walls of the capillaries, and, unless immediately absorbed, organize and result in adhesions and thickened pleura.

More or less of impaired health follows, with obscure symptoms. New exudations take place from time to time, crippling the respiratory organs, and seriously implicating the circulation, until the patient dies, worn out, with resulting complications of all the vital organs.

These serious pathological results have hitherto been unrecognized, except in part and inadequately, during life by physical signs. In March, 1870, I had the honor of reading a paper before the New York Academy of Medicine on Pleuritis. In the discussion which followed, Dr. Flint, Sr., remarked that "He was not aware that there are any distinctive physical signs of permanent adhesions, that can be depended upon as pathognomonic." This was undoubtedly then, and perhaps is largely yet, the received opinion of the profession, Dr. Walshe alone, among the authorities, interpreting certain physical signs as of adhesions and pleural thickening. I had adopted from my teachers the generally received opinions; but considerable clinical experience, obtained in the class of chest diseases at Demilt Dispensary, caused me to question their truth. There were repeated occurrences which seemed to me to furnish incontrovertible proof

that many of the physical signs of the chest had been misinterpreted. But even yet I was not prepared to give up my preconceived idea of the local origin of mucous and crepitant râles.

But about ten years since, a patient came into St. Luke's Hospital, from Bellevue, with a disputed diagnosis. The case had been affirmed to be one of simple hydrothorax, and then again to be hydropneumothorax. Both opinions were correct. Upon examination it was found on the right side that there was dulness and loss of respiratory murmur up to about three inches above the diaphragm; and bordering the upper line of dulness, and encircling the lung, there was crepitus and subcrepitus. It was simple circumscribed hydrothorax. But upon directing the patient to take a forced inspiration and hold the breath, air was forced into the artificial chamber made by the adhesions, and the case was immediately changed into one of hydropneumothorax. In a little while the air escaped, and the case was as at first. To account for this it was necessary to suppose that there was a valvular opening, through which air could come from the lung. The fluid was removed by intercostal incision, and the case kept under observation. After a time, the left side showed signs of disease. There were mucous râles and gurgles, and progressive loss of weight and strength. The case was frequently referred to as an example of tubercular phthisis. The râles were of various sizes, and were considered as signs of tubercular infiltration and honey-combed cavernules.

At the autopsy the circumscribing adhesions, with the valvular opening in the lung, were found on the right side, as was expected; but, to our astonishment, there was no structural change in the left lung. Between the pleural surfaces, however, there was a large amount of plastic exudation, together with a small quantity of viscid fluid; at many points also there were firm adhesions. These interpleural deposits were evidently the only source of the sounds I had misinterpreted as signs of the tubercle and tuberculous cavities of small size. I was convinced that the same conditions had frequently deceived me in other cases.

Since then, I have, in repeated instances, carefully noted and recorded the locality of râles and their distinctive characteristics, for the purpose of testing them in relation to pathological conditions, to be revealed by autopsies. In no instance have I found them to disagree with the interpretation that their cause lay in an interpleural process.

The following case is one in which old, firm, and close interpleural adhesions drew the heart upwards, and caused murmurs by displacements.

Case I.—J. S. T., an honored member of our profession, called on me in company with Dr. Otis, in the spring of 1876. About seven years before he had pleuro-pneumonia, and several times since slight attacks of pleurisy. He was short of breath, and distressed after exertion, or on going upstairs; he had at times severe pain in the region of the heart like angina. Aneurism of the aorta and valvular disease of the heart were feared.

Examination discovered some dulness at the summits of the lungs. There was flat wooden percussion note over both. There was very little expansion, and the movement of the chest was restricted. There were a few râles of various sizes over the greater part of the chest. At the lower angle of the scapula of the left side, there were no râles, nor any movement of the lung even in forced inspiration, but coughing produced short fine crepitus immediately under the ear.

True-respiratory murmur was feeble generally, but absent at the apices of both lungs. The apex beat of the heart was between the fourth and fifth ribs, a little to the left, and there was a systolic murmur.

Diagnosis: old extensive adhesions over both lungs; no disease of the heart or of the arteries.

On the 12th of October, 1876, he received a wound, to the right of the sternum over the right auricle, by a piece of a brass tube imbedding itself in the lung and the pericardium. Pericarditis and pneumonia followed, and he died on the 20th.

The day before his death, and probably for some time previously, there were abundant soft "mucous" râles. These were diagnosticated also as interpleural.

The autopsy revealed pericarditis and pneumonia of the whole of the right lung, which was consolidated—the mould of the ribs remaining on its surface after it was removed from the chest.

Hence, no air could have entered the lung. New exudation had taken place among the old adhesions, and efforts at respiration moved the chest-wall over the solid lung, thus producing "mucous râles."

Case II.—Is kindly given in a letter from Prof. J. L. Little, and is equally decisive: "My dear Doctor, I cheerfully comply with your request, that I would furnish you with the points in the history of a case bearing on the subject of your paper. I was called to see a patient in consultation with Dr. Roëdiger, on August 14th last, and found a man about forty-five years of age, who was suffering slight pain in the left side—no cough, no expectoration, high temperature and frequent feeble pulse. On auscultation, subcrepitant râles could be heard on the posterior surface of the left side of the chest. These were more abundant and of a much coarser quality at the upper part of the lung, although more or less subcrepitation could be heard from apex to base. On percussion, flatness was discovered over the entire upper portion of lung; the lower showed but slight dulness. I saw the patient in consultation on the 17th, 18th, and 19th. At the last visit, eighteen hours before death, subcrepitation was heard, as at first examination. On forced expansion after coughing, the râles were markedly increased in number, and seemed to be very near the ear. Patient died August 20th. Autopsy by Drs. Roëdiger and Nesbitt. Left lung was found solid with pneumonia, except the lower part of the inferior lobe. The upper was in a state of gray hepatization, the middle red. The lower part was very much congested, but crepitated on pressure. The pleuræ were covered with plastic exudation, but the adhesions were slight. The false membrane covering the upper third of the lung was three or four millimetres in thickness. The lower portion was covered with only a thin layer. In this case, Doctor, the râles heard over the posterior surface of the chest were without doubt due to the exudation on the surface of the pleuræ. No air could

possibly have entered the upper or middle portions of the lung for some days before death.

"Yours truly, J. L. LITTLE."

These two cases are evidence of a positive character verified by post-mortem examination.

CASE III.*—C. M., saleswoman, eighteen years old, came to my office for examination on the 14th of September, 1877. Percussion-note dull over lower part of left lung in front and up to the middle of interscapular space behind. There was bronchophony, bronchial breathing with subcrepitant râles. At the lower portion of the lung in front was *fine crepitus*. As the lung was consolidated by pneumonia no air could enter it, and consequently the râles must have been in the pleuræ. This evidence is further corroborated by the fact that since then the pneumonia has cleared up, resonance returning, but subcrepitant râles remain in place of the crepitant.

These three cases may be regarded not as unusual, but as typical, and they furnish proof: 1st, That mucous, 2d, that subcrepitant, 3d, that crepitant râles may all have their local origin within the pleuræ.

It is difficult to overcome preconceived opinions even with evidence perfectly conclusive to an unprejudiced mind. Still the facts, which I have given, and others which I shall further relate, must commend the subject to all candid observers.

Has the generally received opinion that all large, soft, moist râles are caused by bursting bubbles in the bronchi ever been put to the test of careful experiment? On the contrary, is it not the general experience that abundant mucous râles may exist without expectoration, and profuse expectoration without râles, or only with such as are distant from the ear, and which disappear upon expectoration? Has not the received opinion that all the râles of whatever size, liquidity or dryness, have their origin in the lungs,

* This case is of a class common to all practitioners, and is introduced as such. It has no novelty, but after the post-mortem evidence of Cases I. and II., the clinical evidence of consolidated lung becomes proof of the impossibility of the râles being interpulmonary.

and that the size of the bronchia determines the size of the râle, been adopted by pupil from teacher, from the time of Laënnec to the present, without regard to the obvious fact that the large râles are most frequently heard over portions of the chest where the bronchial tubes are very small, and the small râles where they are large?

Early auscultators explained the respiratory murmurs of health, as well as the rhonchi of disease, as being formed by the air passing through the bronchi into the vesicles and out again;—that the friction of this body of air in motion caused vesicular murmur and bronchial breathing; and that should mucus collect, it would be moved along bursting bubbles in its way —crepitant, subcrepitant and mucous, according to the size of the râle. Later, another theory was proposed, and by many adopted, which still regarded the size of the tube as governing the size of the râles. According to this theory, the tube being lined with adhesive mucus, collapsed after expiration, and the sides cohering, inspiration would again force them apart, causing râles. Neither theory recognizes obstruction to the free passage of air into the air-sacs and out again, yet the residual air certainly occupies the true respiratory system and does not admit air moving in a body. The tidal air physiologists estimate to be about one-tenth part of that in the lungs. So that after expiration there still remains nine-tenths, occupying the true respiratory system. This is the residual air. When inspiration again takes place the column of *in*-moving air passes in a body to about the third or fourth division of the bronchia, and can go no further, but mixes with the residual air, obeying the law of the diffusion of gases.

It is evident, in view of these facts, that both theories are impossible. The existence of so large a mass of residual air in the air-cells and smaller bronchial tubes, and also the existence of consolidated lung tissue (in which solid material fills the spaces previously occupied by the residual air), both show conclusively that all râles called crepitant and subcrepitant, when heard under these conditions are not intrapulmonary, and that mucous râles, when not clearly

traceable to the large bronchi, are also not intra-bronchial, and consequently all râles not clearly traceable to the larger air-passages are interpleural.

Mucus in the upper bronchia may cause *mucous râles*, which are intermittent. The mucus accumulates, the râles are heard; it is expectorated, and they are gone. In suffocative catarrh, and in approaching dissolution, the râles are continuous.

It would seem possible that fibroid lung could also produce subcrepitation in cases where the lung is adherent to the chest-wall. But of this I have no proof.

The following case of fibroid phthisis was characterized by a variety of râles and rhonchi, which would deceive any one who did not recognize them as signs of an interpleural pathological process. They were very suggestive of cavernulous phthisis and of disease of the heart.

Case IV.—W. S——, about sixty years of age, merchant, while in Scotland in 1874, had pneumonia, and since then had had frequent colds, causing short, spasmodic cough, with gradual increasing dyspnœa. He came under my care early in 1876. His breath was short and hurried. There were râles over both lungs; in some places coarse and rattling, and in others smaller, even fine crepitus. At the lower part of the right interscapular space, and below the scapula, they were coarse, moist, and gurgling. Under the right axilla down to the diaphragm there were creaking as well as dry râles. Under the left clavicle there were mucous gurgles, and under the left scapula there were fine crepitant and subcrepitant dry râles. There was general flatness under percussion, with raised pitch over most of the chest. There was an audible systolic murmur at the apex beat. The impulse was felt almost as high as the nipple, and there was also impulse in the second interspace.

The diagnosis was extensive plastic exudation between the pleural surfaces, forming adhesions which had drawn the lungs and heart upward. The dyspnœa and cardiac complications were the consequence of these changes, and there was no other serious lesion. At first he improved under treatment, and gained more than an inch in chest expansion, and was able

to get about with much less difficulty than before treatment, but in May an attack of pneumonia increased the amount of plastic exudation, and he lost more by subsequent contraction than he had previously gained in expansion. During the summer, in the country, he was under the immediate care of Dr. Ely, of Newburg. He was able at times to ride out, but renewed attacks of exudation lessened his vital capacity, and finally, after another "cold," he had increased disease, from which he died Oct. 8, 1876.

Autopsy by Prof. Delafield, Oct. 10th. Present, Drs. Jones, Dudley, G. A. Peters, Ely, and Leaming.—"Body much emaciated, cadaverous discoloration already evident on abdomen. *Pericardium* contains a little serum. Apex of heart on level with lower edge of fourth rib—distant three and one-fourth inches from median line. Upper border of heart on level with lower edge of first rib. Long axis of heart turned somewhat in vertical direction.

"*Lungs.*—Left side, very extensive old adhesions covering the entire lung. Left lung, upper lobe at the apex, some bands and patches of pigmental fibrous tissue. Lower lobe, lower third, bands of new fibrous tissue and red hepatization—the red hepatization is recent. Right lung, old adhesions over entire lung. Upper lobe, the same diffuse fibrous tissue, but more abundant.

"*Heart.*—Right ventricle contains large yellow post-mortem clot. Pulmonary valves a little thickened at their attached edges. Ventricle a little dilated, walls of normal thickness, tricuspid valve a little thickened.

"*Left ventricle* contains a small post-mortem clot. Cavity rather diminished. Walls normal thickness.

"*Aortic valves* somewhat atheromatous and stiffened, and on ventricular aspect of one leaf a small fibrous projection. Mitral valve a little thickened and atheromatous.

"*Kidneys.*—Normal size, capsule not adherent, surface smooth, cortex normal in appearance and thickness. Aorta markedly atheromatous."

(Signed) FRANCIS DELAFIELD.

In this instructive case the physical signs were interpreted during life to mean just what was found at the post-mortem examination.

It is needless to say that the ordinary interpretation would have made out a diagnosis of phthisis with cavities and structural disease of the heart as the cause of death. Is it not important that such a mistake should be avoided?

The first step in these complicated pathological changes was plastic exudation between the pleuræ, which, becoming organized, formed adhesions, and these in turn gave rise to all the subsequent diseased conditions in the lungs and of the heart.

Case V.—M. M., æt. 40, single. Saw her in consultation with Dr. E. D. Hudson, Jr., September 7, 1877, morning. Heart and great vessels gave no evidence of disease. Pulse and cardiac sounds were feeble and frequent, suggesting fatty degeneration. Chest expansion was not more than half an inch; respiratory murmur very faint; very little air entering the lungs. No disease of the lungs or pleuræ was discovered. But there was evident obstruction in the air-passages, the patient gasping for breath. Lung free from dulness. Highest local pitch in respiration traced to the larynx, and the obstruction was believed to be at this point.

The laryngoscope, in the skilled hands of Dr. Lefferts, proved this opinion to be erroneous.

Evening.—Consultation with Drs. Hudson and Lincoln. There was now found, at the summit of the left lung, perceptible dulness and flatness under percussion, and soft tearing râles in auscultation, conditions which had developed since morning. Respiration was more difficult, and during the examination became so great that unconsciousness resulted. No time was to be lost, and Dr. Lincoln performed tracheotomy, and the obstruction was found to be below the trachea. No evidence of aneurism was discovered. Dr. G. F. Shrady informs me that he refused to give this patient ether for an operation previously, because he suspected aneurism. Death occurred early on the morning of the 8th.

Autopsy, afternoon of the same day, by Dr. Hudson, in

the presence of Drs. Lefferts, Hitchcock, and Kemp.—"Cause of death, aneurism at the posterior surface of arch of aorta descending. Trachea and bronchia atrophied by the pressure of the tumor. Heart fatty, lungs reduced in volume, but normal otherwise. At the left apex the opposed pleural surfaces were agglutinated, the soft adhesions offering slight resistance in separating. Several older, organized but elastic adhesions spanned the left pleural cavity." (Notes of the autopsy kindly furnished by Dr. Hudson.)

This case is evidence that plastic exudation may be diagnosticated as soon as it takes place. There were neither râles nor dulness in the morning, but there were both in the evening, and fresh plastic material was found at the autopsy. Hasse says: "The first appearance of inflammation of the pleura consists in a congested state of its blood-vessels, which are seen congregated here and there, in dense, though delicate nets, beneath the still transparent membrane. At certain points the bright-red color deepens and becomes more equalized; these points are somewhat prominent, and, though scattered at first, presently crowd together and get encompassed with a progressively enlarging zone of gorged blood-vessels. At the same time patches and streaks are observed either darker than the rest, and not unlike little ecchymoses, or else of a pale red hue, as if from imbibition. The pleura now speedily loses its smoothness and polish, becoming dull and looking, as Laënnec expresses it, as if daubed over with a paint-brush. This redness gradually spreads until in most instances the whole, says 'Gendrin,' becomes uniform.

"The first rudiments of an adventitious membrane now become perceptible, the spots originally reddened, and that chiefly by repletion of the vessels, presenting little dull white or yellowish points which rise above the serous surface in the shape of flat granules, and ultimately coalesce." (Hasse's *Path. Anat.*, p. 183.)

All of the pathological changes described above, from the first congested blood-vessels in nets to the final covering of the whole pleura with lymph, produce the following signs: First, muffling; second, al-

terations of the respiratory murmur; and then, finally, râles and rhonchi, indicating exudation of plastic material. Every step of the pathological process is characterized by its appropriate physical signs.

Experience and a nice education of the ear make an early diagnosis easy and certain, and enable the practitioner to use remedies which, if employed in good season, remove the disability and the danger.

Case VI.—(Plastic signs removed by hygiene.)—F. J., about 26 years of age, while at business in Wall Street, in 1874, suddenly began to raise blood, and came immediately to my office. There was an area over the right scapula, where soft tearing râles could be heard, and there was also flatness under percussion. He was advised to take a walking expedition of two or three weeks' duration. This he did, and returned in health, not a vestige of the plastic râles remaining; nor has he had any return of chest signs or symptoms since.

It is possible that had he remained at his exhausting business under all the depressing influences which had produced their conditions, his lowered vitality would have been still farther depressed, and his case would have resulted in phthisis, as many others have done—so important is it to connect physical signs correctly with their true pathology.

Plastic exudation upon the pulmonary pleural surface has the immediate effect of obstructing the capillary circulation in that part of the true respiratory system which subtends the deposit. If it is not quickly reabsorbed, it becomes organized, and contracts, causing still greater obstruction. Hæmoptysis frequently results—it may be immediately, but in most cases not until after two or three weeks, or even longer.

The reason of this is evident, if we consider the minute anatomy of the circulation of the true respiratory system. The nutrient arteries of the lungs are derived principally from the bronchial, and differ from all others in the body, in the fact that they have no returning veins; no *venæ comites*. The nutrient capillaries after performing their special function, anastomose with the radicles of the pulmonary vein,

and their blood is reaërated even while performing its office, and hence, notwithstanding this apparent anomaly, arterial blood is alone forced into the left heart.

Consequently obstruction to the nutrient capillaries throws their blood back upon the bronchial arteries, which might seriously interfere with the circulation, except for a provision of nature, by which mucus is exuded copiously through the mucous membrane (bronchorrhœa), or perhaps blood (bronchorrhagia). So that either may be an important symptom of plastic exudation, and if carefully sought for, the plastic râles will be found.

Case VII.—G. B., a distinguished surgeon, April 1, 1876, had pneumonic sputa; pulse 100, temperature 100°. Had some oppression in breathing, but no pain. Auscultation discovered no râles on either side. True respiratory murmur was everywhere good, except over a part of the middle lobe of the right lung—a space about as large as the palm of the hand—where there were also perceptible dulness and raised pitch. Diagnosis: Centric pneumonia of the middle lobe of the right lung. The next day the pulse was 70 and the temperature 97°. Sputa the same as the day before, and so it remained on the 3d and on the 4th. Subsequently, the temperature was as as low as 93°.

On the night of the 5th of April he suffered great dyspnœa, and auscultation found abundant râles of crepitant and subcrepitant size, covering that part of the middle lobe of the right lung, posteriorly, over which true respiratory murmur was absent at the first examination on the 1st of April. The centric pneumonia had extended to the pleural surface, exudation had joined the pleuræ together, and crepitant râle and bronchial breathing were plainly heard. The dyspnœa from which he suffered was the consequence of these adhesions.

He gradually improved until the 27th of April, when he went to Fortress Monroe for change of air. Shortly afterwards he had a return of dyspnœa, and as it increased, he came home on the 5th of May. He now had moist tearing râles low down on the *left side.*

The heart was restrained in motion, and the first sound was altered in character. These signs indicated fresh plastic exudation in the left pleural cavity, as a result of which attachments had formed with the pericardial sac, and with the lung, altering the heart sounds and giving an intraventricular murmur at the apex. During the rest of the month of May and of June following, there was progressive plastic exudation, invading more and more of the pleura, and causing distressing dyspnœa.

From the first there had been albumen in the urine, with some casts. But in July there was notable improvement in all the symptoms; yet the râles remained, and exercise was exhausting. In the latter part of August he returned to the city and attended to some professional duties; was out riding daily, and visited the hospitals. But late in the autumn one chilling day, at the hospital, he took cold, and was again obliged to keep his room. There was another advance in plastic exudation in the left side; the heart was more restrained by tightening bands of adhesions; there was general and gradual failure in health, until the 6th of March, 1877, when he died.

Autopsy by Dr. Abbe.—"March 7, 1867—*Pleura:* Each cavity contained about a pint of clear serum.

"*Right lung*, bound by old plastic adhesions over pectoral and inframammary regions and to the pericardium; latterly over entire axillary region, and somewhat below, though not to the diaphragm. Posteriorly along spine up to the summit of the lung, where the apex was completely adherent.

"*Left lung.*—Apex adherent and firm, thence extending along the spine two-thirds downward to base of lung; also bound at upper part of subscapular region. Three or four fine bands of recent plastic extended from pericardium to left lung. The lungs were not diseased.

"*Liver* somewhat contracted and fatty. Gall-bladder contained perhaps a dozen small concretions not larger than mustard seeds.

"*Spleen* somewhat hard and fibrous, tightly adherent to extreme of left lobe of liver by old and thick

adhesions; also adherent to peritoneal wall, to the omentum, and to a little of the intestines.

"*Kidneys.*—Both somewhat contracted, the right much more than the left, weighing about three ounces; both somewhat cirrhotic and granular, and containing numerous small cysts, varying from the size of a small pea to that of a bean; both congested.

"*Heart* considerably enlarged; valves ample, but somewhat thickened (especially on the left, by atheromatous changes, fatty, etc.), beginning atheroma of the aorta, though without calcareous plates.

"*Intestines* and *bladder* normal. *Brain* not examined." (Signed) ROBERT ABBE, M.D.

About two years before his last illness, he had an attack of erysipelas, and at that time careful examination revealed no sign of kidney disease.

When first seen on that first day of April, 1876, there were no signs of chest disease, except slight dulness on percussion and the loss of true respiratory murmur over a space about three inches in diameter, over the back part of the middle portion of the right lung. There were no râles nor rhonchi until the night of the fifth, when subcrepitant and crepitant râles appeared exactly in the place where the loss of true respiratory murmur had first been observed. After this they were never absent, but gradually extended until they covered both lungs, becoming firmer and dryer as they grew older.

It would seem that lowered vitality had placed the capillaries of all the organs in a state of paresis and stasis, whence resulted plastic exudations—a general breaking down, in which all the vital organs were sufferers.

CASE VIII.—(Notes and autopsy by Dr. Stedman, of House Staff.)—"M. A. S., seamstress, admitted to St. Luke's Hospital, September 29, 1877. Has been feeling ill since last spring; has had cough; lost flesh and appetite. The patient is not complaining much of her chest, but comes to be treated for intermittent. She had a chill on the morning of admission.

"Oct. 10th.—Has had a chill every other day since admission. Examination of chest to-day shows that the right lung is free in movement in front without râles, but that there are some râles and signs of thickened pleura in the lower part of this side behind. Over the left lung there are plastic exudation râles, both in front and behind. Closer adhesions (fine dry râles) below. Soft râles are heard in the upper part, but they grow harsher downwards.

"Oct. 22d.—Patient has had no chill since the 11th. At nine o'clock this evening was seized with hæmoptysis and died from suffocation before any aid could be given.

"Oct. 24th.—*Autopsy.*—Right lung free from adhesions, except at lower part, behind. Left lung bound to the chest loosely above, more firmly below, both anteriorly and posteriorly. Lung filled with tubercles (caseous concretions), and two newly-formed cavities, one at the apex and the other at the middle of the upper lobe.

"Into this latter the hemorrhage had taken place from an eroded vessel the size of a crow-quill. The bronchial tubes and trachea were filled with blood. The pericardial and pulmonary pleura were firmly adherent."

The points of interest in this case are: 1. That the interpretation of râles as denoting an interpleural pathological process was correct.

2. That caseous deposits in small scattered masses may fill the lung without being detected when loose adhesions shut off sound, and especially when the true respiratory murmur is feeble or absent.

When the adhesions are firm and close, sound is more directly transmitted, and the pathological condition of the lung may be more easily diagnosticated.

3. Fatal hemorrhage nearly always takes place suddenly. A softened caseous deposit opens into a bronchus, and at the same time erodes a blood-vessel of some size, and the cavity and air-passages are immediately filled with blood, and the patient dies as by drowning.

Case IX.—Pietro Angelo, æt. 29, Italy, sailor, ad-

mitted to St. Luke's Hospital May 1, 1877. Had articular rheumatism, for which he was successfully treated with salicylic acid.

June 10th.—Was examined with the expectation of discovering heart lesions, but none were found; but there were signs of a cavity under the clavicle of the left side. Dry, crackling râles were found over the left side, and in the region of the heart there were a few râles synchronous with the heart-beat.

Diagnosis.—Cavity in clavicular region; old adhesions over whole of lung; also adhesions between the left lung and the pericardium. Right lung free. Patient says he has had cough for some time; complains of no pain, and did not think he had any disease of the chest.

June 20th.—Patient has had high temperature for a day or two. Examination shows abundant soft râles in left side, large and small, which have supplanted the dry râles synchronous with the heart's motion.

July 1st.—Patient is losing flesh, and has cough with purulent expectoration. A creaking sound is heard in the region of the heart, synchronous with the movements of the lungs and also with those of the heart.

Aug. 1st.—Patient failing; considerable expectoration, difficult breathing, hectic, and night-sweats.

Sept. 3d.—Patient complains of severe pain in the *right* side, with increased dyspnœa. Examination showed moist, tearing râles with each respiration over *right* lung, the one hitherto healthy. On the left side, in front, a harsh leathery creak is heard, but no râles synchronous with the heart's motion, although it is evidently restrained; behind, low down, there are numerous dry subcrepitant râles.

Sept. 14th.—Died at 5 P.M.

Post-mortem, Sept. 15th, seventeen hours after death. —"Right side of chest: adhesions over whole lung, attaching it to the chest-wall, but soft and easily separated by the finger. Left side: the lung is firmly adherent to the chest-wall and also to the *pericardial sac*, and could be separated from them only by dissection. There is a dry tubercular or caseous deposit in upper

part of right lung, and a good-sized cavity in upper part of the left."

(Signed) T. L. STEDMAN, M.D.

It will be seen that the soft adhesions easily detached in the right pleural cavity agree in age entirely with the appearance of moist râles of Sept. 3d. The evidence is decisive, for there was no disease of the lung nor of the bronchia to cause râle. The dry harsh râles of the left side also agree in physical conditions (firmly adherent, could only be separated by dissection) in age, with the time they had been under observation. In both sides the age of the adhesions was correctly diagnosticated by the physical signs. Another very interesting fact, and of practical importance, is brought clearly into the light, viz., that of diagnosticating adhesions between the pericardium and the lung, or between the pericardium and the mediastinum, by the sign of râles synchronous with the heart's motion. These signs are not uncommon, and are additional evidence of the interpleural origin of all râles. Cog-wheel respiration is due to adhesions between the lung and the pericardium. If the patient takes a full inspiration, the broncho-respiratory murmur will be interrupted by each beat of the heart during the inspiration, and also during the time while the breath is held. The motion of the heart bringing into sudden tension the adhesions, stops the respiratory sound for an instant at each beat. If the attention is fixed upon the recurrence of these interruptions it will sometimes be possible to analyze this short rhonchus, and to distinctly recognize that it is made up of fine crepitant râles. Occasionally it is heard to the right of the sternum near the cartilage of the sixth rib, and at the diastole of the heart, simulating aortic regurgitant murmur, except that it is not heard to the left of the sternum. In this position its crepitant quality may be very manifest. The adhesions are between the pericardium covering the right auricle and the right lung. When the pericardium is attached to the mediastinum, a systolic murmur of the heart may result. So that interpleural signs falsely interpreted lead to incorrect diagnosis as regards diseases both of the

heart and of the lungs. Many other cases are recorded which furnish equally strong proof of the correctness of the views here advocated.

The late Dr. Sprague, of Fordham, at the House of Rest for Consumptives, made about forty autopsies, in which the evidences were conclusive that the localities of râles were the sites of adhesions; that the localities of adhesions, unless so tight as to prevent all motion, were always the sites of râles. Dr. Sprague's eminent ability and painstaking assiduity render his observations of great value. I am fully persuaded that if those having opportunities will note the locality of râles for the purpose of verifying at autopsies the presence of adhesions, it will become impossible to doubt the mechanism of their interpleural production.

What diagnostic interpulmonary signs have we remaining, if all the râles and rhonchi hitherto considered as evidence of pneumonia, bronchitis, capillary bronchitis, œdema of the lung, tuberculosis, cavities, etc., are to be interpreted as of interpleural origin? Need we be anxious about the consistency of Nature? May we not leave all that to her, resting assured that as our knowledge is increased we will become more consistent observers, and see that she is always right? It is all-important for correct diagnosis, and in the treatment and management of disease, that the physical signs should indicate the pathological conditions. The very frequent mistake of treating bronchorrhœa for bronchitis, and ignoring the interpleural pathological cause, until the lung is irretrievably crippled, will be avoided. If we recognize the earliest signs of plastic exudation between the pleuræ we are enabled in all ordinary cases to promote its entire absorption. But if the favorable time is allowed to pass the exuded plastic material becomes organized, and even, if but of limited extent, may be from time to time the focus of renewed exudations, until the whole lung is bound to the chest-wall. Fibrous bands also extending through the pulmonary tissues contract, as they grow older, and finally result in the miserable conditions of fibroid phthisis.

Diseases of the lungs and bronchi are manifested

by their own signs, after excluding those which we have demonstrated to be interpleural. In so doing the gain is in greater accuracy in diagnosis, and in greater discrimination in the value of signs. The crepitant râle, although having its mechanism within the pleural cavity, is yet a valuable sign of pneumonia, or of phthisis, as it so often accompanies these diseases; but it is not pathognomonic. It may exist in the absence of both, and either may be present without crepitant râles. Centric disease, without cavities and without interpleural adhesions, is without râles or rhonchi. Yet there is an area of dulness and of absence of true respiratory murmur, exactly agreeing with the locality of the disease, which, with the rational signs of temperature, pulse, and sputa, render its detection sufficiently clear to avoid mistakes in treatment. Depending upon crepitant râles as pathognomonic has many times delayed prompt treatment, and has resulted perhaps in the loss of the patient. Convincing demonstrations alone changed my views as to their intrapulmonary mechanism. In pneumonia the exudation of plastic matter into the connective tissue of the true respiratory system is an early phenomenon. I formerly believed that the stiffened air-sacs, yielding reluctantly to the expansive force of inspiration, must separate the newly-exuded fibrine in the cellular tissue, thus giving rise to multitudinous râles.

Dr. Walshe once proposed the same theory, which has so many plausible facts to support it, but was obliged to modify his opinion, as I have since done mine.

He found that crepitant râles, in some cases, could be proved to be due to the presence of thin fluid in the pleural cavity (Walshe on *Diseases of the Chest*, pp. 107 and 108, 3d edition).

In Dr. Chamberlain's case of atheromatous aorta (reported in the *New York Med. Journal*, Oct., 1874) I had the privilege of making a careful exploration of the patient's chest not long after the first serious symptoms were manifested. Over the lower part of the right lung there was crepitus or fine subcrepitus, and at the autopsy blood was found in the right pleura, but both the pleura and lung were healthy.

The signs of bronchitis of greatest diagnostic importance are not râles, but raised temperature, quickened pulse, with harsh and sibilant respiration, which masks true respiratory murmur (it does not supplant it as is done in pneumonia), with appropriate rational signs.

When resolution takes place, then true mucous râles are heard in the upper bronchi, distant from the ear, and at longer or shorter intervals, as it is collected or expectorated. Bronchitis may be complicated with pneumonia or pleuritis, in which case the signs will be more or less blended.

Sympathy between the bronchia and the pleura is very intimate. Severe bronchitis is apt to induce plastic exudation between the pleuræ, and plastic exudation is accompanied more or less with bronchorrhœa. Foreign bodies in the bronchia induce plastic exudation between the pleuræ, even sooner than they do pneumonitis.

Capillary bronchitis may or may not be accompanied by râles; when so, they have their origin within the pleural cavity, and when there is no exudation there are no râles. This is a disease peculiar to children, and is really pneumonitis and has the same signs. That which is generally called capillary bronchitis, on account of the sign of small moist râle, is simply an interpleural plastic exudation, to which children are also very liable.

Fine subcrepitus may or may not accompany pulmonary œdema, but only when there is exudation of some kind within the pleuræ.

The only true sign of pulmonary œdema is dulness under percussion. It is not distinguishable from pleuritic effusion, except when there are fine subcrepitant râles as well, showing that the pleural surfaces are in coaptation and covered with lymph.

The diagnostic signs of interpleural pathological processes may be briefly stated thus: Physical signs —râles or rhonchi; large gurgling, soft tearing, harsh, dry, rattling, crackling, small, fine, creaking. Percussion note: flat, parchment-like, wooden, high pitch, dull. Rational signs: quickened pulse, hurried respiration, dyspnœa, asthma, short hacking cough

when the adhesions are over the summit and upper part of the lung; spasmodic and strangling when in the lower pleuræ. Bronchorrhœa, hæmoptysis, irritable stomach, dyspepsia, emaciation, loss of strength, frequent perspirations, especially when sleeping; and lastly, when advanced and extensive, all the signs peculiar to fibroid phthisis.