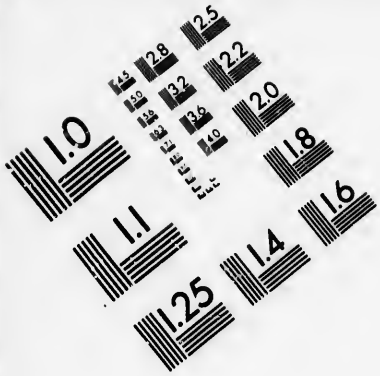

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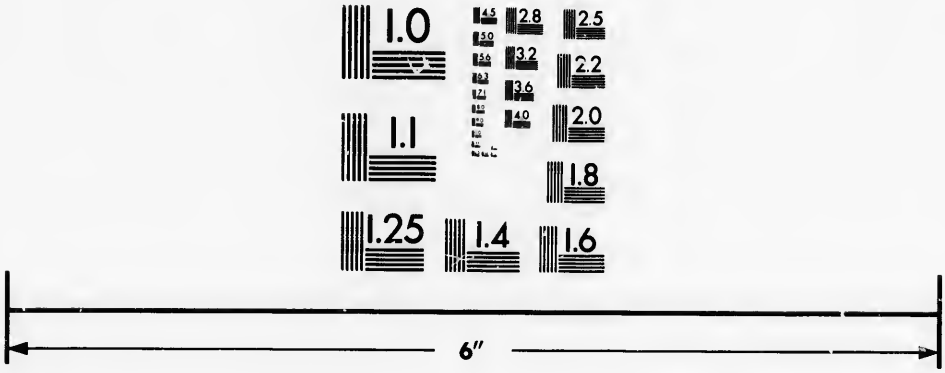

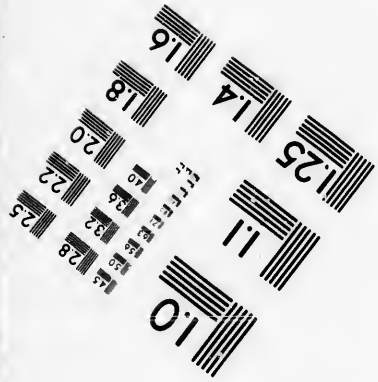

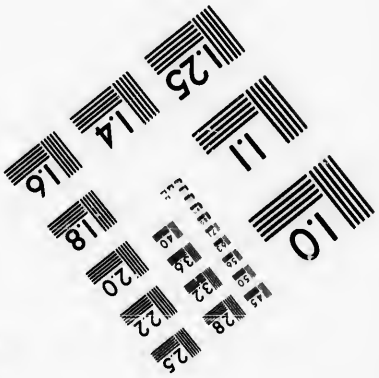

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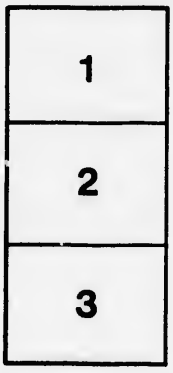

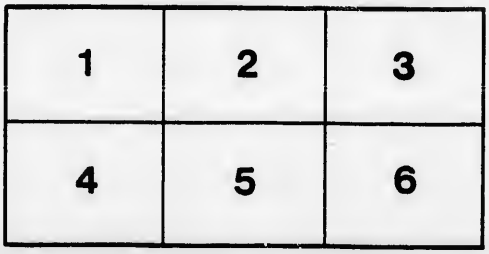

PNEUMOTHORAX, ITS ETIOLOGY, SYMPTOMS AND SIGNS WITH A STUDY OF TWELVE CASES.

BY

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Demonstrator in Clinical Medicine, McGill University; Assistant Physician Royal Victoria Hospital.

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The following cases, with but one exception, have been observed in the wards of the Royal Victoria Hospital, and form the sum total of such cases treated in that institution since its opening. I am greatly indebted to Drs. James Stewart and Bell for permission to study and report their cases.

CASE I.—H. M., male, aged 30, admitted May, 1897, re-admitted in August, again re-admitted Jan. 1898. He presented when first admitted marked pleural effusion of the left side. From the history given it was chronic and of undoubted tubercular origin. The onset was marked by severe pain in the side, with dyspnœa and, after several weeks, with an audible splash, first heard by the patient himself. Two years previous to this onset, his health had failed him somewhat, and he was the subject of cough with expectoration. The family history gave no evidence of tuberculosis. The patient never spat blood. The heart was much displaced to the right, the coin sound was absent and succussion was not made out. The amount of fluid present was so great that aspiration was urgently indicated and several ounces were withdrawn with considerable relief to the patient. After some weeks the splashing sound of which the patient previously complained returned.

The general condition of the patient was much improved for several months, and though the displaced heart returned partially to its normal position after aspiration it remained permanently to the right,

where it was doubtless held by pleuro-pericardial adhesions. The compressed lung never re-expanded, but occupied a small portion of the thorax posteriorly towards the apex and the middle line. Numerous aspirations were made and finally in Feb. 1898, a portion of the 8th rib was excised on the left side posteriorly, and large quantities of pus continued to discharge until the patient died, Aug. 29th, 26 months after the occurrence of this complication. All the physical signs together with a fairly characteristic onset of pneumothorax were present in this case, and the patient afforded a striking example of how pneumo-thorax may arrest the progress of pulmonary tuberculosis. This arrested state was inferred from the following facts. The quantity of expectoration was greatly diminished. There was a total absence of febrile movement for several months, except after the application of the tuberculin test. The patient during this period showed little tendency to emaciate. The case terminated with signs of sepsis and chronic diarrhœa, doubtless of tubercular origin. Clinically the opposite lung showed but few traces of disease.

Case II.—J. L., aged 17, male, was admitted March 20th, 1895, with left-sided pneumothorax of tubercular origin. The onset was sudden, with severe post-axillary pain in left side four months previous to admission, when he felt something giving way in his lung, followed by marked dyspnœa. This was more marked on exertion. His previous health had not been good. For nine months he had had a cough with loss of flesh and weakness. His condition on admission was afebrile, respirations and pulse slightly accelerated. Signs of left pneumothorax were manifest in prominence of left side, character of the percussion note, metallic tinkling, coin sound and succussion splash. The course of the case was favourable, the temperature variable, sometimes febrile, sometimes subnormal. He was discharged, improved, four and a half weeks after admission. Physical signs were not materially changed.

Case III.—K. F., female, aged 38, with left-sided pyopneumothorax. The onset was indefinite. It was not possible to fix the date. Her health had been failing for a year previous. She had spent considerable time in bed; she coughed and expectorated, but never spat blood. Shortness of breath, variable in degree, had existed several months, but had never been severe until two weeks before admission. Six months after onset of illness, hence about six months before admission, she complained of splashing sounds on the left side of her chest, which were observed over a period of about six weeks, then disappeared. She noticed the heart palpitate on the right side of chest. She presented on admission, well-marked signs of effusion into

the left pleura, with compression of the left lung, and displacement of the heart. After two aspirations of 45 oz. of pus in all hippocratic succussion was obtained, while the coin sound and metallic tinkling were absent. The third aspiration was performed nine days after, after which metallic tinkling, the coin sound and succussion splash were elicited. The course of the case in the hospital was afebrile with but one exception and patient showed marked improvement, doubtless due to the aspiration. Further operations were not performed, as the patient left the hospital and was lost sight of. In this patient downward displacement of the spleen was observed, while her case affords a typical example of latent pyopneumothorax.

CASE IV.—M. D. McM., aged 24, male, was admitted August, 1895, with pyopneumothorax of tubercular origin, left-sided. The onset had been sudden with almost total collapse. His previous health had been failing, marked by loss of flesh, cough, chills and fever, malaise and sweating at night. There had been slight expectoration of blood, Physical examination of the chest on admission showed cardiac displacement to the right; the left side of thorax was bulging, with a tympanitic note; the coin sound was present, but no succussion. Metallic tinkling on respiration was present and succussion was elicited after several weeks. It may be said, however, that the patient's condition did not justify the movements necessary to demonstrate the presence of succussion until several weeks after he was admitted. The pulse was constantly rapid, the temperature febrile during most of patient's stay in hospital and dyspnœa was a variable feature. The right lung showed signs of disease, manifest by localised pleuritic rub, and a few fine crepitant râles. The patient after nine weeks in the hospital accomplished a train journey of several hundred miles and lived for some months, fully seven months and a half after the onset of this complication.

CASE V.—K. H., aged 14, female, was admitted on 11th of January, 1896, with right-sided pneumothorax, of tubercular origin. The previous health had been poor as shown by weakness, cough, hoarseness, feverishness. The onset was rather sudden, characterised by severe pain in right side, but no marked shortness of breath. Condition on admission was as follows: Febrile temperature, rapid pulse, dyspnœa, signs of apical infiltration of the right lung with pneumothorax over the lower portion of the thorax, shown in cardiac displacement to the left, dulness beginning one inch to the left of the left edge of the sternum, distant amphoric breathing. Tympanitic resonance, faint respirations, metallic tinklings, the coin test, were observed but no succussion splash. Thirteen days after admission succussion splash was

elicited over the right side. The patient died five weeks after the onset of this complication manifestly with general tuberculous infection as both lungs showed tubercular disease. She was delirious and diarrhœa was a constant feature.

CASE VI.—K. N., female, aged 19, was admitted on Oct. 5th, 1898, with right-sided pneumothorax. The onset was sudden. The health had been failing, marked by loss of flesh, slight cough, tendency to take "cold," recent anæmia and weakness. One sister gave a history of tuberculosis. (More particularly; after a severe chill on awakening one morning and getting up, she returned to bed and slept for about two hours, to awaken again in profuse perspiration, with substernal pain, lasting for two days and then referred to her right side, with catchy breathing, amounting to dyspnœa).

Condition on admission; normal temperature, respirations 40, pulse, 100; with one exception the temperature remained normal throughout the following 18 days of stay in hospital. The pulse and respirations were not accelerated after the 4th day. Dyspnœa was not a marked feature. Attitude in bed was dorsal and lateral, without preference. The thoracic examination showed asymmetry, right side more prominent, hyper-resonance of the right side, weak respiratory sounds with moist râles of a peculiar metallic ring. Vocal resonance had a metallic ring also, the whispering was somewhat cavernous. Coin sound and succussion sound were absent. Cardiac displacement was marked, the mid-axillary line in the 7th interspace showing the apex pulsation. Dulness from above downwards began at the level of the nipple and transverse cardiac dulness began to the right of the mammillary line. Abdomen, right upper quadrant, showed evidence of displaced liver. The progress of the case was favourable throughout, dyspnœa diminished and a state of general wellbeing was experienced. The coin sound was elicited on the 8th of October, seven days after the probable onset, in a very limited area about one inch square just at the level of the seventh rib at the posterior axillary border.

Oct. 12th.—Four days later two other small areas were discovered giving this sign. One was found just below the angle of the scapula and was about the size of the bell of an ordinary stethoscope while the other about the same size was in the axillary space at the level of the 8th interspace.

Oct. 16th.—Yet four days later, the anterior and posterior areas above mentioned, *i. e.*, the first and third failed to give this note and the coin test was positive in one area only.

On Oct. 20th it was absent.

On Nov. 3rd it was still absent.

All metallic quality of voice and respiratory sounds have disappeared. The patient suffers no inconvenience except that due to over exertion and is now attending to light house work.

Signs of fluid were never found.

CASE VII.—J. P., aged 59, male, seen in consultation with Dr. H. S. Shaw, was one of pneumothorax of doubtful origin. Previously he enjoyed good health. On Nov. 27th, 1897, he fell and broke his leg. He remained under treatment in bed till Dec. 22nd, when some pain developed in right side of the chest. This pain passed away by 26th. On Jan. 1st, while still in bed severe right-sided pain developed and a few râles were heard over the area. On the 2nd a dyspnœic attack occurred and the signs of pneumothorax were manifest. The coin sound was obtainable widely over his right chest, weak distant amphoric breathing was present. The patient improved, signs of cardiac displacement disappeared, the coin sound also disappeared and was completely absent on 11th Feb., and March 1st patient was at work, and has been in good health ever since. The cause of this case is doubtful, and must remain so since there is no evidence either from the history or the physical signs pointing to tuberculosis, while the occurrence of such an event in one confined to one's bed by no exhausting illness inducing thrombosis or embolism, with subsequent localized pulmonary gangrene, renders a decision very difficult.

CASE VIII.—H., male, aged 19. The date of onset is doubtful. His previous health had been good. While walking upon the street four or five months previous to admission he experienced dyspnœa. It was not severe, he continued his journey to his place of work performing his duties that day. Some thoracic pain developed after a few days, but no distressed breathing to interfere with his work. About three weeks ago, about Oct. 1st, while pulling on a hoist, he experienced dyspnœa and subsequently pain in the left side. On admission he presented signs of pneumothorax on the left side shown in cardiac displacement to the right side, fulness of the left chest with obliteration of intercostal spaces, hyper-resonant note throughout, metallic echo of vocal resonance, and faintly heard coin sound. There was no succussion. He was afebrile, not dyspnœic. The course of the case has been uneventful except that it is thought the left chest shows less fulness, while the area of cardiac pulsation is less prominent toward the right, succussion splash has not developed, the coin-sound is very variable. It is best heard when tested in the left supra-clavicular area and the lower portion of axillary space. It transgresses the median line, passing to the right above the junction of the 1st and 2nd pieces of the sternum. It may be heard also from axilla posteriorly

over the lower part of the thorax but this is doubtful. The patient presents the type of latent pneumothorax according to the French authors since there is at least attennation of the pain and dyspnœa which so frequently usher in this condition.

The case is in all probability one of partial pneumothorax, for reasons which we may discuss later.

Case IX.—G., aged 19. This is the case to which reference will be made in discussing the etiology of pneumothorax, and will be given in sufficient detail there.

Case X.—M. L., male, aged 9. Pyopneumothorax, origin doubtful. Left pleura. He had been ill with signs of pulmonary disease, either of pleurisy or pneumonia or both, which set in with considerable pain and cough. On sitting up after two weeks in bed considerable thoracic pain and dyspnœa were experienced. The cause of his illness appears to have been empyema. There was no succussion nor coin-sound present. This patient was operated on, a resection of a portion of the 8th rib was done and drain introduced. The recovery was uneventful.

Case XI.—Female, E. H., aged 29. This patient was under treatment for pulmonary tuberculosis of several months duration. The disease was one of severe type and rapidly progressive. No signs of a complication with this condition were present, although signs of cavitation were described. The night before she died she complained of pain in the left lower axillary region. There were no other signs or symptoms recorded, denoting the presence of pneumothorax which was demonstrated by an autopsy and found to belong to the partial type, being situated about the upper lobe.

Case XII.—Male, aged 56. Left pyopneumothorax. The patient had been ill with pleurisy and empyema several months and had been frequently aspirated. One cannot fix the date of the pneumothorax. Hippocratic succussion and the coin-sound were both present. He died three weeks after operation, resection of a portion of a rib, and from the autopsy report it is evident that the original cause of the complication described was tuberculosis.

The Etiology of Pneumothorax.

Much interest has always gathered about the question of the etiology of the somewhat rare condition of pneumothorax which occurs according to good authorities in from 3 per cent. to 12 per cent. of all cases of pulmonary tuberculosis.

The anatomists of many years ago who taught that the arteries were air tubes, regarded the presence of air in the pleural sac as an

"exhalation" from the vessels of the pleura involved. Then also its occurrence was explained by the process of gangrene of the pleura or by a process of decomposition in the pleuritic exudate found so often in such cases. A third explanation had reference to the most common condition—that of *perforation*—which is now the only recognized immediate cause.

Lænnec, who first taught directly concerning the presence of air in the pleura, described the cases under three classes: (*a*) Simple or essential pneumothorax; (*b*) the presence of air or gas together with fluid effusion; (*c*) air or gas with fluid effusion and a fistulous opening communicating with the bronchi.

Thus two forms may be included under the division of non-perforative pneumothorax, while the other, form (*c*) is classified as the perforative variety.

Concerning the first form, essential pneumothorax, authors did but little more than hint at the possibility of the secretion of air or gas by the pleural sac. Walshe, in speaking of the development of tympanitic sound over a pneumonic area asks, whether the phenomenon can depend on temporary secretion of air by the pleural sac. In referring again to this point in another section of his work on Diseases of the Lungs, he says: "It seems admissible as a bare possibility . . . . tympanitic resonance may be caused by air secreted by the pleura." This teaching, never positive however, has long since been regarded as fallacious, and thus one form of Lænnec's classification has passed away. Such an origin for the gas is physiologically impossible.

It was contended by Jaccoud in 1864 that there was no evidence for either form of non-perforative pneumothorax, but such cases were most likely due to minute pleural perforations which soon closed and the air was absorbed. In this opinion many high authorities concurred, among whom may be mentioned the names of Fagge and Powell.

Quoting from the former author we find as follows: "Most writers have admitted that in exceptional cases gases may be found in the pleural cavity as the result of chemical decomposition of liquid effusion, and perhaps by direct secretion (or rather exhalation) from the lining membrane. Such notions, however, accord ill with the general doctrines that are now held by almost everyone—and as neither of these supposed causes of pneumothorax has in its favour the slightest clinical evidence, we may now, guided by the experience of more than half a century, reject them altogether, and assume that air is never found in the interior of the pleural space except as the result of a

breach in the continuity of its surface, placing it more or less in direct communication with the external atmosphere."

This seems scarcely the teaching of the present day, however, and guided by the observation of the last decade, we may assume that simple pneumothorax does exist, or at least, such a condition without perforation, and gases may be formed within the pleural cavity and give rise to all the signs characteristic of that condition. Bacteriological examination into such cases has established the presence of gas producing organisms of anaërobic type.

Three case reports may be cited supporting this view. The first is that reported two years ago by E. Lévy, of Strasbourg, in which no perforation was found, but the exudate contained on two occasions an anaërobic micro-organism capable of producing gas in cultures as well as in guinea-pig tests. From the clinical history of his case, it would seem that this infection was one of secondary character, and in presenting his report Dr. Lévy makes [illegible] or essential pneumothorax.

The second is that reported in ti[illegible]sches Archiv für Klinische Medicin, 61 Band. Dr. Richard May and Dr. Adolf Gebhart describe a case of pneumothorax of this class in which the gas formation was evidently due to the presence of the bacterium coli.

The third case is No. IX., in our series. A patient under treatment for a severe form of appendicitis, was operated on and four or five days later he developed signs of disease in the right lung and subsequently in the pericardium—signs leading to a diagnosis of pneumonia and pleurisy of the right side with hydropneumo pericardium. The autopsy confirmed the diagnosis and further revealed a pyo-hæmo-pneumothorax of the right side, as well as a pleurisy of the left side. The pericardial effusion was similar to that in the pleura, but the pus elements were more scanty.

An examination of the abdomen showed a suppurative track—a retroperitoneal dissecting abscess, extending upwards, traceable along the inferior vena cava to the diaphragm. Viewed from the pleural side no perforation was discoverable, but near that point where the inferior vena cava emerged through the diaphragm a reddish grumous broken-down area existed. The pericardium presented no such area suggesting even the possibility of perforation, although, as we have said, it contained ante-mortem and post-mortem signs of pneumo-pericardium.

Subsequent examination of the organs of this patient, as reported by Dr. A. G. Nicholls, in the *British Medical Journal*, of 1897, showed the presence of the bacillus aërogenes capsulatus in large numbers—and the gas found in the serous sacs was doubtless due to an infection with this gas producing bacillus.

Granting, however, that such cases of pneumothorax do exist, they are very rare, and each case suspected of being such must be carefully followed out in order to determine whether or not perforation is present, and if absent, what forms of organisms account for the formation of gases. Clinically such cases are scarcely within the range of precise diagnosis.

Pneumothorax occurs not infrequently in those apparently healthy, and many observers regard it possible that such may be really healthy. This observation gathers strength in the light of many such cases going on to recovery within a few weeks, leaving no sign of the cause of such a condition.

However, in such cases there is ample ground for doubt, and among those who oppose this teaching no one has done so more strongly nor more logically than Samuel West, who maintains upon the following grounds that in all probability a pulmonary lesion has given rise to a perforation under conditions of over-strain.

Perhaps all will admit the grounds upon which he reasons concerning this point. They are in substance as follows:

1. Lesions exist undetected clinically.

2. Recovery may be complete from pneumothorax, even though there be a tubercular process at the bottom.

3. The bursting power of a healthy lung is greater than could be exerted by any expiratory effort.

Doubtless many cases occurring in the apparently healthy are caused by tuberculosis.

Not only in the advanced cases with cavity formation may this complication occur, but an early case where small superficial caseating areas exist,—so small that they can not be discovered clinically—pneumothorax may develop while the subject of it is regarded as previously healthy.

Of interest under this division of the subject are the statistics of a large number of cases. Some years ago Biach collected from the records of 38 years the reports of 918 cases in three of the large hospitals in Vienna.

His table showed:

Tuberculosis in	715	cases,	76 per cent.
Gangrene of Lung	65	"	
Empyema	45	"	
Injury	32	"	
Bronchiectasis	10	"	
Lung Abscess	10	"	
Emphysema	7	"	
Necrotic Hæmorrhagic Infaret	4	"	Total, 888

Of the remaining 30 cases, 14 were undecided, while the other 16 were divided between thoracentesis, parasitic—peritoneal and intestinal origin—and carious changes in ribs and sternum.

Other observers show about 90 per cent. of cases due to tuberculosis.

In this group of 12 cases the causes are as follows: Tuberculosis, 5 cases; empyema, 2 cases; unknown, 4 cases; B. aërogenes capsulatus, 1 case.

Latent forms were found in (Nos. 3, 11, 8, 12) 4 cases.

Purulent fluid was demonstrated in 9 cases. No signs of fluid were found in 3 cases. Of the twelve cases, six are dead while two are at work, one under observation; one, a child running about; two others showed some improvement under treatment and were finally lost sight of. The left side was involved 8 times, the right side was involved 4 times.

There were 7 men, 3 women, and 2 children, 9 and 14 years of age, and the length of time varied from 2 days to 26 months.

The Diagnosis.

The diagnosis of pneumothorax is not always made clinically. Much stress has been laid upon the two *symptoms* which frequently mark the onset, viz., pain and dyspnœa, while in addition sometimes a sense of tearing or a crackling is realised in the chest. The dyspnœa may amount to orthopnœa,—the pulmonary insufficiency of Wintrich. These, however, may be absent or so slight as to pass unnoticed, as Pierre Angereau has recently pointed out, and the presence of air in the pleural sac may be discovered incidentally when making an examination in the usual way.

This author whose monograph has been published recently deals with the subject of such forms of pneumo-thorax which he terms *latent pneumothorax*. Among other conclusions he states:

1st, that total pneumothorax is attended by much pain and dyspnœa;

2nd, that in partial pneumothorax pain and dyspnœa may be slight or wanting;

3rd, that there exist forms of general pneumothorax absolutely *silent* in their symptoms without pain or dyspnœa.

Lévy is cited by Angereau as saying that partial pneumothorax in its insidious onset is generally in the advanced cases of tuberculosis.

As factors which may mitigate the severity of the symptoms pain and dyspnœa usually noticed on the onset, one may consider the size of the perforation; the directness or indirectness of it; the presence of adhesions, preventing sudden and complete collapse of the lung; the condition of the patient, whether weakened or not; the presence of fluid, already calling for considerable accommodation to this new condition, viz., the use of one lung.

The signs of pneumothorax are as follows:

(1.) Displacement of the heart and mediastinum. (to opposite side)

(2) Tympanitic resonance over the greater part of lung area, with feeble breathing and dulness at the base.

(3) Metallic sounds, including metallic tinkling and metallic echo—the *bruit d'airain.*

(4) Hippocratic succussion.

1. Displacement of the heart or mediastinum does not always occur it is true, yet so constant a sign is it that it is remarkable how recently only it has been described. Notwithstanding the writings of Lænnec and others on this subject, it remained to M. Gaide in 1828 to describe and lay special stress upon this as a point in diagnosis. One may readily suppose that in such cases where strong pericardial and pleural adhesions have been formed as well as in those where the opposite lung is consolidated, such a mediastinal displacement would not be found.

So quickly does this displacement take place that even before the first severe pain following the perforation is over, the heart cannot be found in its normal place.

I recall the remark made by the first patient whom I had under treatment with this condition. He was a very intelligent young man recently at Saranac Lake for phthisis. One day after dinner, while lying upon his bed, he was seized with a severe pain in his left side and experienced a faint feeling. "Believing," as he said, "it was heart failure, I put my hand in my bosom to feel my heart beat, but it could not be found in the right place." Shortly after, his physician found the apex of the heart in the region of the right nipple and every characteristic sign of pneumothorax well marked.

It would appear that such a change of position must arise from one of two possible causes, either increased intrathoracic pressure by which the heart and mediastinum are pushed over, or diminished traction of the elastic lung on the affected side, leaving the other in a normal state of elasticity unopposed. From the rapidity with which cardiac displacement takes place, as shown by clinical observation and experiment, it is pretty well established that increased intrathoracic pressure does not at first exist. Again the lung of the affected side loses its elasticity. Hence we may believe that the heart is pulled, rather than pushed toward the healthy lung.

2. The characteristic variety of percussion note is readily understood, but in this, considerable variation may be observed owing to the tension of chest wall. When pneumothorax occurs, in a very large percentage of those cases where life is prolonged, some form of fluid exudate developes in the pleura. Generally a purulent pleurisy is in-

dneed, and then the characteristic basic dulness is found, but only after considerable effusion has been poured out.

3. Metallic sounds are not characteristic of pyopneumothorax, since they are sometimes heard in other thoracic conditions. Yet they are very frequently associated with the condition, and even the coin sound or *bruit d'airain* may be heard over large smooth-walled cavities. Indeed, according to Osler's note recently published, this sign was present and a localized pneumothorax was strongly suspected. The autopsy revealed a large cavity, the walls of which were covered with "granulation tissue and presented here and there papillary projections which, on section, contained remnants of branches of the vessels and bronchi."

This sign is not always present, and it has been seen to vary from time to time in the same subject.

4. Hippocratic succussion is a conclusive sign, concerning the presence of air (gas) and liquid simultaneously within a cavity.

A question in the diagnosis of perforative pneumothorax often arises, whether one has a valvular or free opening, or if the case is seen after this condition has been present for some time, whether there be any communication remaining between the bronchus and the pleural sac. Some have urged that one can decide upon this question by observing the character of the breath sounds. If one hears the inspiratory murmur and the expiratory murmur one must conclude that the air enters and leaves the pleural cavity. It would appear. however, from the history of several cases that this is not reliable, Such auscultatory findings show at most, perhaps, that air enters the lung involved and doubtless the changed character of the respiratory murmur is a product of collapsed lung and resonating chamber,—the pleura sac.

Powell, of London, in his work on Diseases of the Lungs and Pleura, holds, on the contrary, that an amphoric respiratory murmur is diagnostic of a free opening and of special value on this point is the expiratory portion of the amphoric sound.

Conclusions.

1. There is such a form of pneumothorax as the *non-perforative form.*
2. Latent pneumothorax is not infrequent.
3. Occurring in the course of pulmonary tuberculosis pneumothomay have a retarding effect upon the disease.
4. Recovery of a total pneumothorax may take place without any sign of fluid.
5. In the advanced cases of pulmonary tuberculosis, or at any rate rate where the patients were regarded as the subjects of pulmonary tuberculous, fluid is present and the prognosis is grave.

TABLE OF PNEUMOTHORAX CASES.

No.	Sex.	Age.	Side.	Mode of Onset.	Previous Health.	Cause.	Fluid.	Treatment.	Duration.	Termination.	Remarks.
1	M	30	L	Abrupt	Failing	Tuberculosis..	Pus..	Aspiration, rib resection	26 months.	Death ... . Asthenia.	Latent at first. Improvement of general health.
2	M	17	L	Abrupt	Failing	Tuberculosis..	Pus...	Rest in bed, tonics	5½ months.	Lost sight of	Improving.
3	F	38	L	Indefinite	Pulmonary disease	Empyema ?...	Pus...	Rest, aspiration	8 months..	Lost sight of	Improved. Latent.
4	M	24	L	Very severe Abrupt	Pulmonary disease	Tuberculosis..	Pus...	Rest, tonics.	7½ months.	Death	In early stage.
5	F	14	R	Rather sudden	Very poor ..	Tuberculosis..	Pus...	Supporting .	5 weeks. .	Death	General infection. Both lungs. Delirium. Diarrhœa.
6	F	19	R	Rather abrupt, substernal pain	Recent anæmia, slight cough.....	Tuberculosis?	None .	Supporting, creosote ...	5 weeks...	Patient at work	Slight dyspnœa on exertion.
7	M	59	R	Onset sudden while in bed.	Good	?	None .	Aspiration of air, creosote	No signs at end of five weeks ...	In health...	Still at work, several months after.
8	M	19	L	Doubtful, pain and dyspnœa	Good	?	None .	Rest.....	6 months? 1 month ?.	Comfortable	When quiet. Latent and partial.
9	M	18	R	Sepsis, pleurisy, pneumonia ...	Septic peritonitis, acute	Bacillus aërogenes capsulatus	Serous sanguineous.	Palliative...	2 or 3 days	Death	Pneumothorax and pneumo pericardium *without perforation.*
10	M	9	L	Rather sudden	Two weeks illness	Empyema	Pus...	Rib resection	3 weeks...	Health	
11	F	29	L	Insidiously ...	Failing	Tuberculosis..	Purulent..	Of tuberculosis	Not known ..	Death	Discovered post-mortem. Localized at apex. Latent and partial.
12	M	56	L	Not known...	Ill one year.	Empyema	Purulent..	Aspiration..	Unknown.	Death