PNEUMOTHORAX MISTaken FOR HEART DISEASE.

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Heart disease is increasing in incidence, especially coronary sclerosis, and the symptoms of the latter are frequently mistaken for indigestion or gall-bladder dyspepsia with pain. It is important therefore to realise a reverse state of affairs, that a diagnosis of heart disease may be based on symptoms caused by a pneumothorax.

In particular, the sudden onset of spontaneous pneumothorax with its attendant dyspnoea, pain and collapse, tends to favour a diagnosis of coronary infarction. It is true that spontaneous pneumothorax is found mainly in younger subjects, but such are not immune to coronary vascular catastrophes. There have been two cases in my series in their thirties, and a few in their early forties; while one at the age of fourteen was due to progeria with generalised vascular degeneration.

When pneumothorax arises as a complication in a known tuberculous subject, the diagnosis is much less in doubt on account of the associated symptoms and signs; but we need to remember that the first symptoms in the diagnosis of phthisis may be those resulting from a rupture of the visceral pleura. It has been estimated that tuberculosis accounts for approximately 80 to 90 per cent. of all cases of pneumothorax, and that this condition arises in about 5 per cent. of all phthisical subjects.

Though pneumothorax results from other gross pulmonary lesions such as neoplasm, abscess or gangrene, the diagnosis for obvious reasons is not so likely to be confused with that of heart disease.

Possibly a haemorrhagic infarct or rupture of an emphysematous bleb giving rise to pneumothorax, might cause some confusion.

Some twenty-five years ago we were taught that a sudden pneumothorax in an apparently healthy subject was due to an otherwise symptomless tuberculosis, but we now know as the result of modern diagnostic requirements and the subsequent history of such cases, that the condition can arise in subjects free from any important pathological lesion.

It is in recurrent idiopathic pneumothorax that confusion with coronary disease more easily arises, for the attacks may develop at intervals from a few weeks to several years, during which either side of the chest may become involved.

Cases of sudden spontaneous pneumothorax more readily simulate coronary infarction; those with a slow onset present features more in common with angina of effort and benign cardiac pain. At least one quarter of the cases of spontaneous pneumothorax are of sudden onset, and such are accompanied by a severe pain in the side, stabbing in character, by dyspnoea which may be extreme, and a sensation of suffocation. Added to this may be collapse with pallor, rapid weak pulse, lowering of temperature, coldness and sweating.

On the other hand, an insidious onset is associated with pain or discomfort in the chest and dyspnoea, only on effort. This discomfort is sometimes described as constricting in character and accompanied by a fair degree of flatulence.

In comparing the symptoms of pneumothorax and heart disease I would emphasise the frequency of flatulence and of epigastric or low chest pains in coronary thrombosis; of the left inframammary pain experienced by the nervous, fatigued and frequently hypertensive subject; of the pain over the left sternal and the pulmonary areas in cases of effort syndrome; and, lastly, the possibility of purely right-sided symptoms in coronary occlusion.

The constricting pain of a pneumothorax when excited or aggravated by effort and relieved by resting may readily be mistaken for angina, and I need not discuss in detail the spread of chest pain to the neck, eye, shoulder joints, back and arms in cases of effort angina and coronary infarction, in contradistinction to the more localised chest pain in pneumothorax.

Patients with valvular disease of the heart or subject to paroxysmal tachycardia should be readily identified, but those with effort syndrome, coronary disease, or benign cardiac pain, not infrequently show few signs of local cardiac abnormality.

We have seen from the description above how similar may be the symptoms of certain heart disease and pneumothorax, and it would be as well to refer to signs of the latter malady.

Inspection. The appearance of shock and extreme dyspnoea is common also in cardiac catastrophes. The patient often assumes a semi-reclining position to obtain relief in breathing, sometimes leaning forwards in a sitting posture so often witnessed in badly compensated mitral disease.
One looks for displacement of the heart towards the sound side, and thus in right-sided pneumothorax a mistake of cardiac enlargement might be made.

If there be much rapidity of the respiratory movements and weakness of the heart's action, this displacement will be difficult to determine on inspection. The interspaces of the chest wall should be observed for evidence of obliteration on the affected side which would show a much diminished or an absence of respiratory excursion.

**Palpation** would confirm this and be useful in determining any displacement of heart and liver. The heart would not be displaced if bound down by adhesions. Tactile fremitus is absent over air spaces.

**Percussion.** The note is usually hyper-resonant but will convey a sense of dullness if the tension in the pleural cavity is high—as seen in valvular defects of the pleura when air passes into the pleural cavity without finding an exit. If fluid is also present, the patient should be examined in an upright posture in order to estimate the uppermost fluid level, observing that when he is lying on the back or on the sound side it is easy to miss effusion. One should also examine for shifting dullness.

Succussion of air is regarded as the most certain sign provided the patient can tolerate it. I have encountered it also in a case of large herniation of the stomach into the left pleural cavity where the signs were almost identical with those of pneumothorax.

Succusion must not be confused with ordinary stomach splash or pneumopericardium. When air and fluid co-exist in the pericardial sac, one hears “waves beating against the side of the boat” at each heart systole; and this sign is pathognomonic.

**Auscultation.** Breath sounds over a pneumothorax may be absent, distant, bronchial or amphoric. The last named are heard especially towards the root of the lung in the interscapular area.

Vocal resonance may be absent or diminished. Metallic tinkle is heard as a musical echo of the voice or breath sounds. The coin test is the most certain single sign next to succussion. It is important to try this in various areas of the chest, avoiding a rib which would conduct sound direct from the coin to the stethoscope.

**Partial Pneumothorax** is encountered in a large proportion of cases from all causes, and is due to adhesions of the lung to the chest wall or diaphragm.

It is in such conditions that a diagnosis may more readily be missed, as there is commonly an absence of abnormal findings either in front or in the back of the thorax; furthermore, the symptoms may be trivial or sufficiently obscure to warrant an assumption of morbus cordis—particularly in those rare cases of bilateral partial pneumothorax.

I have encountered the following cases, two of which were regarded as suffering from heart disease, and three with symptoms of heart distress confused the diagnosis.

**Case 1.**

A man of twenty-six was sent to me on account of symptoms referable to his heart. There was no significant family or past history. For three weeks he had experienced pain in the upper part of the chest, especially on the right side, after walking approximately a distance of 100 yards in a leisurely manner; but if he hurried the pain came on quickly and spread to the right shoulder joint and through to the right shoulder blade. If he ran upstairs he was obliged to rest, and he used to hold the affected side with his hand. He became easily tired and breathless on exertion.

After careful questioning he admitted a sudden onset of this pain when he thought he had strained a muscle in his chest; this made him feel ill and with the development of severer pain and breathlessness, he went to bed. Next day he got up and felt better, but was left with pain and dyspnoëa of effort, and some tightness across the front of the chest. In addition he experienced flatulence which improved as the pain lessened.

Examination of the heart revealed a forcible apex beat with a rate of 96 per minute lying, and slight displacement to the left of the mid-clavicular line in the upright position. The first sound was impure and the aortic second sound rather faint. No murmurs were detected. B.P. 160/90.

**Lungs:** There was diminished movement of the whole of the right chest, which was hyper-resonant to percussion. Breath sounds were diminished on the right side except where amphoric breathing could be heard in the interscapular area. The coin test proved negative. Vocal resonance was decreased and fremitus absent on the right.

Examination of the abdomen and nervous system revealed no evidence of disease. Urine: normal.

An electrocardiograph was taken and this showed tachycardia and regular rhythm.

In Lead 1 the P. wave was almost absent. R. was very small, S. deep and T. poor.

In Leads 2 and 3 there were no significant changes.
Screening the thorax decided the true nature of his complaint, a right-sided partial pneumothorax. He was referred to me again some months later. The pneumothorax had disappeared and his symptoms almost gone.

The electrocardiograph had changed.
Lead 1, a definite though small P. wave, a larger R. wave and a good T. Leads 2 and 3, the waves had considerably increased in amplitude.

The abnormal changes in the first electrocardiograph were due to the pneumothorax causing rotation in the heart's axis. Bourne and others have drawn attention to this phenomenon. Bourne (1940) states that there may be alteration in the picture simulating coronary occlusion with S–T deviation and T. wave abnormalities.
Remarks:

From the symptoms alone and particularly had they been left-sided, one needed to consider seriously a coronary arterial affection, giving rise to an angina of effort following an initial coronary infarction. I have encountered cases of true coronary infarction where the pain has been felt purely either in the right chest and right arm, or in the right shoulder joint. In this case the age and to a lesser extent the absence of pains or numbness in the upper limbs were features against a coronary occlusion; but they should not negative such diagnosis.

I have already referred to young people with coronary disease, and I have known a young man suffer from typical coronary symptoms during staphylococcal septicemia and due no doubt to a septic embolism in the coronary vessels, produced in the same manner as they occur in other organs such as the kidneys, liver and brain.

Case 2.

A man of thirty-two was seized with sudden dyspnoea and sensation of fainting with discomfort and pain in the chest while dressing one morning. I was asked to see him within a few hours of the onset. He was pale and slightly cyanosed on the lips and ears and finger nails, and had been sweating. There had evidently been some shock from which he was recovering. The pulse was of poor volume and the rate 120. Some dyspnoea was still in evidence. There was no pyrexia, no vomiting, but he had felt sick and complained also of flatulence.

The condition at first was thought to be due to a "heart attack."

I could not gather any history of important previous illness.

On examination the apex beat could not be found and there was a hyper-resonant note over the precordium. The heart was displaced to the right, and the sounds were distant. B.P. 112/80.

**Lungs:** Diminished mobility of the left chest. Percussion note hyper-resonant over the left lung, with absence of breath sounds in front and amphoric breathing in the interscapular area behind. The coin test was positive.

A diagnosis of pneumothorax was made. The patient improved from day to day, but became breathless on effort, with discomfort in the left sternal area. Screening his chest a few weeks later confirmed the diagnosis, but the pneumothorax had then nearly gone. There was no electrocardiogram.

Remarks:

The onset of dyspnoea, collapse, pain and discomfort in the chest with flatulence and sweating made one think of the possibility of a coronary thrombosis. The weakened heart sounds and low B.P. were suspicious factors.

It is again significant that the pain did not extend up into the neck, jaw, or arms, and that his age was below the average seen in coronary cases. I would emphasise, however, the similarity in his symptoms of discomfort and dyspnoea arising after effort.

Case 3.

A child of four years was recovering from pneumonia when he became suddenly seriously ill with pain in the chest, dyspnoea, pallor and cyanosis on the tenth day. After considering the possibility of sudden heart failure and pulmonary embolism, the doctor diagnosed a pneumothorax from the following clinical signs, hyper-resonance of the left chest with diminished mobility and a pronounced positive coin test. Amphoric breathing could be detected over the upper half of the left thorax behind, where there was dulness to percussion. The temperature was 103. Respiration 50. Pulse 130. He was admitted to hospital. A few days later fluid was detected in the pneumothorax, but was not evident on the X-ray film with the patient lying down, but obvious in the sitting posture. Aspiration revealed pus and air. Repeated aspirations were carried out, and eventually the child became fit enough for adequate continuous suction drainage, and he finally made a good recovery.

Remarks:

The sudden onset with respiratory distress and chest pains pointed to a catastrophe either in the heart or lungs. Pulmonary embolism seemed an unlikely diagnosis; acute heart failure was probable in view of the cyanosis, pallor and dyspnoea, particularly if associated with septic pericarditis. But the suddenness of the attack with pain favoured a lung condition involving the pleura. The development of an empyema would scarcely be so acute unless causing perforation of the pleura with pneumothorax, and this we believe took place in this child. It is held by some authorities that empyema commences in many cases as a cortical abscess of the lung which soon involves the pleura. Poynton many years ago referred to multiple cortical abscesses found post mortem in children after pneumonia, and I have known aspiration of a suspected empyema reveal the presence of 1 cc. of thick pus, followed next day by a large serous effusion, making it evident that the aspirating needle had punctured a superficial abscess of the lung, and produced further contamination and serous reaction of the pleural cavity.
It is interesting to observe the complete recovery in this case, knowing how badly pneumothorax behaves in response to surgical measures and open drainage.

Case 4.

A young man of thirty-two had been treated rather ardently by artificial pneumothorax for some ten years on account of a right-sided phthisis.

He told me that following collapse therapy he had experienced a tightness in the chest on several occasions and this had lasted for a couple of days. This discomfort was felt across the front of the chest soon after treatment, and he felt unduly fatigued and was breathless on exertion. It was easy in this case to recognise that the pneumothorax had caused the pain and dyspnoea; but increased difficulty arose the last time he subjected himself to this treatment, for he developed two days later much pain in front of the chest, rapid respirations, a temperature of 102 and a pulse rate of 120. With the knowledge of his treatment one expected and found signs of pneumothorax. Some dullness at the base of the affected side indicated a pleural effusion, but he assured me that this had happened before.

Heart symptoms were simulated by his dyspnoea, constricting pains in the chest and cyanosis. There was no distribution of the thoracic pains into the neck or arms. The blood pressure was low. He had felt sick and vomited. Definite rigors followed a period of feeling cold; then sweating set in.

The heart beat was forcible and displaced to the left. X-ray films showed pneumothorax and effusion in large amount. He had developed an empyema which was treated at first by aspirations of large amounts of pus, and later drainage by rib resection. He succumbed a week later.

Post-mortem examination revealed an almost complete absence of the right lung.

Remarks:

The symptoms of constriction must have been due to over-distension of the pleural cavity first by induction of air and later by the increasing effusion. Septic infection of this effusion gave rise to the signs of toxemia. This shows again how altered tension in the pleural cavity by air may simulate cardiac affection.

Case 5.

A male aged forty-two years was seized with severe pain in the precordial region while going to his work as a stoker. The pain radiated through to the region of the angle of the left scapula, also upwards into the root of the left neck; it was accompanied by a state of collapse and severe dyspnoea. The condition was considered to be due to a heart attack and he was admitted to hospital. Here he made the statement that the pain was worse on breathing and on coughing. The pulse was rapid. Temperature not raised. B.P. 120/80.

The physical signs were those of a left-sided pneumothorax; there was displacement of the heart to the right, hyper-resonance of percussion note over the left chest, absent or diminished breath sounds over most of the left chest. Râles and rhonchi were detected in the right side.

An X-ray film confirmed the diagnosis of an extensive pneumothorax. The electrocardiograph revealed no diagnostic features of coronary occlusion, though the P. and T. waves were markedly depressed in Lead I. He is now convalescing.

It was interesting to find such marked moist sounds in the right lung and this must be attributed to cardiac failure resulting from the large pneumothorax and mediastinal shift.

The case illustrates the similarity of cardiac symptoms with precordial pain radiating to the neck and through to the back in coronary infarction.

Summary.

Five cases of pneumothorax are described, each with different characteristics.

The symptoms simulated heart disease, either angina of effort, coronary thrombosis, benign cardiac pain, or acute heart failure.

It is recognised that the electrocardiograph can be altered by a pneumothorax and thus may add to difficulties in the diagnosis.

The main point of distinction in the symptoms of pneumothorax from coronary disease is that the pain does not spread to the jaw and arms, or usually to the neck. There is also a younger age incidence. The signs may also be confusing, especially in a partial pneumothorax, where, without the aid of X-rays, the diagnosis may become extremely difficult.

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