has the effect of momentarily suspending the choreic movements just as it arrests the attacks.

I have, perhaps, already too long detained you over a case the like of which you will doubtless very rarely meet in practice. But the clinical observer must not disdain the exceptionally abnormal cases which furnish him with a special mode of exercising his sagacity; besides, they must not be considered in the light of mere food for vain curiosity. Many a time, in fact, they will be found capable of furnishing to the pathologist the solution of difficult problems.

CLINICAL LECTURE

ON

A CASE OF PHTHISICAL PERFORATION OF THE PLEURA, WITH PNEUMOTHORAX.

BY GEORGE JOHNSON, M.D., F.R.S.,

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GENTLEMEN,—I have now (January 7th, 1878) under my care a case in which there is very conclusive evidence that a softened tuberculous deposit in one lung has caused perforation of the pleura, with resulting pneumothorax—that is, an escape of air into the cavity of the chest. The symptoms and the physical signs which occur in connection with this serious accident are very remarkable and very interesting; and I propose now to give you a brief history of the case, as recorded by my clinical clerk, Mr. Macdonald, with some practical and explanatory comments thereupon.

G. G., aged 26, an Italian stonemason employed at the new Law Courts, was admitted into Sambrooke Ward December 26th, 1877. He came to this country two months ago. For about six weeks, he has had cough, with expectoration of phlegm and occasional slight blood-spitting. Two days before his admission, he was suddenly seized with pain in the right side of the chest and great difficulty of breathing, which confined him at once to his bed. On admission (December 26th), the respirations were 54, pulse 140, temperature 105 deg. He complained of pain below the right nipple, extending through to the back. There was frequent short cough, with scanty muco-purulent expectoration. On inspection, it was seen that, while the left side of the chest moved freely, the right side was nearly motionless. The apex of the heart was seen and felt beating in the sixth interspace external to the left nipple. The liver was felt extending at least two inches below the margin of the ribs on the right side. On percussion, the whole right side of the chest was more resonant than the left, though the resonance had not a tympanitic character. Over the whole left side, there was loud puerile respiration; while on the right side the first impression was that respiratory sounds were entirely absent; but, on careful auscultation below the nipple, I discovered at each inspiration a distinct so-called "metallic tinkling"; and some of you will remember that I then expressed my opinion that a tuberculous deposit near the surface of the lung had softened, and perforated the pleura, so that, at each inspiration, air passed into the cavity of the pleura, giving rise to the condition called *pneumothorax*. The inspiratory metallic tinkling is caused by bubbles of air bursting through a small opening on the surface of the lung into the air-filled cavity of the pleura, and there giving rise to a resonant echo. The air enters the cavity of the pleura more freely than it passes out (no expiratory sound was heard). The consequence is, that the right side of the chest has become distended with air, which pushes the heart to the left, and the diaphragm and liver downwards.

There is yet another remarkable physical sign. When the patient speaks or coughs, the voice and the cough are heard to resound with what is called an amphoric echo: a similar echo to that which is heard when we speak with the mouth over the orifice of an empty decanter, or over the bung-hole of an empty—that is, only an air-containing—wooden cask.

The prognosis was very grave. The past history—in particular the hæmoptysis—renders it nearly certain that there is tuberculous disease of the lung; and now the right lung, being compressed by the air upon its surface, has collapsed, and is thus rendered useless; in addition to this, some pus and softened tuberculous matter has escaped into the cavity of the pleura, and acute pleurisy has been the result.

The right side of the chest was ordered to be covered by a linseed

poultice, and fifteen minims of liquor morphine to be given three times a day.

On December 31st, when I saw him for the second time, the metallic tinkling sound, or amphoric crepitation, as it is better called, was replaced by a loud inspiratory amphoric blowing, most distinctly heard below the right mamma in front, and behind in the right inter-scapular region. The amphoric echo of the voice and cough remained as before. The change from amphoric crepitation (metallic tinkling) to amphoric blowing is probably to be explained by the enlargement of the opening in the pleura permitting a fuller current of air to pass at each inspiration into the cavity of the pleura. The sound is exactly imitated by blowing sideways into a decanter, or into a large metal tube, such as a gun-barrel.

January 3rd. The physical signs on the right side remain the same, except that there is some dullness on percussion extending to near the angle of the scapula, indicating liquid effusion into the pleura. On the left side, near the angle of the scapula, a dry friction-sound is heard on inspiration. Inflammation has extended from the right to the left pleura, which is now roughened by lymph.

January 27th (the day on which this lecture was given). The auscultatory signs have again undergone a remarkable change. The amphoric blowing is no longer audible, and no respiratory sound of any kind is heard over the right side of the chest; while the amphoric echo of the cough and voice continue. What is the explanation of this change? The most probable interpretation of it is, that the orifice in the pulmonary pleura has been closed by a recent exudation of lymph, by which the passage of air from the lung into the cavity of the pleura is prevented. The temperature has ranged from 101 to 103 deg. until within the last two days, when, with increasing weakness, it has fallen to 98 deg.; the respirations, from 36 to 54; and the pulse, from 120 to 140. There has been a rapid decline of strength, and it is evident that the disease must soon terminate fatally.

Subsequent History, with a Report of the Inspection of the Body.—Death occurred on January 8th, within twenty-four hours after the preceding lecture was given: and the following is a condensed report of the inspection which was made by the Pathological Registrar, Dr. Barrow, thirty-three hours after death.

On opening the abdomen, the liver was found to be pushed down to within an inch of the umbilicus. The convexity of the diaphragm on the right side was on a level with the ninth rib, while on the left it extended as high as the fifth or sixth rib. The heart was displaced entirely to the left of the median line. On puncturing the right side of the chest, a quantity of air escaped. The right pleural cavity contained about six pints of greenish sero-purulent liquid. The right lung had completely collapsed; its entire surface was covered by a thick layer of recent lymph; and at its base were two masses of soft lymph, the larger one being as large as a kidney. When the lung was inflated by blowing air into the trachea, no air escaped from the surface until the soft lymph had been carefully scraped off, when a circular opening large enough to admit the blunt end of an ordinary probe was discovered at about the middle of the posterior surface of the lower lobe of the lung. Through this opening, after the removal of the recent exudation, air escaped freely when the lung was inflated. On incising the lung at that spot, the opening was found to communicate with a cavity about the size of a large walnut; the cavity being partly filled by a purulent secretion, and surrounded by condensed lung-tissue. The apex of the lung was consolidated, and contained several caseous masses; but in no other part had softening and excavation occurred. The posterior surface of the left lung was partly covered by a thin layer of recent lymph. The apex of the left lung was condensed, of a greyish colour, and having a shotty feel, with here and there a small mass of caseous deposit. No other noteworthy morbid appearance was discovered.

Subsequent Clinical Commentary on the Case.—It will be seen that, in this interesting case, the *post mortem* appearances corresponded very exactly with the physical signs observed during the various stages of the disease; and in particular my bedside interpretation of the replacement of the amphoric crepitation (metallic tinkling) by amphoric blowing, and the final cessation of the latter auscultatory sign, seem to be completely confirmed. The phenomena of this case are entirely in accordance with Fournet's doctrine that, in cases of pneumothorax from perforation of the pleura, the phenomena of metallic tinkling and amphoric blowing will cease if complete closure of the opening be effected. Without doubt, a crepitating echo (metallic tinkling) may occur in a closed pleura containing air and liquid, as a result of the agitation of the liquid by coughing or movement of the body (succussion), or the fall of drops of liquid from the upper to the lower parts of the pleural cavity; and perhaps even, as Dr. Walshe suggests, "the echo of rhonchi in the adjacent bronchial tubes may cause tinkling" (*On Dis-*

eases of the Lungs, 4th edition, p. 156). But, for the production of a true amphoric blowing, I am firmly persuaded that a free current of air into a large cavity is essential. In all the cases in which the phenomenon of amphoric blowing has come under my observation, this physical sign has been associated with one or other of the three following conditions: 1. A large cavity in the lung communicating with a bronchus; 2. Perforation of the lung, with an inspiratory current of air into the cavity of the pleura; 3. Pneumothorax, with a fistulous opening in the chest-wall, and a consequent inflow of air during inspiration. Some of you will probably remember a boy named Ratcliffe who was under my care in Sambrooke Ward in the summer of 1875. After being tapped for empyema, he had a fistulous opening in the chest-wall for several weeks, but ultimately recovered completely. While the fistulous opening with pneumothorax continued, we heard sometimes amphoric crepitation (metallic tinkling) caused by air-bubbles bursting through the fluid secretion of the wound into the air-containing cavity of the pleura; at other times, amphoric blowing, when a fuller current of air was inspired through the fistulous opening nearly or quite free from fluid. These phenomena could at any time be temporarily suspended when the fistulous orifice was closed by the pressure of a finger or a plug of lint.

In the great majority of cases of perforation of the pleura with pneumothorax, the diagnosis is attended with little or no difficulty. The physical signs which I have before referred to, occurring in a patient who has been suddenly seized with hurried and difficult breathing and pain in one side of the chest, are in the highest degree conclusive. In some cases, however, the physical signs are not so unequivocal as in that which I have here recorded. Some years since, I had in the hospital two cases in which there was a somewhat perplexing inconsistency of physical signs. In one case, with tympanitic resonance over the lower and back part of the chest, amphoric blowing and amphoric echo of the voice and cough, there was dulness on percussion over the upper and front part, with moist rattling sounds. This was explained after death by a pretty extensive adhesion of the upper lobe of a tuberculous lung to the chest-wall. As these adhesions between the pulmonary and the costal pleurae are the chief protection against perforation, so the forcible tearing asunder of an adhesion during a fit of coughing is the most frequent cause of the accident. The other case of pneumothorax to which I referred was one in which, with tympanitic resonance over the whole of one side, a distinct, though feeble, respiratory murmur could be heard down to the very base of the lung. On inspection, it was found that the centre of the base of the lung, over a surface about two inches in diameter, was firmly adherent to the diaphragm. This adhesion prevented the complete collapse of the lung, and the descent of the diaphragm in inspiration drew a limited amount of air into the lung.

The case which we have been considering affords an illustration of the fact that perforation of the pleura with resulting pneumothorax may occur in an early stage of phthisis. Many years ago, I saw, with my old friend the late Mr. Lavies, a case in which this fatal accident occurred at a yet earlier stage. An adult male prisoner in the Westminster House of Correction, having had a cough for a few weeks, was suddenly seized with pain in the right side and distressing rapidity of breathing. There was excessive resonance on percussion over the affected side, while the normal respiratory sound was replaced by a loud amphoric blowing. The diagnosis of perforation of the pleura with pneumothorax was obvious. The man died in a few days, and in the apex of the lung was a scanty caseous deposit, the only part that had undergone softening being a piece not larger than a pea immediately beneath the perforated pleura. In all cases, perforation of the pleura with pneumothorax is attended by the sudden onset of symptoms of more or less intensity. The collapse of the lung and the consequent sudden great diminution of the vital capacity of the chest always causes more or less urgent dyspnoea. The pain and the other symptoms associated with the onset of acute pleurisy are, I believe, mainly influenced by the amount of purulent secretion which escapes from the lung into the cavity of the pleura. When a pus-secreting cavity of considerable size opens into the pleura, the escape of its contents rapidly excites intense inflammation in the serous cavity; while, on the other hand, a perforation which does not communicate with a cavity is attended with little or no pleuritic inflammation. On looking back to some notes of cases seen when I was physician to the Public Dispensary, I find that, in the case of a man whom I saw in March 1847, although he lived for five days after the occurrence of perforation with pneumothorax, no liquid in the pleura or other evidence of pleurisy was found after death. In that case, a very small perforation had occurred at the apex of the left lung in contact with a piece of softening tubercle, but not communicating with a cavity; and this, as I believe, explains the absence of pleuritic complication.

In that case, there was absence of amphoric blowing, explained, as I then believed and noted, and as I still believe, by the extreme smallness of the aperture in the pleura. The diagnosis of perforation with pneumothorax was based upon the sudden onset of pain and dyspnoea in a phthisical subject, exaggerated percussion resonance over the left side, very feeble breath-sounds and crepitation, which was attended with amphoric echo, beneath the clavicle. From this, I inferred that the apex of the lung was partially adherent to the chest-wall; and this diagnosis was verified by the *post mortem* examination.

You are aware that when there is a mixture of air and liquid in the pleura, a sudden jerk (succussion) of the patient's body when he is standing or sitting causes a splashing sound, which is easily heard when the ear is applied over the affected side. This physical sign has been known to the profession since the time of Hippocrates. We did not seek for it in our patient's case. We had abundant diagnostic evidence without it; and the patient's feebleness and suffering were so great, that it was undesirable to add to his distress by the rather rough procedure which is requisite to develop the phenomena in question. An over-zealous auscultator is in some danger of forgetting that a minute and prolonged, and especially a rough, examination of the chest may be a source of fatigue, distress, and even danger to the patient. A short time since, a case of aneurism of the aorta came under my care in the hospital. After two or three days' stay in the ward, the patient discharged himself, and wrote me an impertinent letter complaining of the repeated and prolonged examinations to which he had been subjected. This man, although he had a very serious disease, was not suffering acutely either from pain or from weakness; and I consider that he had no sufficient cause for complaint against me, or against any of you who may have examined him in my absence. I refer to the case only to remind you of the need for so tempering your diagnostic zeal with discretion, that you may avoid giving just ground for complaint even to a captious and querulous patient, such as we sometimes though rarely meet with either in public or in private practice.

FEEDING VERSUS FASTING.

By J. A. CAMPBELL, M.D., Medical Superintendent, Garlands Asylum, Carlisle.

In the *JOURNAL* of February 2nd, the case of "the Market Harborough Fasting Girl" appears. An account of the symptoms of the malady, the treatment pursued, the result, and the *post mortem* appearances of the thoracic and abdominal viscera, are given. Now, when cases are published in the medical journals, it is understood that they may be made use of, if such be possible, discussed from all points of view, and fairly criticised. From an asylum physician's point of view, a short *résumé* of the salient points of this case, the treatment it received, and the result, a consideration of the subject of "fasting girls" generally, and some remarks on the subject of persistent refusal of food and its proper treatment, may not be out of place.

1. *The Market Harborough Case*.—Martha White had been healthy up to 1872; then she had an attack of real or feigned illness, during which she thought she could not swallow. An explanation of the mode of using, and a threat of the use of, the stomach-pump made her swallow. She recovered and went to work. In 1873, another attack is reported, which was considered principally, if not entirely, hysterical; "but, on this occasion, moral force was futile, and all we could do was of no avail". After this had gone on for some weeks, hypodermic injection of morphia and nutritive enemata were recommended, "as the girl seemed sinking for want of food". At this time, she took nothing but butter-candy, of which she sucked nearly one pound daily. In April 1874, the patient had convulsions; after which she took no food, and died on December 15th, 1877. No urine or fæces were supposed to be passed for twelve months. The account of the body and the viscera examined was what might be expected. The body was very emaciated; the organs are reported to have been free of organic disease, and the stomach and intestines from contents, except that, in the stomach, there was half an ounce of dirty mucus, and from four to six ounces of the same material in the small intestine, and a few lumps of hard fæces in the rectum. The urinary bladder is reported as atrophied.

In the remarks made on this case by Mr. Frederick Grant, he states that there was no difficulty of diagnosis; and that there can be no doubt that it was hysteria; and he calls attention to the singular point of the prolonged starvation.

2. *The Welsh Fasting Girl*.—In vol. ii of the *BRITISH MEDICAL JOURNAL* for 1869, at page 685, particulars concerning this case are to be found. It suffices for my purpose to say that, after much wonder was excited by reports of the case, four trained nurses were set to