A British Medical Association Lecture ON MASSIVE COLLAPSE OF THE LUNG (ACTIVE LOBAR COLLAPSE).

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BY

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THIS condition has been described under various titles, such as acute massive collapse of the lungs, post-operative massive atelectasis, post-operative pulmonary atelectasis, obstructive massive atelectasis, acute lobar collapse, apneumatoses, and so on.

The title "massive collapse of the lung" was used by Pasteur in his last communication on this subject. It is clear, self-explanatory, avoids the use, as Gairdner puts it, of *sesquipedalia verba*, and covers all deflations of a large area of lung, whatever the cause may be. The condition is found associated with pleural effusions, pneumothorax, bronchial obstructions by growth or foreign bodies, pneumonia, pulmonary tuberculosis, and other inflammatory conditions of the lungs, with certain nerve lesions, and as a complication following surgical operations and injuries. In some of these conditions the lung plays a passive part, but in the post-operative and traumatic cases the lung plays an active part. This was recognized by Pasteur when he used the subtitle "active lobar collapse," and my observations in this paper are limited, more or less, to that type.

ACTIVE LOBAR COLLAPSE.

I define this type as an airless condition of the lung, or part of the lung, of sudden onset, lobar in distribution, with coincident accumulation of tenacious mucus in the corresponding bronchi, and accompanied by characteristic physical signs, the chief of which is displacement of the heart towards the affected side.

The condition may be so slight as to pass unnoticed unless specially sought for, or it may be so extreme as to menace the life of the patient. It usually occurs within forty-eight hours of the operation or injury, but cases have been recorded after longer intervals.

In a typical case the onset is sudden; the patient is seized with a sharp pain in the side, has difficulty in breathing, and becomes more or less cyanosed. There is usually a moderate rise of temperature, and it generally precedes the onset; it is rarely above 102° F. There is also an increase in the rate of the pulse and respiration. The tendency is to lie on the affected side or, if pillowed up in bed, to curl up towards the affected side, which, on inspection, is seen to be immobilized and retracted, with narrowing of the rib interspaces. Further examination reveals a dullness corresponding to the area of lung collapsed, and over this area there is an absence of breath sounds and tactile vocal fremitus. The heart is displaced towards the affected side, and the apex beat may have shifted two or more inches; in cases where the left lower lobe is collapsed, it is also displaced upwards. Cough is slight and sputum scanty, or they may be absent. There is a compensatory emphysema of the unaffected lung. Later on, these physical signs present great variation; the chest wall is not completely immobilized; the breath sounds become tubular, and tactile vocal fremitus is increased. The cough increases, and there is expectoration of tenacious mucus, which, however, is never rusty. As time goes on the expectoration becomes more copious and less tenacious. Daily variation in the physical signs may occur, auscultation over one area of lung at different times giving quite different results.

The x-ray examination, an extension of the physical examination by inspection, and in the following cases made in the wards by a portable apparatus, clearly shows:

1. The site and extent of the lesion, as evidenced by a dense homogeneous shadow.

2. Displacement of the mediastinum and its contents towards the affected side, the heart shadow being merged with that of the airless lung in the lower half of the hemithorax, thus giving an abnormal density. If the right upper lobe is alone involved there may be only displacement of the trachea.

3. Reduction in width of the chest cavity on the affected side with narrowing of the rib interspaces.

4. Elevation and fixation of the leaflet of the diaphragm on the affected side.

5. Pendulum movements of the heart—seen in the later stages when the immobilization of the chest wall is incomplete.

6. Evidence of injury—for example, fractured ribs.

7. Compensatory emphysema of the unaffected lung.

The following selected cases illustrate most of these points.

CASE I.

Male, aged 19 years. A case of acute appendicitis. Appendicectomy at 2 a.m. on January 25th, 1928. Anaesthetic: ethyl chloride and ether. Drugs: before operation, atropine 1/100 grain; after operation, heroin 1/12 grain, repeated once.

At 5 p.m. on the same day he complained of a choking sensation; the temperature was 101.4°, pulse 100, and respirations 40. He had pain in the right side, and wanted to cough, but could not do so. He was cyanosed and had difficulty in breathing. The heart was displaced to the right, and the chest wall was fixed on the right side. There was dullness to percussion over the right base; the breath sounds were absent over this area, and vocal fremitus diminished. The x-ray examination revealed the typical appearances of massive collapse of the lower lobe of the right lung: (1) a dense shadow corresponding to the right lower lobe; (2) displacement of the heart and trachea to the right; (3) narrowing of the rib interspaces on the right side; (4) the line of the diaphragm could not be seen; (5) pendulum movements of the heart were not present; and (6) there was no x-ray evidence of injury.

There was little alteration in his physical signs during the second day, but he "felt easier." On the third day he was decidedly improving, and the x-ray examination demonstrated the pendulum movements of the heart. Some movement of the chest wall was taking place on the right side—breath sounds with some crepitations were heard low down over the dull area, and the sounds over other areas were bronchial in type. The temperature fell to 99.2° F. There was some cough and a little sputum. On the fourth day the x-ray examination showed a continued clearing of the affected lung and less displacement of the heart. On the fifth day a bronchoscope was passed and a quantity of mucopurulent secretion drawn off. He continued to improve, and on the twelfth day the x-ray appearances were practically normal.

CASE II.

A miner, aged 21, sustained an injury to his chest while working in the pit. He was taken home, and on the following day his doctor, finding that he had a temperature and signs of pneumonia of the left lower lobe, sent him to the infirmary. He was admitted to a medical ward. On admission his temperature was 101° F., pulse 108, and respirations 36. There was dyspnoea and cyanosis. Physical examination: marked dullness with increased V.R. and V.F., and bronchial breathing over the lower half of the left hemithorax. These signs varied from day to day. The heart was markedly displaced to the left, 2½ inches, the apex beat being also displaced upwards. He lay in bed curled up towards the left side; this was his most comfortable position.

The x-ray examination, on the tenth day of his illness, showed an almost complete obscuring of the lower two-thirds of the left hemithorax, with displacement of the heart and trachea to the left. The contour of the left leaflet of the diaphragm was not visible. On the right side there were fractures of the fourth, ninth, and tenth ribs.

The sputum was scanty, tough, and tenacious, but never rusty. He slowly improved; the highest temperature was 101.4°; it reached normal on the ninth day, but it was not until the twentieth day that the x-ray appearances were normal.

This was a post-traumatic contralateral massive collapse of the lung.

CASE III.

Female, aged 13 years. A case of appendicectomy with no complications. Anaesthetic: mixture and ether. Drugs: before operation, atropine 1/100 grain; after operation, heroin 1/12 grain.

The patient was doing well, but about fifty-eight hours after the operation the temperature rose to 102° F., respirations 120, and pulse 30. She suddenly developed pain in the right side, dyspnoea, and became cyanotic. In a few hours she was more comfortable. On the following day physical examination showed dullness at the right base with absence of breath sounds, V.R. and V.F. The heart was displaced to the right. There was slight cough and a small quantity of tenacious sputum. The temperature rose to 103°, respirations 140, and pulse 36.

The x-ray examination on the second day after operation showed all the typical appearances of a massive collapse of the middle

and lower lobes of the right lung, with displacement to the collapsed side of the heart and trachea. The narrowing of the rib interspaces was also well shown.

Progress was uneventful. By the eighth day after the operation the temperature had reached normal, and on the sixteenth day the chest presented normal radiographic appearances.

CASE IV.

Female, aged 15 years. Appendicitis—appendicectomy—no complication. Anaesthetic: ethyl chloride and ether. Drugs: before operation, atropine 1/100 grain; after operation, heroin 1/12 grain, repeated once.

Six days after the operation this patient had a sharp pain in the left side of the chest and some dyspnoea. The temperature was 97.6°. Physical examination: marked dullness over lower half of left chest, V.R. and V.F. increased. Movements of left chest diminished. Heart displaced to the left. On the following day she was much better, having coughed up some thick tenacious sputum.

The x-ray examination was not made until two days after the onset of chest symptoms. It showed some obscurity of the lower lobe of the left lung, with displacement of the heart to the same side. Pendulum movements of the heart during respiration were noted. Five days later the chest presented normal x-ray appearances. The temperature never rose above 99° F.

CASE V.

Female, aged 9 years. This little girl's illness began suddenly on the morning before admission to the medical wards. She woke with a headache, felt shivery, and vomited once. During the day she coughed more than usual, for she had been troubled by a recurring cough for some years, and complained of pain in the left side. In the evening she became delirious. Next day the doctor sent her into hospital.

On admission her temperature was 102.6° F., respirations 40, pulse 154; the sputum was scanty, greenish, not rusty, and contained Group IV pneumococci. Physical examination: left lung dull all over, but especially inferiorly, where the breath sounds were hardly audible; elsewhere breathing was audible, and posteriorly about the middle was bronchial in character.

There was a tendency for the temperature to settle by lysis, and on the ninth day of her illness it had fallen to 97° F.; on the tenth day it rose to 100.6° F., and on the following day to 102° F., after which it became subnormal, ranging from 96° to 98° F. during the rest of her illness. It was not until the twelfth day that an x-ray examination was made, and it revealed almost complete obscurity of the left hemithorax, with displacement of the heart and trachea to the same side. There was no elevation of the diaphragm on the left side, but it was fixed.

The x-ray examination was repeated from time to time, and the gradual return to the normal watched—about twenty-eight days after the onset of her illness. Fifteen days later, however, while still in hospital, a further x-ray examination revealed a patch of consolidation in the middle lobe of the right lung, which cleared up in the course of a few days. There was no rise of temperature, nor did she seem in any way affected by it. A week later the chest was apparently normal, and she went home in good health.

CASE VI.

Female, aged 13 years. Appendicectomy, no complications. Anaesthetic: mixture and ether. Drugs: before operation, atropine 1/100 grain; after operation, heroin 1/24 grain, repeated once.

On the day following the operation she developed a cough and brought up small quantities of muco-purulent sputum. There were numerous rhonchi over both lungs—most marked at the right apex. Both lungs were equally resonant to percussion. On the third day after the operation (about forty-eight hours) the temperature rose to 102° F. She was very breathless; pulse was 140 and respirations 120.

Physical examination: Areas of patchy dullness alternating with areas of normal resonance over the right lung—tubular breathing and whispering pectoriloquy noted. Later there was dullness over the whole of the right lung and almost complete suppression of breath sounds. The heart was displaced to the right side.

The x-ray examination showed complete obscurity of the right hemithorax, with displacement of the heart and trachea to the same side. The condition quickly cleared up, and by the fifteenth day was apparently normal.

CASE VII.

Male, aged 32. Acute gangrenous and perforated appendix. Appendicectomy and drainage. Anaesthetic: ethyl chloride and ether. Drugs: before operation, atropine 1/100 grain; after operation, heroin 1/12 grain.

On the evening of the sixth day after the operation the temperature rose to 102° F. There was slight dyspnoea and cyanosis—dullness at the left base and tubular breathing over

this area. The heart was displaced to the left, the apex beat being well outside the nipple line. The following morning the whole condition had practically cleared up, and the x-ray examination showed a little obscurity at the base of the left lung. He made an uninterrupted recovery.

CASE VIII.

Male, aged 8 years. Operation for repair of ventral hernia. Anaesthetic: ethyl chloride and ether. Drugs: before operation, atropine 1/100 grain; after operation, heroin 1/12 grain, repeated once.

On the day following the operation he developed chest symptoms. There was dullness and diminished breath sounds at the left base; the heart was displaced to the left, the apex beat half an inch outside the nipple line. There was marked cyanosis and breathlessness. The x-ray examination on the fourth day showed a typical massive collapse of the lower lobe of the left lung; the temperature had risen to 103° F. By the fourteenth day he had made a complete recovery, and I think was greatly benefited by the administration of CO₂.

COMMENTARY.

These cases illustrate the post-operative type, the post-traumatic type, and a type found in Case v which, I think, must be said to be associated with a pneumonic condition of the lung. They also illustrate the varying degrees of severity of the condition, so slight that it might easily have passed unnoticed, in others so severe as to cause doubts regarding the recovery of the patient. It will also be noted that in the post-operative cases the anaesthetic used was ether and that atropine was given before the operation, followed by heroin after the operation. In Case viii morphine was also given. In Cases ii and v no anaesthetic and no drugs of the nature of heroin and morphine had been administered. Most of the cases I have seen have occurred in young people, and the condition is a common accompaniment of lobar pneumonia in children. Cases have been recorded by Sewall associated with pulmonary tuberculosis, and Jacobaeus records similar cases; Jacobaeus also described its occurrence associated with pneumonia and with lipiodol injections of the bronchi. Others have recorded cases as occurring after slight injury, severe injury, and during infective processes. Regan records a case associated with acute poliomyelitis. The more one hunts for this condition, the oftener one finds it, and I cannot help feeling that Bowen is right in emphasizing the teaching of Sewall to "think atelectasis" when engaged in any phase of lung examination.

At this point it is necessary to differentiate clearly the condition under discussion—active lobar collapse—from massive collapse of the lung in cases of pleural effusion, pneumothorax, and bronchial obstruction by growth or foreign body. In these cases the collapse is comparatively a gradual process and the symptoms are rarely of a distressing character. Even in spontaneous pneumothorax the initial distress quickly passes off, and in complete obstruction by a foreign body the bronchus soon becomes tolerant, the collapse of the area of the lung supplied taking place slowly as the air is absorbed by the blood stream. In this process the lung plays a passive part. The density of the collapsed lung in these cases, if the collapse is complete, is just as great as it is in active lobar collapse. In a bronchial carcinoma it is impossible to separate out the shadow of the tumour from that of the collapsed lung, yet at the post-mortem examination the tumour was of relatively small size. Sante states that "the transparent structure of the lung collapsed in pneumothorax is difficult to understand and has never been successfully explained"; but in pneumothorax it is rare to find a completely collapsed lung. There is usually some air present, and if a portion of the lung is completely collapsed it gives a shadow just as dense as in active massive collapse. The conditions which Sante describes are not analogous. In the one air fills the pleural cavity and exerts an elastic pressure on the lung, seldom causing complete pulmonary collapse; in the other there is a complete deflation of the lung with a falling in of the ribs, elevation of the diaphragm, and displacement of the mediastinal contents to compensate for the space lost.

The name of William Pasteur of Middlesex Hospital must ever be associated with active lobar collapse, a clinical entity first clearly described him.

In 1890, writing on "Respiratory paralysis after diphtheria as a cause of pulmonary complications," he records his observations on fifteen cases of diphtheritic paralysis in which the muscles of ordinary respiration, especially the diaphragm, become more or less involved, with evidence of disturbed function of the lungs—"In other words there was evidence in the majority of cases of imperfect entrance of air into, and of some oedema of, the hinder parts of the lungs, more especially towards the bases, with condensation or consolidation of the organs in several of them."

In all these cases, with the exception of one, the condition was more or less bilateral. Regarding that one (Case vi) he writes: "I have little or no hesitation in attributing the physical signs to wholesale collapse of the right lung induced by the immobility of the right half of the chest, and the paralysis of the diaphragm."

In 1908 he delivered the Bradshaw Lecture, on the subject of massive collapse of the lung, using the term "massive" for the first time. In this lecture he refers to 64 cases of diphtheritic paralysis, in 28 of which the diaphragm was affected; 15 died, 12 recovered, and 1 was lost sight of. There were eight post-mortem examinations, and these revealed varying conditions from small patches of collapse on the posterior parts of both lungs to collapse of a whole lobe; in some of the cases a broncho-pneumonic condition with patchy areas of collapse in both lungs was recorded. He describes four cases of massive collapse of the lung found in the post-mortem room, one following laparotomy, the other three being medical cases associated with: (1) chronic ulcer of the stomach; (2) liver abscess; (3) hydrochloric acid poisoning with extensive damage to the oesophagus, stomach, and duodenum. He thinks that the extent and degree of collapse is as great in these cases as in those due to paralysis of the diaphragm, regarding it as caused by intense reflex inhibition of movements of respiration of sufficient intensity and duration to cause massive collapse of the lung, and suggests that some cases diagnosed as post-operative pneumonia are really massive collapse. He differentiates massive collapse, an active process in which the elastic property of the lung plays its part, from lobular collapse, a passive process of removal of air after plugging of a bronchus.

In 1910 he wrote on active lobar collapse of the lung after abdominal operations, and here is found the first full and complete account of the post-operative condition with its cardinal sign of cardiac displacement.

In 1914 his final paper was published under the title "Massive collapse of the lung" (*syn.* active lobar collapse). It is a clear statement of his work on this subject from the post-diphtheritic cases of 1890 to the post-operative cases which he correlated with them. He defines the condition as a

"total deflation (as opposed to lobular or patchy collapse) of a large area of lung tissue, of sudden onset—in the absence of any signs of obstruction of the airway or of any known cause of obstruction—due to failure of inspiratory power and attended by definite physical signs and symptoms."

He recognized two well-defined varieties: (1) cases in which the lesion is demonstrably due to paralysis of some of the essential muscles of respiration (diaphragm—intercostals); the collapse is usually bilateral; (2) cases in which collapse supervenes suddenly, without any premonitory signs of respiratory paralysis, and in which the lesion is apparently always unilateral.

He regarded failure of respiratory power as the essential cause of massive collapse, and post-operative collapse as being the result of (reflex) arrest of action of one-half of the diaphragm.

If Pasteur's definition of this condition be accepted so must also his theory, as the definition includes the theory, and it is for this reason that I, at the beginning of this paper, submitted another definition. Pasteur's theory might be called the theory of failure of inspiratory power, the lung playing an active part.

I have dealt somewhat fully with Pasteur's writings, first, because they are a striking example of a brilliant clinical investigation extending over a large number of years, in which the development of the author's ideas can be traced from the post-diphtheritic cases up to his classical description of the post-operative condition; secondly, because he is so often misquoted, and writers forget that he definitely regarded the lung as playing an active part, so much so that he uses as a synonym the title "active lobar collapse," although, in my opinion, he did not stress sufficiently the active part played by the lung.

In 1914, following Pasteur's paper, Elliott and Dingley published eleven cases; all followed abdominal operations—appendix, gall-bladder, gastro-enterostomy, and hernia. They admit that the bronchial muscles are strong enough to cut off the air entry to the alveoli, but think it most unlikely that they would be excited to such a reflex action locally and cause lobar

collapse, nor would the condition be established without a preceding phase of asthma. They advance the theory that the cause is the absorption of air where the bronchioles are obstructed owing to inflammatory oedema and secretion, stating that in all their cases there was the possibility of such local obstruction, for every patient had muco-purulent expectoration before or immediately after the onset of the collapse.

They failed to induce collapse in animals by: (1) division of the phrenic nerve on one side; (2) hemisection of the spinal cord at the second and third cervical segments—paralysis of the intercostals and diaphragm on one side.

In 1918 Crymble recorded fifteen cases following gunshot wounds, and suggested that the contralateral cases were due to the patient lying on that side.

In 1920 Rose Bradford, writing on this subject, stated that while it seemed incredible the evidence would seem to indicate that a reflex spasm of the bronchioles was the cause of the obstruction.

Charlton Briscoe in 1920 analysed the possible causes of massive collapse, and concluded that it was produced

"by the onset of inflammation affecting the muscles of the crus situated behind the peritoneum, one half of the diaphragm and its synergistic and antagonistic muscles are out of action owing to inflammation of the muscle or the pleural membrane covering it."

In 1921 Sewall of Denver drew attention to the frequency of atelectasis in cases of pulmonary tuberculosis, and this has recently been emphasized by Jacobaeus.

In 1924 Regan reported a case following poliomyelitis.

In 1925 Jackson and Lee analysed the diverse condition under which massive pulmonary collapse occurs, and showed two factors to be practically constant: (1) obstruction of the bronchial tree, followed by absorption by the pulmonary circulation of the air imprisoned in the alveolar air spaces distal to the obstruction; (2) some interference with respiratory movements—partial or complete—but always resulting in decreased aeration of the pulmonary tissue on the involved side.

Later in the same paper they speak of two of the factors underlying pulmonary collapse, either massive or partial—namely, inhibition or arrest of respiratory movements, and subsequent obstruction of the bronchi with accumulated bronchial secretion.

In 1927 Sante reported three unusual cases, one of which was bilateral; and another bilateral case was reported in the same year by Bergamini and Shephard. Sante concludes that

"the cause is not known, but it seems most probable that some infection or insult to the region of vagus supply produces a reflex action on the bronchioles, permitting their temporary collapse. Once approximated the walls of these bronchioles are held in apposition by adhesion, collapse of the lung rapidly following owing to absorption of the alveolar air by the circulating blood. Relief of the obstruction results in sudden re-inflation of the lung."

Mastics, Spittler, and McNamee write of it as a common post-operative complication, and regard the majority of post-operative pneumonias, bronchitis, and broncho-pneumonia as cases of atelectasis.

Scott and Joelson think the original cause is not yet proven, but regard the fundamental condition which initiates massive collapse as being a nervous reflex which affects both lungs and commonly occurs in a minor degree. They suggest that the posture of the patient determines the localization of the condition. This had been suggested previously by Crymble.

In 1928 Coryllos and Birnbaum came to the conclusion, from both the experimental and clinical work done by them, that the only cause is the complete occlusion of a bronchus by a plug of mucus acting as a foreign body, the other causes being predisposing factors.

In the present year they have published the results of further research work, and advance the mucus plug theory to explain not only massive collapse of the lung, but also pneumonia. They assert that in pneumonia it is the blocking of the lung air passages, bronchi, or bronchioles by plugs of thick sticky secretion which is the critical pathological factor. They regard massive lobar collapse as the condition from which post-operative pneumonia develops. They advocate the treatment of pneumonia, and, of course, of massive collapse by the inhalation of an oxygen mixture containing 5 per cent. carbon dioxide, and report several hundreds of cases treated in this way with excellent results.

In 1929 Bowen of Philadelphia published his admirable monograph on acute massive collapse. He deals with the subject in all its phases, and gives an exhaustive account of the literature with 220 references. He supports the theory of bronchial obstruction and advocates "bronchoscopic drainage, skilfully done" as an important method of treatment.

From the foregoing it is obvious that there are four main theories as to the cause of this collapse:

1. The theory of failure of respiratory power, the lung playing an active part. (Pasteur.)

2. The mucus plug theory. (Elliott and Dingley, Jackson and Lee, Coryllos and Birnbaum, Bowen, and many others.)

3. Reflex spasm of the muscles of the bronchioles. (Rose Bradford, Sante, and others.)

4. Inflammation of the crus and one half of the diaphragm or the pleural membrane covering it. (Charlton Briscoe.)

Pasteur's contention that the condition is brought about by paralysis of the diaphragm and other respiratory muscles explains the cases, which he first described, of respiratory paralysis after diphtheria in which he found an airless condition of the lower lobes of the lungs with some oedema, but it does not account for the massive collapse which we are discussing, in which the leaflet of the diaphragm is raised and fixed, but not paralysed. It is fixed in the expiratory position, which is the position of rest. A complete paralysis of one leaflet of the diaphragm is not followed by collapse of the lung, and Elliott and Dingley were unable experimentally to produce collapse by division of the phrenic nerve or hemisection of the spinal cord at the second and third cervical segments.

Briscoe's theory of retroperitoneal infection of the crus also fails, as too many cases have been recorded where there was no possibility of this taking place.

The theory of bronchial obstruction by mucus plugs is a very old one.

Gairdner in 1850 advanced his well-known ball valve theory to account for lobular collapse of the lung, and ridiculed the theory of Fuchs that the air imprisoned in the alveoli behind the obstruction was either received into the circulating blood or in some other way absorbed, as gaseous substances are when they are extravasated into the cellular tissue. He quotes the experiments of Mendelssohn and Traube, who introduced "leaden shot," balls of paper, and gum into bronchi in the dog, producing collapse of the corresponding portion of lung. He quotes the experiments of Reid, Longet, Schiff, and others on the effects of sections of the pneumogastric trunk, or of its visceral branches, on the lungs and bronchi. These experiments concurred in proving that these operations are followed by a very large accumulation of frothy mucus in the bronchi. The lung changes were imperfectly described. At the present day Gairdner's ball valve theory has been displaced by that of Fuchs's, which is ridiculed.

Lichtheim proved experimentally that areas of lung tissue distal to a plugged bronchus became atelectatic, if the circulation were not interfered with, but that no collapse occurred if the blood vessels to that part were tied.

Elliott and Dingley were the first of the modern writers to advance the theory of bronchial obstruction due to inflammatory oedema and secretion, and they have been followed by Jackson and Lee, Coryllos and Birnbaum, Bowen, and many others.

Now the accumulation of mucus in the bronchi is a slow process, the mucus becoming gradually tough and tenacious, and the absorption of air imprisoned in alveoli behind the obstruction is a slow process—the nitrogen is not readily absorbed; but the onset of massive collapse is sudden, often dramatically so.

The advocates of the mucus plug theory have to explain, first, why the mucus accumulates; secondly, why in a particular situation; and, thirdly, the formation of a plug large enough to cause a bilateral collapse, for such cases have been recorded.

The post-mortem examinations recorded by Pasteur and Rose Bradford did not reveal any gross bronchial obstruction such as would cause the collapse, and Rose Bradford further states that there is often no clinical evidence of bronchial obstruction.

The mucus theory by itself is untenable, but Jackson and Lee were careful to postulate two factors—namely, inhibition or arrest of respiratory movements, and subsequent obstruction of the bronchi with accumulated bronchial secretion.

The posture which patients tend to assume after certain operations or injuries would provide this inhibition or arrest of respiratory movements, and there is no doubt that experimentally the condition can be induced. This was accomplished by Mendelssohn and Traube in 1845-46,

and in our own time by Coryllos and Birnbaum, and by Lee, Ravdin, Tucker, and Pendergrass. These latter introduced into the bronchus of a dog a mucus plug removed from a man by the bronchoscope. Gum arabic solution was also used by them. They found that they could produce a typical collapse if they inhibited the cough reflex. There is thus no doubt that, experimentally, mucus, plus the inhibition of the respiratory function, will cause a collapse of the lung in dogs; but does this occur in active lobar collapse as we know it in man?

In many cases there is no question of inhibition of respiratory function by posture. One case recorded by Rose Bradford occurred as the result of a gunshot wound. No anaesthetic had been given. The wound was so slight that the patient had not been confined to bed prior to the onset of the collapse. There were many cases investigated by the same writer in which neither the clinical examination nor the post-mortem examination lent any support to the mucus plug theory. He further states that in these cases, seen during the war, the essential factor in the etiology was the wound, and that the condition might ensue in its most marked form, where other supposed factors, such as anaesthesia, posture, injury to the diaphragm or its nerves, exudates, secretion, or blood in the bronchial tubes, could be excluded. He was driven to the conclusion that the cause was a reflex stimulation of constrictor nerve fibres to the muscles of the bronchioles or some such process leading to their occlusion. In this view he is strongly supported by Sante, who states that

"all cases reported have followed either some toxic condition, such as diphtheria, poliomyelitis, streptococcus septicaemia, or some insult to the nervous supply of the vagus; abdominal or chest injuries or operations. Any one of these conditions could serve as the necessary reflex stimulus to the constrictor fibres of the bronchioles."

The post-mortem examinations are few, for nearly all the cases fortunately recover. Ritvo states that

"the involved portion of the lung is blue or violet in colour, and sharply demarcated from the normal lung tissue. It is soft, tough and wet; it feels heavy, does not crepitate, and sinks when placed in water. Microscopically the alveolar surfaces are closely approximated, the alveolar spaces being obliterated."

Any theory to be sustained must account for: (1) the accumulation of mucus in the bronchial tubes; (2) the rise of temperature often before the collapse occurs, or no rise of temperature; (3) the sudden onset of the major symptoms—often dramatically so; (4) the varying time after operations or injury of the sudden onset; (5) the occurrence of cases apart from anaesthetics, operations, or serious injury; (6) the occurrence of cases as a complication of inflammatory diseases in the lungs; (7) the contralateral cases; (8) the bilateral cases.

Any one of the theories previously mentioned can account for some of the cases, but none of them can account for all of them.

It is only when the lung is considered as a "muscular organ," to use Macklin's term, playing an active part, that the mechanism of the collapse is fully explained.

As far back as 1822 Reisseisen traced the muscle coat, by dissections done with a hand lens, down to the finer bronchioles, and inferred that it continued to the ends of the air tract. Baltisberger states that there is hardly a cubic millimetre of lung tissue which does not contain smooth muscle, and Macklin's work shows that the lungs are muscular organs in a very real sense. This has become increasingly obvious in recent years, the musculature being of great functional importance and indispensable in respiration.

The bronchial musculature can be regarded as a contractile net embracing the mucosa of the entire bronchial tree from the larynx to the alveolar mouths. Intermingled with the muscle coat is a loosely woven layer of elastic fibres, the whole being described by Macklin as "myo-elastic tissue," the elastic property ensuring evenness of action.

During inspiration the musculature is in a state ready for dilatation, and this dilatation is associated with lengthening and separation of the bronchi, the angles becoming smaller. The inspiratory act is passive so far as the lung is concerned, active as regards the chest wall and

the diaphragm. It is during expiration, which is a passive movement in the external muscles, an active movement in the lungs and bronchial tree, that the lung plays an active part; shortening and narrowing of the bronchial tubes takes place, the angles becoming wider. It is in all probability a wave-like motion, resembling peristalsis, travelling from the terminal bronchioles upwards. This movement was suggested by Gairdner, who in 1850 described it as the deobstruent action of the bronchial tubes. It is one of the factors in the movement of mucus upwards in the bronchial tubes.

That spasm does occur in the lung is evident in cases of asthma, and recently Jacobaeus has found that, in lipiodol injections of normal bronchi, massive collapse does occur. In hundreds of pathological cases in which lipiodol injections into the bronchi were made he never came across any massive collapse, but in cases where the bronchi were normal he found it of common occurrence. It was not the quantity of lipiodol, which was in some cases relatively little and could not possibly have caused complete occlusion of the bronchi, which caused the condition, and he could only explain the phenomenon as due to some contraction or spasm of the bronchi. The capacity for contraction or spasm in pathologically changed bronchi is greatly reduced.

There is no doubt that in many inflammatory diseases of the chest massive collapse is found, and the more one looks for it the more frequently one finds it. It has been found in pneumonia and in pulmonary tuberculosis, and it is an accompaniment of lobar pneumonia in childhood. Out of eight cases of lobar pneumonia recently investigated in young children, six presented the cardinal signs of massive collapse, and I have come to the conclusion that, while it is a common accompaniment of lobar pneumonia in children, it is not uncommon in young adults.

I am of opinion that the primary cause of active collapse of the lung is a reflex disturbance of the sympathetic and vagus innervation of the lungs, resulting first in a dilatation of the bronchi with cessation of the wave-like movement and a consequent accumulation of mucus. This I regard as due to an overacting sympathetic, and it is at this stage that the slight rise of temperature so often occurs. The balance between the sympathetic and vagus may be quickly restored and the condition rapidly clear up with the return of cough and the expectoration of mucus. On the other hand, as the overaction of the sympathetic passes off, the vagus may reassert itself too strongly and cause a sudden spasm of the musculature involving a whole lung or a lobe of the lung, or even, in rare cases, both lungs, for the lung is a muscular organ.

It is not necessary to conceive of the spasm persisting any great length of time for the condition to be established; it not only empties the lung tissue more or less of its air content, but when the walls of the alveoli and terminal bronchioles are brought into apposition, the molecular adhesion effectively prevents the re-entry of air. This accounts for the sudden onset and the varying time after operation or injury at which it occurs.

The chest wall and diaphragm accommodate themselves as far as they can to the altered conditions in the chest, but they cannot fully compensate for the smaller space occupied by the lung, consequently the mediastinal contents are drawn over to the affected side, the chief physical sign being displacement of the heart. It is during this stage that the patient has pain in the side and is cyanotic. As he becomes accustomed to the sudden alteration in the chest conditions the pain passes off, sometimes in a few hours; the cyanosis is of varying duration and persists until there is sufficient diversion of the blood to the sound lung to ensure adequate aeration.

Fortunately, the termination of these cases is almost always favourable. If untreated one finds in the course of a day or two that the patient is coughing up mucus, which, however, is never rusty; in other words, the wave-like movement of the bronchial tubes is at work again; the balance between the vagus and sympathetic has been restored. Gradually air re-enters the alveoli, never all at once, unless some powerful force has been applied as in forcible compression of the chest with sudden relaxation which causes the ribs to spring out, and in some cases induces the entry of air in much the same way as the first entry of air to the lungs in the newborn.

I agree that there are many predisposing causes, such as drugs which inhibit the cough reflex, post-operative postures which interfere with the free movement of the chest wall and diaphragm, and any condition in the lung itself which interferes with the nervous mechanism of its musculature, but the primary cause is a sympathetic and vagal disturbance.

Yet one question remains. Is there any analogous condition which might throw light on this subject? I think there is. Acute dilatation of the stomach is recorded as occurring under very similar conditions, accompanying or following operative procedures or associated with traumatism. Post-diphtheritic cases and cases associated with multiple peripheral neuritis have also been recorded. The onset is sudden, often dramatically so, and the gastric dilatation must be regarded as a primary, not a secondary, phenomenon. There is a sympathetic nerve disturbance, and in many ways massive collapse of the lung and acute dilatation of the stomach may be regarded as analogous conditions.

I feel sure that, in the examination of the chest, the more one keeps in mind the possibility of active lobar collapse the oftener it will be found, more especially in children and young adults.

SUMMARY.

1. Collapse of the lung, lobular and diffuse, was well known to the clinicians from the time of Laennec onwards, but William Pasteur was the first to give a clear account of the condition known as massive collapse of the lung (*syn.* active lobar collapse).
2. Active lobar collapse must be clearly differentiated from massive collapse of the lung of a passive character.
3. The function of the musculature of the bronchial tubes may be deranged by various causes acting on the sympathetic and vagal innervation either directly or reflexly.
4. The weight of evidence is in favour of the theory that spasm of the bronchial musculature is the essential feature of active lobar collapse, but the disturbance of the sympathetic innervation must also be taken into account.
5. In the examination of the chest the more one keeps in mind the possibility of active lobar collapse, the oftener it will be found, more especially in children and young adults.

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NOTE ON A CASE OF LIPOID NEPHROSIS.

PART I.—CLINICAL AND BIOCHEMICAL.

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THE term nephrosis was first suggested in 1905 by Friederick Müller¹; it is usually employed nowadays to distinguish a bilateral degenerative and mainly tubular diffuse renal lesion in contrast with nephritis, which is limited to diffuse inflammatory and proliferative kidney changes. Valuable contributions to the problem of nephrosis were made from 1913 onwards by Munk,² Volhard and Fahr,^{3,4} Epstein,⁵ Elwin,⁶ and others. In 1914 a special lipoid form of nephrosis was described by Volhard and Fahr, and has since received a good deal of attention, the following characteristics being defined.

Lipoid nephrosis has an insidious onset, but some think it may follow a comparatively mild or unnoticed attack of acute nephritis; its course is chronic; there is marked albuminuria, oliguria, decreased blood protein with relative globulin increase and reversal of the normal albumin-globulin ratio, lipaemia, hypercholesterolaemia, normal renal function tests, oedema, and no retention in the blood of nitrogenous waste products. In the urine are found casts and double refractive lipoid droplets, but no erythrocytes. The blood pressure is normal, and the basal metabolic rate is frequently below the normal. With regard to the basal metabolic rate, it should be noted that most determinations in the published cases were made when the patient was oedematous, and that the increase in body weight due to water retention tends in itself to lower the rate, which would have a higher reading were the patient oedema-free.

The main lesion in lipoid nephrosis is supposed to be a degenerative one chiefly affecting the renal tubules, but its occurrence is very rare. Very few necropsy findings of pure lipoid nephrosis have been recorded, partly because of the infrequency of the condition, and partly because the patients exhibiting this syndrome usually live for a considerable time, or die of intercurrent infections, or progress into chronic nephritis with glomerular as well as tubular changes. Thus, sometimes the clinical syndrome of nephrosis is present, but at necropsy the changes characteristic of nephritis are found as well as those of tubular degeneration.

Generally speaking it may be said that, while we have

no pathological classification of diffuse degeneration or inflammation of the kidneys which may be used clinically to predict accurately the nature of the kidney lesions, nevertheless it appears that the clinical syndrome of nephrosis does exist. It must be remembered also that the physiological unit of architecture of the kidney is in itself a complex structure, consisting of glomerulus, proximal convoluted tubule, loop of Henle, and distal convoluted tubule which empties into a collecting tubule; that this tubular system is lined by a layer of epithelial cells which are of different types in the different parts; and that between the afferent glomerular arteriole and the efferent tubular venule a double system of capillaries exists. It is reasonable to expect that if this complex secreting structure is injured in any part, its other parts may become secondarily affected, either functionally or structurally. It is thus readily realized that a diffuse renal lesion seldom follows a simple course, but that one type of lesion frequently merges into another. The view is gaining ground that, while the most obvious early kidney lesion in lipoid nephrosis is a degenerative one affecting the tubules, changes may be found also (and possibly at an early stage in the development of the disease) in the glomeruli; these glomerular changes at first may be slight, and may require special pathological methods for their demonstration. Lipoid nephrosis is regarded by some as a particular form, or development in the course, of Bright's disease, but others think that it may be primarily due to a special type of disturbance of the lipoid-protein metabolism. At any rate, its position as a *clinical* entity is doubtless established.

The work of Barker and Kirk⁷ is interesting in this connexion. These authors decreased the blood proteins in dogs by repeated bleedings, but returned the corpuscles to the circulation; when the serum protein was diminished to a certain degree by this procedure oedema appeared, and its amount seemed to be more closely related to the level of the serum albumin than to that of the total serum protein. At the time of the appearance of the oedema, albumin, fat droplets, and casts appeared in the urine, and the basal metabolic rate fell; pathological study of these dogs showed definite degeneration of the renal tubules, and later, with progression of the disorder, glomerular changes. In other words, the clinical picture of nephrosis was experimentally produced.

Christian's⁸ views are of importance. This investigator believes that the majority of cases of nephrosis are mixed cases in which, clinically and pathologically, there are evidences of both nephrosis and nephritis, and that as they progress they tend to lose the clinical evidences of nephrosis, and to gain the clinical features of nephritis. He seems to consider the protein loss through albuminuria of considerable significance, and points out the necessity of liberalizing the protein of the diet to replace the protein lost from the blood through the albuminuria. He believes that the source of the albuminuria is in the passage of albumin through the walls of blood vessels, most probably glomerular capillaries, because of functional changes. He considers nephrosis as simply a form of chronic nephritis, and thinks that the name should be used merely to describe a clinical syndrome, since it does not imply that a definite and single form of pathological lesion will be found in the kidney.

Report of Case.

A man, aged 34, was admitted to the Mater Misericordiae Hospital on January 10th. His weight was 151½ lb. and height 5 ft. 7 in. The previous history is irrelevant. From six months before admission he had oedema of the legs and face, and he first noticed oedema in the legs on the day after he had marched in a procession for several hours. On admission to the hospital the legs and lower part of the back were considerably oedematous, and the face was puffy and pasty pale. Albumin was abundant in the urine, which also showed hyaline and fatty casts and a few leucocytes, but no erythrocytes. The blood pressure was 170/100, and the pulse rate 70 per minute and regular; there was slight accentuation of the aortic sound at the base of the heart. X-ray examination by Dr. J. Geraghty revealed pyorrhocal absorption of the alveolar borders of most teeth present, but Professor Edward Sheridan stated that, owing to recession of the gums, there was almost complete absence of pockets, and consequently only a small area of infection at the time of examination. The nose, accessory sinuses, and throat were normal (Dr. P. Dempsey). There was dullness on percussion, and diminished breath sounds over both pulmonary bases. X-ray examination of the lungs by