A LECTURE

ON

VESICULAR EMPHYSEMA OF THE LUNGS.

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Definition—Anatomical Characters—Physical Signs—The Causes, the Pathology, and the Mechanism of the Disease—The Relation of Emphysema to Bronchitis—The General Symptoms and Results of Emphysema—Prognosis—Treatment.

LAENNEC applied the term Emphysema to two different conditions of lung. *Vesicular Emphysema* consists in dilatation of the air-cells. The walls of the cells often become ruptured, so that several contiguous cells unite to form a single cavity. This is a very common and very interesting form of disease. *Interlobular Emphysema* is characterised by an infiltration of air into the cellular tissue between the lobules; sometimes, too, beneath the pleura, and into the cellular tissue of the mediastinum and of the body generally. This is a comparatively infrequent and unimportant accident.

On the present occasion, I shall speak only of vesicular emphysema.

Emphysema is, as you know, Greek for *inflation*, and is correctly applied by surgeons to accidental inflation of the subcutaneous areolar tissue, as occasionally happens after a wound of the lung by a fractured rib, or during the operation of tracheotomy.

Inflation of the air-cells of the lungs is a normal condition; but, in the so-called vesicular emphysema, the cells are dilated; and Dr. Stokes uses the term "dilatation of the air-cells" to designate Laennec's vesicular emphysema. The disease consists essentially in dilatation of the air-cells and rupture of their walls.

Anatomical Characters.—The cells are much increased in size, and very irregularly so. The greater number, perhaps, equal the size of a millet-seed; while some attain the magnitude of a hemp-seed, a cherry-stone, a French bean, or even a still greater size. The larger cells are probably formed by the union of several, through rupture of their intermediate partitions. The larger cavities often form projections on the surface of the lung, when the chest is opened. The air does not readily escape from the dilated cells, partly perhaps on account of some obstruction in the bronchi which lead to them, partly from loss of elasticity of the walls of the dilated cells. When the lung is inflated by blowing into the trachea, the tissue round the projecting vesicles rises to their level, and the surface of the lung becomes smooth.

A good way of showing the dilatation of the vesicles is, as Laennec directs, to make thin sections of a lung which has been inflated and then dried. It may thus be seen that, not only are the cells dilated, but that many of their septa are more or less completely destroyed.

Emphysema may affect either a part or the whole of one or both lungs. It is either partial or general. The emphysematous dilatation is usually most advanced and conspicuous in those portions of the lungs which receive least uniform support from the walls of the chest and the surrounding viscera: thus, the apices of the lungs where they project above the clavicles; the anterior margins which lie behind the somewhat yielding cartilages of the ribs, and which are to some extent deprived of direct pressure by the intervention of the heart and great vessels. (Jenner, *Med.-Chir. Trans.*, vol. xi, p. 31.)

Again, the base of the left lung is less firmly supported by the stomach, than the corresponding part of the right lung by the liver; and, accordingly, Louis found that the lower lobe of the left lung was twice as often the seat of emphysema as the lower lobe of right; while the apex of the right lung was more frequently emphysematous than the apex of the left. (Jenner, *loc. cit.*, p. 32, note.)

When the whole or a great part of the lung is affected, the lung does not collapse as usual when the chest is opened. The organ appears to be enlarged, and almost to project beyond the margins of the divided ribs. When compressed, the lung does not crepitate like healthy lung, but as Laennec describes it, "the sensation conveyed by pressing the parts is very like that produced by handling a pillow of down." (Dr. Forbes's translation, p. 144.) Emphysematous lungs float with great buoyancy on water. They are usually more or less of a pale grey colour, and they have a peculiar dry feel. The pale colour and the dryness are a result of the small quantity of blood which they contain. The capillaries, many of them, are obliterated. The posterior parts of the lungs are sometimes engorged.

In fatal cases of emphysema, the anatomical signs of bronchitis are commonly found: Congestion and thickening of the mucous membrane of the bronchi; obstructed bronchi, with here and there collapsed lobules; dilated bronchi; and, more rarely, bronchial abscess.

The Microscopic Characters of Emphysematous Lungs.—Mr. Rainey has published (*Med.-Chir. Trans.*, vol. xxxi, p. 297) an interesting account, with illustrations, of the microscopic appearances in an emphysematous lung. He describes the pulmonary membrane which forms the walls of the vesicles as being perforated or cribriform; the perforations being well defined, of an oval or circular form, of various sizes, and more or less numerous, according to the stage of the disease. The membrane in other parts is studded over with oil-globules, either single or clustered; and Mr. Rainey believes that this oily degeneration precedes the perforation of the membrane. In an injected specimen, the capillaries are seen to be much attenuated, while the meshes of the capillary network are expanded and dilated. These changes in the capillaries are proportioned to the dilatation of the air-cells; and they appear to indicate that there has been a considerable stretching and expansion of the membrane, together with its capillary network.

The oily degeneration of the walls of the air-cells is not a constant phenomenon. I have repeatedly looked for it without finding it; and, not unfrequently, dilatation and rupture of the air-cells, with expansion and partial obliteration of the capillary network, are the only definite structural changes which are visible in an emphysematous lung. I agree with Dr. Waters (*Diseases of the Chest*), that "it is quite possible that the elasticity of the yellow fibres may become impaired, or even destroyed, without any structural alteration resulting, such as could be appreciated even with the highest powers of the microscope; and it is also equally possible that changes may occur in the blood-vessels, giving rise to malnutrition of the pulmonary tissue, yet that we may be unable to distinguish them."

Amongst the most constant of the *post mortem* appearances are dilatation of the right cavities of the heart, with hypertrophy of the walls. Less frequently there is some hypertrophy and dilatation on the left side.

Such being the anatomical characters of emphysema of the lungs, we may next consider the *Physical Signs* of the disease.

In cases of advanced and general emphysema, the whole chest appears enlarged. There is an unnatural bulging and prominence both in front and at the back. The chest becomes "barrel-shaped." There is an unusual fulness above and below the clavicles. A few months since, I was consulted about a lady, who was supposed to have a tumour at the root of the neck on one side. I found that the swelling was produced by the bulging of an emphysematous lung upwards into the neck above the clavicle. In some cases, the chest is irregularly and unsymmetrically prominent on one or both sides. The *semi-circular measurement* of one or of both sides of the chest is frequently increased. The disease, however, may be highly marked without such increase. This is especially common in persons beyond middle age, whose costal cartilages are ossified. The *movements* of the ribs are very limited. Their position is that which they ordinarily occupy at the end of a deep inspiration; and now, during even forced breathing, the whole chest rises in a mass, but there is very little movement of individual ribs. The breathing is chiefly diaphragmatic and abdominal. The lower part of the chest sometimes sinks in during inspiration. The *duration of the expiratory movement* is seen to exceed that of the *inspiratory*. This is a result of the diminished elasticity of the lung.

Percussion.—There is increase of resonance, an exaggerated pulmonary resonance, sometimes of a tympanitic character, but never so purely tympanitic as in cases of pneumothorax or distension of the stomach by air. The excessive resonance extends over the cardiac region; the heart being overlapped and pushed downwards by the enlarged lungs. The resonance extends, on both sides, lower than its normal limit owing to the pushing down of the diaphragm. And it extends beyond the middle line in front, if one lung only is affected, or one much more than the other. Exaggerated resonance is not constant; sometimes the arched form of the ribs renders them less yielding, and therefore less resonant on percussion, more especially in old persons when the cartilages are ossified.

Auscultation.—The chief auscultatory sign of emphysema is feebleness of the respiratory murmur. This is a result of limited expansion of the chest, in consequence of which the air enters the lungs with less force, and therefore with less noise. The respiration over certain parts of the chest is sometimes harsh and exaggerated; for the same reason that it is puerile over the sound lung when one is rendered more or less impervious to air. The *Rhythm* of the sounds is altered. The sound of inspiration is comparatively short; that of expiration is greatly prolonged. Very commonly when the expiratory sound is much prolonged, it is rather a sibilus that is heard than the natural sound of expiration. All the auscultatory signs of *bronchitis* are frequently heard, and the

fine crepitation of oedema of the lung. Laennec described a sound, which he called a *dry crepitant rhonchus with large bubbles*, which, he said, resembles the sound produced by blowing into a dried bladder. This he considered pathognomonic of vesicular and interlobular emphysema, but more distinct and constant in the latter. Some writers assert that they have heard such a sound on rare occasions. Others—and amongst them Sir Thomas Watson and Dr. Stokes—state that they cannot distinguish between this sound, if it really exists, and the crepitations which accompany bronchitis. It is probable, as Sir Thomas Watson suggests, that the sound in question “is really large crepitation in the neighbourhood of the dilated cells.”

Laennec himself says, “We have a sound like this in the common subcutaneous emphysema, on pressing interruptedly with the ear on the stethoscope, or with the fingers, on the affected part.” (P. 55.) This I have several times heard; it is like the fine crepitation of pneumonia; and is evidently produced by air being pressed through the moist meshes of areolar tissue; it is a *moist*, not a *dry* sound.

Laennec also describes a rubbing (*frottement*) of ascent and descent, which he believed to be a result of the friction of sub-pleural vesicles against the costal pleura in cases of interlobular emphysema. A friction-sound probably never occurs except as a result of pleurisy; in which disease Laennec overlooked it.

To recapitulate then. The chief physical signs of vesicular emphysema are: Local or general bulging of chest; diminished expansion; respiration chiefly abdominal. Increased resonance on percussion, not constant. Resonance extending over the whole of the cardiac region. Feeble respiratory murmur, not constant; prolonged expiration. Often the signs of catarrh and bronchitis. The *vital capacity* of the chest is small. An emphysematous patient can blow but little air into the spirometer. The heart beats in the epigastrium.

The Causes, the Pathology, and the Mechanism of Vesicular Emphysema.—Most writers on Emphysema have assumed that this condition of lung is a consequence of bronchitis; but they have differed in their explanation of its mechanism.

Laennec's theory was that, as a result of catarrh, there is plugging of some bronchi by secretion, and a consequent accumulation of imprisoned air, which, being expanded by the high temperature of the surrounding lung, causes dilatation of air cells. He erroneously supposed that inspiration is more powerful than expiration, and so conveys air beyond the obstruction, which expiration fails to expel.

Dr. Williams maintains the catarrhal basis of Laennec, but supposes that the air-cells communicating with the plugged bronchi escape distention, while the surrounding air-cells dilate, in consequence of the extra work and pressure thrown upon them.

Dr. Gairdner maintains that emphysema is a result of the pulmonary collapse, which he has shown to be a common consequence of bronchitis and obstruction of the bronchi. Dr. Gairdner's theory of emphysema is, that it “is an increase in volume of those portions of the lung to which the air has access, to supply the place of diminished volume in those parts from which it is excluded.” (*Brit. and For. Med. Chir. Rev.*, April 1853, p. 472.) He denies that the chest is enlarged in emphysema, and says “there is great reason to believe that it is usually smaller than natural; the arching of the front and the increase of the antero-posterior diameter being more than counteracted by a diminution in all the lateral diameters, particularly at the base of the chest.” (P. 470.) This denial of the enlargement of the chest is contrary to my own observations, and to those of most physicians who have directed attention to the subject. Dr. Gairdner's theory appears sufficient to explain the emphysema which occurs in the neighbourhood of lung collapsed after obstruction of the bronchi, or condensed and atrophied by a tubercular or an inflammatory deposit; but it affords no satisfactory explanation of cases of emphysema affecting the whole lung, and causing general enlargement of the chest. Moreover, in many cases of this kind, the history clearly shows that the emphysema has preceded the symptoms of bronchitis. Since my attention has been particularly directed to the relation between emphysema and bronchitis, a period now of about ten years, I have met with a considerable number of persons, both in hospital and in private practice, who, presenting all the physical and general signs of emphysema in a very marked degree, have had no bronchitic symptoms up to the time of their coming under my notice, or they have had these symptoms so recently as to preclude the theory that the bronchitis had caused the emphysema.

The fact that emphysema sometimes precedes the symptoms of catarrh has been noticed by several authors. Thus, Laennec states that “he has some reasons for believing that, in certain cases, the dilatation of the cells is the *primary* affection, and the catarrh *consecutive*.” (Forbes's translation, p. 148.) Louis affirms that, in many instances, catarrh has not shown itself until one or more years after the commencement of oppressed breathing and other symptoms of emphysema. (*Recherches sur*

l'Emphysème des Poumons, Mém. de la Soc. Méd. d'Observation, Paris, 1837, tome i.) And, more recently, Dr. Waters mentions, amongst other reasons for believing that emphysema depends on a primary degeneration of the lung-tissue, “the high degree of development which the disease often reaches without any previous history of violent or long standing cough.” (*Researches on Emphysema of the Lungs*, p. 34.)

I shall presently explain in what way emphysema acts as a powerful predisposing cause of bronchitis; but I must first indicate the chief circumstances under which the disease occurs. These are very various; but there is reason to believe that, in all cases of general emphysema, there has been an impaired nutrition of the lung-tissue, a loss of its normal elasticity and cohesive power, and a consequent expansion and rupture of the air-cells.

1. First, there is a class of cases in which the tendency to this disease appears to be inherited. Several members of the same family are found to suffer from the malady, and successive generations of the same family. When the disease is hereditary, it sometimes manifests itself at an early age—even as early as between the ages of 20 and 30—and the chest soon becomes much enlarged.

2. There are cases of what may be called *senile emphysema*. Advancing age brings on the disease in those who have no hereditary tendency to it. As the arterial and other tissues degenerate and decay with advancing years, so does the elastic tissue of the lung. In these cases of senile emphysema, there is usually no visible enlargement of the chest; for the cartilages of the ribs being ossified, the chest-walls are incapable of being much expanded. Emphysema, without enlargement of the lung and of the chest, is sometimes called *atrophic emphysema*; while the enlarged emphysematous lung is designated *hypertrophic*. But there is really no hypertrophy of the lung. We do not call a simply dilated bladder hypertrophied; neither do we speak of the uriniferous tubes of the kidney as being hypertrophied when they are dilated into cysts.

3. In another class of cases, the excessive use, or rather abuse, of alcoholic liquors has probably been the exciting cause of the disease. This, at any rate, seems in some instances to afford a sufficient explanation of that impaired nutrition of the pulmonary tissues, which is the immediate precursor of the dilatation and rupture of the air-cells. In some instances, we find that the emphysematous patient has had one or more attacks of gout; and it seems probable that the gouty dyscrasia may contribute to the malnutrition of the pulmonary tissue. (Dr. Greenhow.)

4. In a fourth class of cases, the emphysema has supervened upon repeated attacks of bronchitis. In such cases, it is probable that the nutrition and the elasticity of the pulmonary tissues have been impaired by the recurring bronchitic attacks, while the air-cells have been dilated and ruptured by the forcible efforts of coughing.

5. In another class of cases, valvular disease on the left side of the heart appears to have been the cause of the emphysema. The association of emphysema with disease of the mitral or aortic valves is so frequent, that it cannot be a mere casual coincidence. For several years past I have observed this association of emphysema of the lungs with valvular disease on the left side of the heart, and I have very frequently demonstrated it in the wards of the hospital. In these cases, we often find a history of rheumatic fever dating some years back; then, after a time, increasing shortness of breath and palpitation; and, on examination, the physical signs of emphysema, and of old valvular disease, are observed. In these cases, it is probable that the impaired nutrition and the loss of elasticity of the lung-tissue result from the impeded circulation consequent upon the defective valve.

6. There is a class of cases to which I desire to direct your especial attention—cases in which emphysema is associated with an excessive accumulation of fat in the subcutaneous tissue, in the abdomen, and about the heart. Many of these cases would come under one or other of the classes to which I have already referred. In not a few cases, the emphysema and the tendency to accumulate fat are hereditary; and in some the same tendency is a result of intemperance in eating and drinking. But these cases require a separate and particular mention, on account of their peculiar and distinctive features, and on account of the important influence of diet in the prevention and mitigation of their distressing symptoms. What, then, is the connexion between emphysema of the lungs and an excess of fat throughout the body? We find that, in a large proportion of cases, this excess of fat is associated with what Dr. Quain (*Medico-Chir. Trans.*, vol. xxxiii) has very appropriately designated “fatty growth” about the heart; and this fatty growth is always most abundant on the right side of the heart. The effect of this growth is to weaken the muscular walls of the right cavities. The result is, that the circulation through both the pulmonary and the bronchial vessels is impeded. There will be a diminished afflux of blood through the pulmonary artery, in proportion to the diminished contrac-

tile power of the right ventricle; and the backward pressure of blood from the distended right cavities will impede the return of blood from the bronchial, as from the other systemic veins. The impeded circulation leads to impaired nutrition and lessened elasticity of the pulmonary tissue, and thus the lung becomes emphysematous. An excess of fat, therefore, in particular a fatty growth on the right side of the heart, may be a primary cause of emphysema. Then emphysema, having reached a certain stage, undoubtedly increases the tendency to obesity, and that, too, in a very intelligible way—by impeding respiration, and so lessening the combustion of hydro-carbon. Exercise lessens the accumulation of fat in the system by increasing respiration and combustion. An emphysematous patient cannot take active exercise, because he is short-winded; and, whether he take exercise or not, the pulmonary circulation is so much impeded, and the vital capacity of the chest so far diminished, that the respiratory changes are always in arrear of the needs of the system. Unburned hydro-carbon, therefore, accumulates in the form of fat; and many of these unhappy patients carry about with them a great weight of material which would be very useful to the candle-maker, but which is simply an encumbrance to them. This great accumulation of fat, again, mechanically interferes with the movements of the chest and of the diaphragm, so that the respiratory changes and the pulmonary circulation are still further impeded, while the heart is embarrassed by the growth of fat upon its surface; and thus, by these mutual reactions, the evil grows rapidly worse.

7. There is yet another class of cases quite distinct from any of those to which I have before referred: I mean cases in which emphysema has supervened upon repeated attacks of spasmodic asthma. However purely spasmodic asthma may be at its commencement, there is a continual tendency to the development of structural changes in the lungs; and by far the most common of these changes is emphysema, with its usual catarrhal complications, and with hypertrophy and dilatation of the right cavities of the heart. The following I believe to be the explanation of the emphysema which results from spasmodic asthma. During the paroxysms of asthma, there is a partial apnoea, caused by spasmodic narrowing of the bronchial tubes. This partial apnoea involves, as its necessary correlative, a partial asphyxia. The movement of blood through the lungs is checked by the stopcock action of the minute pulmonary arteries in proportion to the degree in which the respiratory changes are limited by the bronchial spasm. The arrest of blood in the lungs causes engorgement of the right cavities of the heart, and of the whole systemic venous system. This retrograde engorgement, extending to the bronchial veins and capillaries, has for its invariable sequence mucous exudation from the bronchial mucous membrane. The earlier attacks of asthma leave no permanent structural changes in the lungs. With the cessation of the bronchial spasm, the freedom of the circulation is restored, and the lungs are left structurally sound; but, when the asthmatic paroxysms have been frequent, severe, and prolonged, the lungs become emphysematous. The frequently recurring spasmodic apnoea, with the consequent blood-stasis and engorgement of the bronchial veins and capillaries, causes an impaired nutrition of the pulmonary tissues, a loss of elasticity, and then dilatation of the air-cells.

It will be seen, then, that emphysema consequent on disease of the mitral or aortic valve, emphysema the result of fatty growth on the right side of the heart, and emphysema the result of spasmodic asthma, have this in common, that the impaired nutrition of the lung-tissue, and the consequent dilatation of the air-cells, are the result of impeded pulmonary and bronchial circulation; so that, while the cause of the impediment is different in each of these three classes of cases, the ultimate result is the same.

Some difference of opinion has existed upon the question whether the dilatation of the air-cells occurs during the act of inspiration, or during expiration. Most writers have insisted chiefly upon the inspiratory distension of the air-cells as the cause of their dilatation; but Sir William Jenner (*Med.-Chir. Trans.*, vol. xl) has directed attention to the influence of violent expiratory efforts—such as occur in coughing, etc.—in distending and dilating the cells, particularly in those portions of the lung which do not receive uniform support from the walls of the chest—namely, the apices and the anterior margins of both lungs, and the base of the left lung. The air-cells in these portions of lung are not unfrequently dilated and torn suddenly during the violent respiratory movements which occur when the entrance and egress of air are impeded, as in cases of croup, diphtheria, and whooping-cough.

To sum up, then, the immediate physical causes of vesicular emphysema:

1. It may be a result of increased strain and pressure upon air-cells which are previously healthy, as in the cases just now referred to, in which acute emphysema occurs during the progress of diseases which

involve a great impediment to the entrance and exit of air. Violent and prolonged gymnastic exertion is an occasional cause.

2. It is a result of over-distension acting upon cells in the immediate neighbourhood of portions of lung collapsed during the course of bronchitis, or condensed and contracted by a tuberculous or an inflammatory deposit.

3. Lastly, when emphysema comes on slowly, and affects the greater part of both lungs, it is a result of impaired nutrition and diminished elasticity of the pulmonary tissue.

The Relation of Emphysema to Bronchitis.—A local and a very partial emphysema may result from lobular collapse of the lung, which is one of the accidents of bronchitis; a more general emphysema may be induced by repeated attacks of bronchitis, as I have already explained; but whence comes it that general emphysema, when not preceded and caused by bronchitis, is associated with a continual liability to catarrh and bronchitis? I look upon it as certain, that this tendency to bronchitis is a consequence of the emphysema. I had arrived at this conclusion from a careful observation of the order in which the various phenomena of the disease occur, before I saw any explanation of their relation to each other; and my opinion is now confirmed by what I consider to be the rational interpretation of well-ascertained facts. We have seen that one of the chief anatomical features of emphysema is the obliteration of a considerable portion of the capillary network of the lung; hence arises an impeded pulmonary circulation, and hypertrophy with dilatation of the right side of the heart. The systemic veins are always more or less distended; and the bronchial veins, which are tributary to the systemic veins, are partakers in this distension. The bronchial veins and capillaries, therefore, being in a constant state of passive engorgement, there is a continual tendency to serous transudation through the bronchial mucous membrane, which is manifested by the wheezing sound audible on a deep inspiration; and a catarrh from exposure to cold, which, occurring to an individual with sound lungs, would be only a slight and transient ailment, is apt to become a severe and chronic bronchial inflammation. The impeded circulation through an emphysematous lung, acting backwards through the right side of the heart and the vena cava upon the bronchial veins, predisposes to bronchitis, and prolongs and aggravates it when it occurs, just as varicose veins in the legs act as a predisposing cause of inflammation of the skin, and often render its cure difficult. This, then, is the explanation of the fact that general emphysema of the lungs is almost constantly associated with a tendency to winter-cough and bronchitis. This explanation of the relationship between emphysema and bronchitis receives additional confirmation from the fact before referred to and accounted for; namely, the invariable sequence of bronchial catarrh upon every paroxysm of spasmodic asthma; the catarrh being, as a rule, profuse and prolonged in proportion to the duration and the intensity of the bronchial spasm and the consequent blood-stasis.

It can scarcely be doubted that attacks of bronchitis tend to increase the emphysema, partly by causing a yet greater impairment of the nutrition of the lung, and partly by the forcible distension and tearing of the air-cells during the paroxysms of coughing and dyspnoea.

The Symptoms of Emphysema.—*Dyspnoea* is the most frequent. At first it is slight, and felt only on unusual exertion, such as walking fast or running up stairs. It gradually increases, and is subject to great variations of intensity. It is liable to occasional aggravations through bronchial spasm, flatulent distension of the stomach and bowels, and intercurrent attacks of bronchitis.

With the dyspnoea, there is often more or less *palpitation*, and a painful sense of distension at the bottom of the sternum. We have before seen that, in consequence of the impeded pulmonary circulation, the right cavities of the heart become distended and dilated, and their muscular walls hypertrophied. The more or less extensive obliteration of pulmonary capillaries in an emphysematous lung impedes the passage of blood from the right side of the heart as effectually as a constricted pulmonary artery or a diseased valve. The circulation through the coronary veins, too, must be impeded by the distension of the right cavities; and this impeded circulation must be a source of cardiac weakness and distress.

Cough, as a symptom, ranks next in frequency and importance to dyspnoea. It is not an essential symptom of emphysema alone; but it is a constant attendant on the catarrhal and bronchitic complications of emphysema.

Expectoration, more or less abundant, and of variable character, is associated with the cough during the bronchitic periods. Some patients expectorate the semi-transparent pearly material which is characteristic of Laennec's "dry catarrh".

Hæmoptysis is a more frequent symptom than is generally supposed. Its occurrence often excites a suspicion of phthisis; but it is notorious that patients who are in a marked degree emphysematous rarely be-

come tuberculous. The blood expectorated is usually of dark colour, and more or less mixed with bronchial mucus. Its colour is the over-gorged veins and capillaries of the bronchial mucous membrane; and the immediate exciting cause is often a fit of coughing, which throws back the blood with great force into the bronchial veins and capillaries.

The countenance is usually more or less puffy, congested, and dusky—a result of defective aëration of blood and venous obstruction. Headache is a common result of the same state of blood and circulation.

The conjunctivæ, too, are often congested, and the eyes have a glistening watery appearance.

Emaciation is not usually observable, except in the advanced stages of the disease, or as a result of severe attacks of bronchitis. In consequence of the enlargement and air-distension of the chest, the buoyancy of the body in water is increased; so that a patient finds, to his surprise, that he has acquired an increased facility of swimming, notwithstanding his loss of muscular power and his increasing dyspnoea on exertion (Walshe).

The pulse and breathing are usually not quickened, except on exertion, or when bronchitis is present. Very commonly, the breathing is remarkably slow and heaving, and attended with a wheezing sound.

The voice loses tone and strength; and the patient is incapable of a sustained note, or of long or loud speaking. Sometimes the voice becomes husky from congestion and cedema of the mucous membrane of the larynx.

Cedema of the ankles, either with or without congestion of the kidneys and albuminuria, may occur as a result of obstructed pulmonary circulation and consequent dilatation of the right cavities of the heart.

The appetite and digestion are commonly impaired, and the patient is often distressed by flatulent distension of the stomach after food. This impedes the descent of the diaphragm, and tends to cause or to increase dyspnoea. It is not difficult to explain the flatulent distension of the stomach. The process of digestion depends upon the solvent action of the gastric secretion upon the food. The secretion of this gastric fluid is checked by the impeded circulation of blood through the stomach, consequent upon the obstruction in the lungs. In like manner, the secretion of urine is checked by the impeded circulation through the kidney. In consequence of the deficient secretion of the gastric solvent, the food ferments, decomposes, and gives off gases which distend the stomach.

Prognosis.—The prospects of an emphysematous patient are by no means favourable or pleasant. He may live for a considerable number of years; but he must expect a continual increase of his malady, with its attendant dyspnoea and liability to bronchitis. Emphysema, although in its early stages it is attended with no immediate danger, involves much chronic suffering, and tends materially to shorten life. The most unfavourable cases are those in which the disease is hereditary, and manifests itself at an early period of life. The immediate danger to life is usually associated with the bronchitic complications, the increasing obstruction to the circulation, and the consequent renal congestion with albuminuria, a scanty secretion of urine and dropsy, passive engorgement of the bronchial veins and capillaries, and a resulting serous effusion into the bronchi, which renders the lungs cedematous.

Treatment.—In the treatment of emphysema, we have to consider, first, the emphysema itself; and, secondly, the bronchitic and other complications. When once the lungs have become emphysematous, that condition is permanent, and we can do nothing effectual for its removal; but a knowledge of the various causes which tend to bring on the emphysema will enable us to do much to ward off the disease, and to prevent its increase. Now, it is manifest that amongst the determining causes of emphysema there are some over which we have more control than over others. Hereditary tendency to the disease, and the degenerative changes which occur with advancing years, are little influenced by treatment. But we can treat spasmodic asthma with more or less success. By diet and various medicines we can for a time prevent the mischievous reaction of a damaged valve upon the muscular walls of the heart, and the consequent hindrances to the pulmonary and systemic circulation; and we can warn our patients against the abuse of alcoholic stimulants, and in each of these cases our advice and our assistance may be productive of more or less benefit. But the class of cases in which, by timely warning, we may render the most valuable aid, are those in which there is an increasing tendency to the accumulation of fat beneath the skin and in various other parts. These subjects should be warned of what is in store for them if this fat-making disposition be not checked and counteracted; and then they should be taught that their chief safeguard consists in active exercise in the open air, and a carefully regulated diet. There is no rule without exceptions; but it may generally be assumed that very fat men and women are hearty and indiscriminate feeders; and however active they may have been in early life, in proportion as they increase in adipose bulk, they are little dis-

posed to active exercise. Now, if these subjects will escape the miseries attendant upon emphysematous lungs, they must restrict themselves to a diet somewhat after the fashion of that which is popularly known as Bantingism. Mr. Harvey of Soho Square conferred a great benefit upon Mr. Banting by cutting off his favourite bread and milk, and putting him on a judicious course of diet. Mr. Banting, zealous and grateful, appealed to all his fellow-sufferers from excess of fat to change their diet and improve their figures. Many of his disciples, acting without medical advice, did themselves serious harm by a too sudden and rigorous abstinence from fat and farinaceous food; they, in fact, starved certain tissues which require those hydro-carbon compounds for their nutrition, and thus they brought suffering upon themselves, and discredit upon Bantingism. Moderation is to be observed even in abstaining. Each case may require some special directions, but as a general rule, these fat-making subjects should be directed to take the fibre of fish, flesh, and fowl liberally, to eat sparingly of fat, butter, sugar, and farinaceous substances, including, of course, vegetables, bread, and pastry, to abstain entirely from malt liquors, and to take no more wine or spirit than is absolutely needful for health and comfort. This plan of diet, with daily walking or horse-exercise in the open air, will do much to check, although it may not entirely arrest, the tendency to accumulate an excess of fat.

I have said that these emphysematous patients are commonly dyspeptics, and suffer from flatulent distension of the stomach after food, and I have given the explanation of these symptoms. A complete cure of this form of dyspepsia can be effected only by removing all impediments from the way of the circulation—an impossible task. Yet something may be done to mitigate the symptoms. A combination of tincture of perchloride of iron and tincture of digitalis is sometimes beneficial; the iron as a tonic, while the digitalis, as a diuretic, tends to lessen venous engorgement, and so to increase the freedom of the circulation. A combination of mineral acids—hydrochloric or nitro-hydrochloric—with vegetable bitters, quinine, cascarilla, or calumba, and tincture or syrup of ginger is often very useful in assisting digestion, and preventing flatulence. One great object of medical care is to prevent catarrh and bronchitis, and to cut short these attacks when they occur. Amongst the most important means of prevention are warm clothing, protection from cold and wet, and, during the winter, a residence in a warm, dry, and equable climate.

The treatment of catarrh and bronchitis, whether occurring as a complication of emphysema, or apart from that condition of lung, I must make the subject of another lecture.

THERAPEUTICAL MEMORANDA.

[Under this head, we shall publish from time to time, as materials accumulate, short records of remarkable cases in practice which are sufficiently rare, interesting, or instructive, to deserve record, but do not call for lengthened statement or comment. Brevity and point should be the valuable characteristics of cases forwarded for this column.]

TREATMENT OF HÆMOPTYSIS BY ERGOT OF RYE.

By HORACE DOBELL, M.D.,

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FOLLOWING the lead of Dr. Symonds and Dr. Kennion, I venture to give the following memorandum. In common with other physicians who, like myself, are connected with hospitals for diseases of the chest, I see every year a large number of cases of severe pulmonary hæmorrhage resulting from a variety of causes.

There is scarcely any complaint that gives such serious alarm to the friends of a patient as profuse hæmoptysis; and there are few occasions on which a consultation is so urgently requested, and so readily granted by the family doctor, as when a patient appears to be "bleeding to death". But every medical man of experience considers himself perfectly qualified to treat hæmoptysis; and it is almost the rule, therefore, that, when called to these cases in consultation, one of the first remarks of the doctor in attendance is, that "everything possible has been done, and every remedy tried, but in vain." It is assumed, in fact, that the only object of the consultation is to sanction the inevitable death of the patient. Yet, according to my experience, it is exceedingly rare for a patient to die of hæmoptysis. In these remarks, I confine myself to cases of pulmonary hæmorrhage due to tuberculous disease of the lungs, which make up the large majority of all cases of severe hæmoptysis.