An Address

ON

SOME OBSERVATIONS ON BRONCHITIS AND EMPHYSEMA.

DELIVERED BEFORE THE ENGLISH DIVISION OF THE
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I HAVE chosen as my text to-day the subject of bronchitis and emphysema, a subject with which you must all be very familiar, and so there will be no necessity for me to traverse the whole ground; I shall therefore confine my attention to some practical points in the etiology, prevention, and treatment of these affections. I am afraid familiarity breeds contempt, and these diseases are not nowadays receiving the attention they merit.

At one time a chill was looked upon as the forerunner of a whole host of diseases, and so it was treated with the respect due to a progenitor; but since the introduction of microbes as the causal agents of all acute diseases, and owing to the fact that no specific one has been associated with bronchitis, a common cold was beginning to get neglected when influenza became a popular disease and carried in its wake nearly all catarrhal affections. Dr. Prosser White and others have spoken of a *Micrococcus catarrhalis*, but while many of us believe that there may be a regular flora of micro-organisms in the bronchial tree, we are not willing to credit any one with a specific action in the causation of bronchitis. If we could find any definite organism to which we could always ascribe this disease, it might help us along the road of prevention, though the discovery of the influenza bacillus has contributed nothing towards the prevention of that ubiquitous disease. In studying the etiology of bronchitis, we must content ourselves with the conditions under which it arises, and such a study will not, I hope, be barren of results. It has been said that if you know the cause of a disease you have advanced a long way towards its treatment, but in dealing with a disease which still holds the second place among the causes of mortality in this country, I think that prevention is of much greater importance than cure, and therefore it is with the object of prevention, and when not prevented with the object of obviating the dire effects of this insidious malady that I shall approach the subject.

ETIOLOGY AND PREVENTION.

Heredity plays a very important part. It is often said that every one has got a weak spot in his constitution, and those who have a tendency to any catarrhal or arthritic condition should be very careful in their mode of life, and pay special attention to the state of their digestive organs.

A *chill* due to exposure is often considered the most important cause of bronchitis, and while it is undoubtedly an etiological factor in the production of the disease, its importance can be easily overrated. A cold, dry atmosphere cures the bronchial catarrh frequently associated with tuberculous disease of the lungs. When a person is feverish or well supplied with internal fuel he can stand much external cold without any damage to his economy, but it is quite a different matter when the internal temperature is subnormal and the vital powers are depressed from any cause. A visit to Switzerland in the winter lessens the liability to bronchitis, provided your bedroom be clean and well ventilated and suitably heated, but it is very difficult to avoid *catching cold* on the return journey in the overheated, badly ventilated, and dirty Continental trains, which are nothing better than incubators for all manner of germs.

When the vital resistance of the individual is lowered, and during sleep, when the resistance of the nervous system is partially suspended, the individual is much more liable to the catarrhal effects of cold, hence bronchitis is more frequent and fatal in the very young and very old than in adults. In this cold, moist atmo-

sphere many aged individuals contract a fatal bronchitis in bed. Dry air is a very bad conductor of heat, hence a very dry, still atmosphere, no matter how cold it may be, cannot do much damage to the bronchial tubes. Of course the dryness of the atmosphere is a very relative term, as the amount of moisture which it can hold will largely depend on the temperature; if, therefore, a cold atmosphere is not to do any harm, the amount of moisture which it contains should be a long way below the saturation point. The more saturated the atmosphere with moisture the less will it take up of the watery exhalations from the lungs, and there may be even a condensation in the bronchial tubes, hence the super-saturated atmosphere of a greenhouse soon becomes almost suffocative. When cold dry air becomes warm in the air passages it readily takes up a large amount of watery exhalation. It is, therefore, the excess of moisture rather than the coldness of the atmosphere which works mischief, but yet, strange to say, some physicians still continue to use the bronchitis kettle. I have often said that the inventor of that appliance should have been hanged, and I am now inclined to think that his disciples should each get ten years' penal servitude for attempting, and often succeeding in the attempt, to drown their young patients in their own secretions.

In the cold dry climate of Canada and Switzerland bronchitis is much less common than in the moist and comparatively mild climate of this country. With nasal breathing there is not much danger of any degree of dry cold damaging the bronchial tubes. There can be no doubt that sudden variations in temperature do injure the body; the intense cold of liquid air will as effectually kill the part with which it comes in contact as a red-hot iron. What we have to do is to protect the body from the injurious influence of the sudden abstraction of heat which is chiefly affected by a cold moist atmosphere, especially when in rapid movement. The natural protection arises from the reflex action of the nervous system preventing any rapid loss of heat, but the nervous system is not always on the alert, and its regulative mechanism is not always sufficient to meet the contingencies arising from the sudden variations of temperature in this country. In the young and aged much greater protection than that afforded by the nervous system is required.

The influence of cold acting reflexly through the nervous system on the bronchial tubes has been experimentally shown by Rossbach. He found that when he applied cold to the skin "the blood vessels of the trachea contracted reflexly after one to two minutes, and this was soon followed by venous hyperaemia and an increased secretion of mucus." Personally I cannot put my foot on a cold stone without inducing nasal catarrh, hence I cannot recommend pedestrian exercise on bare feet, though I have no doubt this excessive reflex action might be easily overcome by a little hardening process. I recently saw a healthy old gentleman of 83 who was very lightly clothed, and did not wear singlet or drawers in this cold weather. He said that he believed in fresh air, which got much better at your skin when you did not wear any woollen underclothing. He acknowledged that for some years he had had some difficulty in maintaining his temperature and so had given up sea bathing in the winter. I noticed that his hands were enormous and spade-like, but his finger ends were not so bulbous as you find in pulmonary osteo-arthritis; the enlargement was uniform, and to my mind due to chronic local asphyxia owing to the action of long-continued cold. There can be no doubt that cold sponging or a rapid cold bath in the morning with good friction afterwards has a marvellous effect in preventing one "catching cold," but, even with the experience of this old gentleman before me, I cannot recommend insufficient clothing. The hardening process is excellent when judiciously carried out, otherwise it may be a rapid Darwinian method of getting rid of the unfit; but you have got to deal with the fit and the unfit, chiefly the latter, so be careful in the application of your methods of prevention.

When the cold has damaged the mucous membrane and caused some shedding of the epithelium of the bronchi, the inflammatory reaction is readily maintained by the numerous micro-organisms which infest the air passages, and by the dust in the atmosphere; hence the necessity for a clean mouth and nose and a pure atmosphere. The streptococci, staphylococci, pneumococci, the influenza

and diphtheria bacilli, and numerous other pathogenic bacteria are often very active on the bronchial mucous membrane. Measles are usually associated with bronchial catarrh. In the majority of cases of enteric fever the *Bacillus typhosus* finds its way into the alveoli and bronchial tubes, and sets up a low form of congestive bronchitis in the lower and posterior aspects of the lungs. The bronchial mucous membrane, when intact, is not a good culture field for the micro-organisms; at any rate, it is chiefly the minute divisions and alveoli which are attacked by the tubercle bacilli, the pneumococci, etc.

The injurious effects of various dusts on the bronchial mucous membrane are too well known to require any attention here. Numerous gases and vapours, such as chlorine, bromine, ammonia, sulphuric acid, hydrochloric acid, nitric acid, chlorinated lime, ether, etc., damage the mucous membrane and set up a bronchitis which is readily perpetuated by the pus-generating organisms.

Various constitutional disturbances are credited with the production of bronchitis, but we need only here refer to gout, which undoubtedly is a causal factor. In mitral disease, especially mitral stenosis, which leads to overloading of the pulmonary circuit, we frequently get a congestive bronchitis affecting the alveoli, infundibulae, and bronchioles, the blood supply of which belongs to the pulmonary circulation, but in the early stages the bronchi escape as their circulation belongs to the systemic system and the venous blood is returned by the azygos vein to the right side of the heart. In chronic kidney disease acute pulmonary oedema is not at all uncommon, but a true bronchitis is rather rare.

I think a very large percentage of cases of bronchitis is due to the excretion of toxins or irritating substances absorbed from the digestive tract. In bronchitis, asthma, and emphysema you will very frequently find a dilated stomach, and constipation; and the best way to prevent and cure such attacks is by attention to the digestive tract. If we include alcohol among the toxic substances absorbed from the stomach, then I think we may associate 50 to 70 per cent. of the cases of chronic bronchitis with the digestive tract. It might be worth while inquiring how alcohol causes and maintains chronic bronchitis, and in this way is such a potent agent in the production of emphysema. It is generally supposed that the excretion of alcohol sets up an irritation, but I am very dubious about this, because if such arose from excretion we should get a capillary bronchitis owing to the alveoli being supplied by the pulmonary vessels, but in bronchitis due to alcohol it is invariably the bronchi, trachea, larynx, and throat which are involved, all supplied by the systemic system of blood vessels. I look upon the effect of alcohol as a vasomotor paresis of these vessels, which are not well supported, and this gives rise to vascular turgescence and oedema of the mucous membrane. In short I look upon alcoholic bronchitis as an active rather than a passive hyperaemia. These cases frequently end in emphysema. Tobacco tends to raise the blood pressure, and then there is the direct irritation of the smoke. In carbolic acid poisoning, where the irritation arises during excretion, we get a capillary bronchitis or a congestive pneumonia.

TREATMENT.

In dealing with the etiology I incidentally referred to prevention. In the treatment of any disease the first indication is, if possible, to remove the cause, and when that is not possible to lessen or obviate its effects. The patient should be hardened, and protected against the injurious influences of cold, dust, micro-organisms, irritating gases, etc. He should live as far as possible in a pure dry atmosphere, which, at least during the acute stage of the disease, should be warm. In certain dry climates the amount of moisture in the atmosphere has to be considered, but in this country this is not necessary as there is always too much. Every person with a liability to bronchitis should forswear the use of alcohol in all its forms. It is one of the most frequent causes of the disease in the chronic form, and is never useful in treatment.

When bronchitis arises in connexion with any infectious disease, or with disease of the heart or other organs, the treatment is chiefly symptomatic in addition to that of the associated disease. When it arises, as it frequently

does, from disturbances in the digestive tract, the attention should be especially devoted to the stomach and bowels. As a rule, carbohydrates and fats should be cut down, and the patient may be granted a liberal allowance of nitrogenous food. The diet should not be too liquid, though a fair amount of hot water is permissible. If there be any deficiency of lime salts in the blood, which is often the case when the expectoration is free and albuminous, a moderate amount of milk may be given, also some preparation of gelatine. In cases where the expectoration is scanty and tough, and where there is much bronchial spasm with sonorous and sibilant râles, I think the lime salts in the blood will be found to be excessive, and in such cases milk and gelatine should be avoided. Large, corpulent, plethoric individuals should be strictly dieted and their weight reduced. In my experience the use of sodium chloride, which has a high osmotic equivalent, should be restricted. In many cases a good emetic is an excellent preliminary treatment both for the stomach and the lungs; this is especially useful in the capillary bronchitis of children. In all cases the bowels should be well cleared out; calomel and sodium sulphate are perhaps the best purgatives.

Drugs.

I shall not say much about drugs, because in this disease the treatment should be largely hygienic and dietetic, and as a rule the fewer drugs you use the better. The so-called expectorants had better be reserved for the chronic stage of the disease; ammonium carbonate, which seems to be the basis of nearly every expectorant mixture, is certainly injurious in acute stages. I very seldom prescribe it except as a stimulating emetic in cases of children suffering from capillary bronchitis. I am very pleased to see that Dr. Eustace Smith has recently been strongly advocating the use of antimony, because antimony and opium have long been favourite remedies with me in the treatment of many chest affections. Compound tincture of camphor with ipecacuanha can also be highly commended. I am not likely to be accused of underrating the use of lime salts in disease, but I have long recognized that you can have too much of a good thing; and so, if there be much bronchial spasm and tough expectoration, decalcifying agents, such as the citrates of soda and potash, should be freely administered. When the expectoration is very liquid, profuse, and albuminous I think the lime salts will be found deficient; and as a certain amount of free calcium ions in the blood is necessary as a cardiac tonic and to lessen secretion, the administration of calcium lactate, milk, and jellies is desirable. Now that my friend Dr. Blair Bell has given us an accurate method of estimating the free calcium ions in the blood, I should like to see these observations of mine, which are based on long experience and, I think, fairly accurate investigations, either corrected or corroborated. In bronchial spasm the nitrites are very valuable, and potassium or sodium iodide is an important adjunct; when the acute stage has passed, tonics such as quinine, strychnine, and hydrochloric acid should be given. As a cardiac tonic digitalis is occasionally necessary, and apomorphine and strychnine make an excellent expectorant mixture. In very chronic cases terebene and others of the turpentine series are very useful.

Counter-irritation is often extremely valuable, and personally I prefer mustard poultices to anything else. The profession in this country are very much indebted to Dr. A. G. Auld of London for drawing attention to the lung reflexes of Albert Abrams of San Francisco, which afford a reasonable explanation of the good effects of counter-irritants. I have often corroborated the statement of Dr. Auld that if in a case of bronchitis you rub the chest for a minute or two over an area of lung where the breath sounds are deficient you can hear the air entering freely, and the percussion note becomes more resonant. For many years I have used *tapotement* for hastening the recovery of unresolved pneumonias, but since Dr. Auld drew attention to the lung reflexes of Abrams I have used a combination of *tapotement* and *effleurage*—*tapotement* makes the lung contract, and *effleurage* causes it to expand. These are facts which anyone can demonstrate for himself, and in bronchitis I think *effleurage* will be found of most value, though it may be alternated with *tapotement*.

VESICULAR EMPHYSEMA.

In order to understand the production of emphysema I think it is necessary to have a working knowledge of the elasticity of the lungs. So far as the lungs are concerned, the best illustration of elasticity is a piece of rubber, and it is not with any abstract property of the lungs or rubber called elasticity with which you are concerned, but with the active manifestation of that property. Professor Bottomley recently said that if he were asked to tell what life was he would answer: "A metabolic activity of protoplasm, but candidly he knew nothing more than anyone else. He was simply telling them that life was life. You could only tell life by its manifestations." So elasticity is elasticity, and you all know what that is as well as the greatest scientist among us.

You cannot tell whether a body has the property of resisting a given deformation, or of recovering from the strain when the stress is removed, unless you try. The abstract question of the possession of a property is of no value until the use of that property is made manifest. You can only know whether the rubber or the lungs retain their elasticity by trying. You can stretch the rubber, and see if it recover its original form after you cease stretching, and with the spirometer you can test whether the lungs are elastic, and how elastic. The elastic force of even healthy lungs is not very great, but it is quite sufficient for all the purposes which it is called upon to perform. In fact, I can quite see that if it were much greater there would be a constant danger of producing pulmonary collapse. In my Bradshaw Lecture I showed that in exhausting diseases, where there was not much demand for oxygen, even a whole lung might collapse.

The first experiments on the elasticity of the lungs were performed by Carson, of Liverpool, at the beginning of last century. His results were rather higher than those which are given by physiologists in the present day.

In calves, sheep, and in large dogs the resiliency of the lungs was found to be balanced by a column of water varying in height from 1 ft. to a 1½ ft.; and in rabbits and cats by a column of water varying in height from 6 in. to 10 in.² (An inch column of water is equivalent to 1.87 mm. of mercury.)

Donders calculated the elastic pull of the lungs in the expiratory condition as about 7.5 mm. Hg. After an ordinary inspiration this is increased to 9 mm., and after the deepest possible inspiration to about 30 mm. It is evident that these figures also represent the negative pressure in the thoracic cavity under the same conditions.³

Even in expiration the lungs are in a state of elastic tension and therefore exert traction on the thoracic walls, and as Sir Douglas Powell has pointed out, there is a tendency for the thoracic walls to recoil in the opposite direction, thus the inspiratory muscles start work at an advantage owing to the elastic recoil of the chest walls balancing the tug of the lungs. The inspiratory muscles enlarge the chest and thus increase the negative pressure in the pleurae, but if the lungs were not free to follow the movements of the chest the inspiratory muscles would be absolutely powerless to expand the chest against the atmospheric pressure, and if the lungs were free to follow the movements of the thoracic parietes but non-resistant there could be no negative pressure in the pleurae. In cases of pleurisy with effusion, or pneumonia, or fibroid phthisis of one lung the inspiratory muscles have little or no power over the affected side of the chest, and in cases of advanced emphysema the chest is filled to its utmost capacity and the negative intrathoracic pressure disappears. In ordinary healthy breathing the negative pressure in the chest is slight and varies during the different phases of respiration, but it is continuous and affords a constant assistance to the forces carrying on the circulation. In my Toronto address I showed that

when the resistance is *nil*, a pressure-gradient of 5 mm. of mercury in a vein will give you a velocity of 1,120 mm. in the second,

but in order to obtain a similar velocity where the resistance equals 23 mm. of Hg, you require a fall in the pressure-gradient from 40 mm. to 23 mm. of mercury. It therefore follows that while a slight constant negative pressure affords great assistance to the circulation, in emphysema, where the negative pressure not only disappears but may become positive during expiration, not only is there no assistance but a positive hindrance to the circulation. I have demonstrated that

in the Valsalva experiment the pressure can be raised so high as to shut the blood out of the chest. The lungs are always, both in inspiration and expiration, more or less stretched, and in order that the elasticity should be maintained in a healthy condition they should have free play. The inspiratory muscles starting at the end of expiration at an advantage, owing to the tendency to recoil of the chest walls, pull against the atmospheric pressure, and tend to leave a negative pressure on the inside of the thorax, but as these muscles when in most powerful action are only able to support a load of about 100 mm. of mercury, or less, the negative pressure in the thorax can never exceed that amount. The elasticity of the lungs only accounts for a fractional part of this amount, so you can only attain this great negative pressure by shutting the air out of the chest.

In emphysema the lungs have lost their elasticity, and hence the negative pressure due to their traction disappears; the non-elastic lungs fill the enlarged chest, and cannot be even partially emptied during expiration, so it is impossible to establish any marked negative pressure in the thorax under such circumstances. In tuberculous disease of the apices of the lungs with or without pleuritic adhesions, and in fibroid phthisis of the upper lobes, the affected portions of the lungs cannot take any part in filling up the enlarged thorax, consequently the costal cartilages are pushed in by the atmospheric pressure, not dragged in or sucked in; the healthy expansile portions of the lungs—which from over-stretching may lose their elasticity and eventually become emphysematous—fill up the rest of the enlarging thorax. Recently, in a very instructive case of pneumonia in a youth involving the whole of the right lung, I noticed that the costal cartilages over the sound lung were driven in during inspiration, while the lower part of that side expanded freely; this was an indication that pneumonia was starting in the left upper lobe, which was losing its expansile power, and the following day this lobe showed signs of consolidation. When the lobe becomes enlarged and hepatized the negative pressure over it disappears and the chest wall is not driven in. In cases of pleural effusion and pneumothorax which prevent the lungs from following the movements of the chest walls the inspiratory muscles are quite incapable of expanding the affected portion of the chest against the atmospheric pressure. However, the inspiratory muscles, like the individual, often attempt the impossible, and in cases of asthma and emphysema try to enlarge the chest beyond its utmost capacity in order to get air in. If patients were only taught to drive out a little of the air already in there would be no difficulty in inspiring a little fresh air to replace that displaced. The patient may have been foolishly taught that it was an advantage to keep his lungs *taut*, and that he should in emphysema get his thorax as large and rigid as possible so as to keep the lungs always on the stretch. If the patient were of an inquiring mind he might ask his mentor what he should do with his diaphragm, because on the same parity of reasoning it should be an additional advantage to have the diaphragm low down and rigid so as to keep the lungs *extra taut*.

Nature often gets credit to which she is not entitled. I do not look upon rigid chest walls as ever advantageous or a natural process at all, but a piece of bad art. Nature copiously provides for the perpetuation of the species, and has endowed man with a vital capacity five or six times more than is requisite in health, but if he fritters this away, for heaven's sake do not blame Nature, but his own crass ignorance or that of his physician. Those who ascribe to Nature anything which they do not comprehend should remember the advice of Samuel Haughton to beware lest *their* Nature be driving nails in their coffins. If you wish to keep your lungs healthy and free from emphysema you must keep your chest walls pliant and give your lungs plenty of exercise; the more their elasticity is exercised the longer it will last. The way to exhaust the elasticity of the lungs or of a piece of rubber is to keep it steadily on the stretch. An enormous number of people have never been taught how to breathe properly, or erroneously taught to breathe with their diaphragm. For very many years I have been teaching patients to breathe with their thorax, and Nature will look after the diaphragm.

Huttkrantz made a comparison between the diaphragm and the thorax in regard to their respective shares in respiration

by enclosing the lower part of the body and the trunk, with the exception of the head, separately in an air-tight metal vessel provided with a water manometer. He concludes from his experiments that of the 490 cubic centimetres (29.9 cubic inches) of inspired air, 320 (19.5 cubic inches) are accounted for by the expansion of the thorax, and 170 (10.4 cubic inches) by the depression of the diaphragm. The influence of the depression of the diaphragm, as determined by this method, may, it is true, be somewhat less than it is in reality, because the air in the abdomen suffers compression at each inspiration, but in spite of this source of error the experiments clearly demonstrate the great importance of thoracic movements.¹

The lesson to be learned is, keep your thorax moving, and I know no better form of respiratory gymnastics than that to be obtained by hill climbing; you have got plenty of hills in Cumberland. This sets all the respiratory forces in action, and as a health-giving exercise golf and Scotch whisky are not in it with hill or mountain climbing.

In 1858-9 Freund stated that a certain number of cases of emphysema originate in a specific calcification of the costal cartilages, and are not primarily due to pulmonary disease. This, perhaps, is an overstatement, but it contains a large amount of truth, especially in the aged. Some old people retain pliant costal cartilages and healthy lungs; others, without even waiting to be aged, get rigid costal cartilages and emphysema without any previous history of catarrh or other pulmonary lesions. If the costal cartilages are not kept in healthy exercise some of the lime salts which are frequently in superabundance in the blood get deposited in them; they become rigid, the chest walls assume the inspiratory position; the lungs are kept constantly on the stretch, lose their elasticity, and become emphysematous. Freund recommended division of the affected cartilages, and three very successful cases have been recorded. I can refer you to an interesting leading article in the BRITISH MEDICAL JOURNAL of January 25th, 1908. I see no objection to this operation in advanced cases; but in this, as in everything else, prevention is better than cure. Make your patients exercise their chests, and keep down the unnecessary amount of lime in the blood. I have known physicians to forbid patients to drink hard water on account of the lime which it contained, and at the same time order them plenty of milk diluted with some Continental spring; really, the absurdities of medical practice are appalling. No wonder the surgeons have a frequent look-in. Whatever you do, do not continuously fix your chest walls either in the position of inspiration or expiration. I have known irreparable mischief done by the long use of spinal supports. Follow Nature, give your lungs plenty of exercise if you wish to keep them healthy, and do not adopt the deplorable art of some physicians and surgeons in fixing the chest walls.

In every complete examination of the lungs the spirometer should be used; it gives far more valuable information than any stethoscope. The extraordinary method of estimating the capacity of the lungs by the circumference of the chest in inspiration is still in force in the army. One of the best light-weight boxers that I know, a man who could have knocked any one of his examiners out in the first round, was nearly rejected because the circumference of his chest did not come up to the standard. A big, emphysematous chest would have more than satisfied them. The movements of the chest, and the vital capacity of the lungs should be considered rather than the tape-measure.

The foregoing considerations are extremely necessary for the clear apprehension of the causes and effects of alveolar emphysema of the lungs. A good deal of contention has arisen as to whether emphysema is inspiratory or expiratory in origin. My friend and teacher, the late Sir W. T. Gairdner, held to the inspiratory theory, but he also held that healthy lungs were always capable of filling the thorax which contained them, and consequently they could not become emphysematous until there was some destruction or loss of function of some part or parts of the lungs, so that the remaining healthy portions were incapable of filling the thorax and consequently became emphysematous. Thus emphysema follows such widely differing diseases as tuberculous disease of the lungs, fibroid phthisis, pleurisy with effusion, bronchitis, asthma, etc., and the emphysema does not occur in the collapsed or damaged portions of the lungs, but in the neighbouring healthy alveoli. He found perfectly healthy lungs in an

aged piper, and therefore he would not accept the expiratory theory of the production of emphysema, because he held that during expiration the lungs were well supported and there was no tendency to excessive distension. According to him, emphysema was always *complementary*, so as to fill up the space left by the collapsed or otherwise damaged portions of lung, and never *compensatory*, as the enlargement of the alveoli with destruction of the septa must diminish the aerating space—the vital capacity is always diminished—and so there can be no increase of function.

I wonder what he would have said of that monstrous expression, "hypertrophous emphysema," which is commonly used in the present day. You may, if you choose, use the terms "large-lung and small-lung emphysema" of Jenner, which imply no theory as to nature or causation, though I think the terms are unnecessary, as the emphysematous lungs are no larger and no smaller than the cavities which contain them; if the emphysema occurs late in life, in small, rigid chests, which are incapable of expansion, then you must get the small-lung emphysema.

Dr. A. G. Auld² says:

Now, as to the air cells, the first effect of the increased tension is to produce a compensatory reaction in the respiratory elements. It is a mistake to suppose that they immediately submit to the injurious influence. Both the epithelial and connective tissue cells enlarge and many of them divide. They behave, in fact, like any other tissue on which an inordinate demand is made, and this hypertrophy is, indeed, the analogue of what occurs in the muscle nuclei of the infundibulae. . . . The reaction of the respiratory cells is not of long duration; they shortly enter into the degenerative stage. Their protoplasm becomes granular and fatty and the nuclei vacuolate and fall to pieces. The part has, however, become permanently enlarged through atony of the elastic fibres, and many of these show a granular change—a finely beaded state of the fibrils. Small holes appear in the granular protoplasm, which speedily enlarge, and in this way many of the septa become completely destroyed.

This hypertrophy is a futile attempt to prevent further mischief, but it never can produce any increased respiratory function.

For a long time I implicitly accepted the teaching of Sir William Gairdner, until I began to work at the subject for myself; I then found that the inspiratory theory would not mechanically explain all cases of emphysema. I quite agree with Sir William Gairdner that there must first be loss of function or destruction of some parts of the lungs, and if this loss of function be at all considerable, and there be an increased demand for oxygen, then the inspiratory muscles, having been taught that it is their duty to get in the oxygen, start active work, and thus tend to increase the negative intrapleural and intrathoracic pressure; and all the more so as the healthy portions of the lungs, being still elastic, and the diseased portions collapsed, are not free to follow the movements of the chest walls. Let us suppose that under these circumstances the negative intrathoracic pressure is 30 mm. of mercury, and it is never likely to be much more than this, as these patients are not capable of doing a good Müller's experiment. In an atmosphere of 760 mm. of mercury pressure this would give you an intrathoracic pressure of 730 mm. of mercury. If, at the same time, the patient were breathing freely through the mouth, the intrapulmonary pressure might be 758 mm. of mercury. You could therefore have an intrapulmonary pressure of 28 mm. of mercury stretching the lungs at all points where they were not directly supported by the external atmospheric pressure. Above the clavicles, and to a less extent behind the soft pliant costal cartilages, and above the diaphragm, the intrapulmonary pressure would be to a large extent balanced, or more than balanced, by the external atmospheric pressure. Above the clavicles the intrapleural negative pressure might disappear, and so the inspiratory theory could not account for the emphysema of the apices of the lungs. Under these circumstances the lungs might be said to be taut, though the lungs can never be said to be taut in the sense of being tightly stretched. In these cases the stress in the lungs is slight, amounting, as I have said, at the most in inspiration to 28 mm. of mercury, but as it is greater than the elasticity of the lungs, there is more or less constant stress of a few millimetres of mercury in the healthy alveoli of the lungs, which diminishes, but in the early stages of the disease does not entirely disappear, during

expiration. This stress affects the healthy but not properly supported alveoli in the neighbourhood of the collapsed portions, and is probably sufficient to give rise to permanent distension with the production of emphysema. When the lungs have lost their elasticity and become emphysematous, they readily follow the limited movements of the chest walls and the intrapleural and intrathoracic negative pressures disappear. This readily explains a large number of cases of emphysema, but it will not explain those partial cases where it occurs at the apices, along the anterior margins, and at the bases, and for such we must consider the expiratory theory.

During a violent fit of coughing the intrapulmonary pressure may readily rise to 840 or 860 mm. of mercury; this not only abolishes the intrapleural and intrathoracic negative pressures, but exceeds the atmospheric pressure by 80 or 100 mm. of Hg. Where the lungs are well supported by the ribs this extra pressure cannot matter, because such support must equal the internal pressure, and there cannot be at these points any hyperdistension of the lungs; but the parts which are not thus supported, such as the apices, the anterior margins, and the bases lying on the diaphragm, are subjected to a stress equivalent to the difference between the atmospheric and the intrapulmonary pressures. The same stress will apply to cases of cicatricial puckering of the lungs, which prevent portions of the lungs being uniformly supported by the ribs. I have seen the whole surface of both lungs covered with large bullae, and fibrous bands down in the sulci between them. Of course this might be attributed to the strain of the lungs during inspiration, but I think I rather overstated the stress in such cases when I put it down at 28 mm. of mercury, and when the lungs begin to lose their elasticity the stress can never amount to anything like that, and under no circumstances in such cases can it equal a third or a half of that produced by coughing. During inspiration the stress on the lungs depends on the difference between the intrapleural and the intrapulmonary pressures, and as this perhaps never exceeds 30 mm., and, I think, rarely exceeds 10 mm. of Hg, it depends on your idea of strain as to whether you consider that inspiration can make the lungs taut or not. In Müller's experiment, where the intrapulmonary, intrapleural, and intrathoracic pressures are very low, the difference between the intrapulmonary and the intrapleural will entirely depend on the elasticity of the lungs. It therefore follows that Müller's experiment cannot produce emphysema of the lungs, and the best way to prevent inspiration causing any excessive tension in the alveoli is to make the patient breathe through his nostrils so as to keep the intrapulmonary pressure markedly negative. An even better way would be to teach the patient to expire so as to get rid of the supplemental air and allow inspiration to be largely performed by the recoil of the chest walls.

Nature's object in respiration is to oxidize the blood and render some assistance to the heart in carrying on the circulation; when the lungs are becoming emphysematous, the elasticity getting impaired, and the vital capacity diminishing, these objects can be better carried on at the lower end of the scale than at the upper. The individual and his inspiratory muscles have been taught and acquired the habit of getting air into the lungs by expanding the chest; but this is art, not Nature. You should determine which is the best way of aerating the blood under any given set of conditions. When in emphysema the chest is expanded to its utmost capacity the lungs are stretched; but they cannot be tense as they have in great part lost their elasticity, and the only means of getting air into and out of the chest is by the action of the diaphragm. Fortunately the diaphragm does not follow the example of the ribs and become taut, otherwise all the oxygen which could be introduced to the lungs would be through the natural diffusion of gases, which would be utterly inadequate. Don't blame Nature for your own bad art, keep your lungs as elastic and as slack as you can, the less taut the better so long as they are expanded; the tautness or tension depends on the difference between the intrapulmonary and intrapleural or intrathoracic pressures, and of course in healthy elastic lungs there must always be a certain amount of tension, as the pulmonary tissue is not completely relaxed even at the end of

a deep expiration. When the pleural space is obliterated in cases of fibroid phthisis, there may be a negative intrathoracic pressure, in which case the pressure outside is greater than that within the chest, and when the thorax is expanding in inspiration the expansile portions of the lungs fill up the increased space as far as possible, but the portions of the chest over the non-expansile portions of the lungs, especially at the apices and behind the costal cartilages, are caved in by the atmospheric pressure, not dragged in by any fibroid adhesions, or imaginary suction power. If a negative intrathoracic pressure be constantly maintained there can be no obstruction to the entrance of blood into the chest as far as the right ventricle, and it must lessen the obstruction to the output of blood, but beyond this it is the duty of the right ventricle to carry on the pulmonary circulation, and in order to accomplish this in emphysema it often becomes much hypertrophied. In advanced cases of fibroid phthisis and emphysema the negative intrathoracic pressure disappears and may even become positive; there is then general venous obstruction, giving rise to enlargement of the liver, congestion of the kidneys and other abdominal organs, and clubbing of the finger ends.

I have dealt pretty fully with the mechanism of the production of emphysema, and it now remains to refer to a few of the ultimate causes of the disease. Incidentally I have dealt with those causes which give rise to destruction or apneumotosis of certain tracts of lung with complementary enlargement of neighbouring tracts. Among these causes may be mentioned pleural effusion, fibroid adhesions, chronic pneumonia, fibroid phthisis, tuberculous disease of the lungs, chest deformities, traumatism, chronic congestion of the lungs from heart disease, and chronic catarrh. Of all these causes chronic bronchitis is the most frequent, because the catarrh may involve the whole of the bronchial tree and give rise to extensive collapse of numerous alveoli, and this leads to complementary distension of the neighbouring alveoli under the inspiratory and expiratory forces. Alcohol maintains a chronic bronchial catarrh and gives rise to excessive strain from frequent coughing. Asthma gives rise to acute pulmonary distension of the inspiratory type, which, if long maintained, may lead to permanent emphysema, and more especially so as asthma is frequently associated with bronchial catarrh. In dealing with bronchitis we dealt with occupation, irritating dusts and gases, infection, and affections of the digestive tract, all of which take an indirect part in the causation of emphysema, but dilatation of the stomach is here not such a common feature as it is in bronchitis and asthma.

Emphysema becomes more common as age advances. It may occur in the young as a sequel of some destructive lesion, and when it occurs in association with asthma and bronchitis while the costal cartilages are still pliant it is fairly amenable to treatment. Senile emphysema occurs in the aged when the chest walls are rigid, and is generally looked upon as an atrophic lesion due to the loss of elasticity of the lungs, though I am quite willing to accept the opinion of Freund that the calcification of the costal cartilages and rigidity of the thorax may be the primal cause, and the loss of elasticity and atrophic condition secondary to the loss of free play of the lungs. The so-called hereditary tendency may, therefore, be the outcome of arterio-sclerotic change and early calcification.

TREATMENT OF EMPHYSEMA.

What I have said about the treatment of bronchitis largely applies to emphysema; it should be chiefly dietetic and hygienic, and prevention is always better than cure. These patients have frequently too good an appetite, are stout and plethoric, and thus there is a demand for more oxygen than the lungs are able to supply; moreover, the plethoric and cyanotic conditions lead to congestion of the lungs, greater respiratory efforts are made, and the emphysema is increased. The patient thus gets into a vicious circle, from which it requires some judgement and determination to extricate him.

In cases of emphysema try and starve your patients as much as possible; they and their friends won't let you, as it is most difficult to restrict the diet of a patient with a good appetite who has never learned to practise any self-denial. At any rate, you should cut down the fats and carbohydrates, and you can give a fairly liberal

allowance of nitrogenous food so long as the excretory functions of the kidneys are well maintained. The thin, spare man with no excess of oxidizable material in his blood does best. The fat, pot-bellied individual cannot make proper use of his diaphragm, and there is an excessive demand for oxygen which his emphysematous lungs cannot supply. He can have a fair amount of hot water. Two or three pints of milk form a light diet if it be not supplemented by much else. If there be any excess of lime salts in the blood, milk and gelatine are objectionable, but frequently the lime salts are rather deficient, so I think it is always well to examine the blood. I prohibit the use of much table salt, but I allow plenty of fruit, and a fair amount of green vegetables. The latter have the advantage in helping to regulate the bowels, which are usually confined. Alcohol and tobacco should be strictly interdicted. Tea and coffee may be allowed in moderation.

Hygiene.

The patient should live in a clean, dry, moderately warm atmosphere, as pure and free from dust as possible. He should practise respiratory gymnastics, and be taught to expire rather than inspire; the difficulty is in getting rid of the stagnant supplemental air. Establish a negative intrapulmonary pressure, and the atmospheric pressure will soon drive some fresh air in. Teach the patient to keep his costal cartilages soft and pliant, give his lungs as much play as possible, and thus maintain their elasticity, and adopt thoracic breathing rather than abdominal breathing; from faulty teaching and long acquired habit the latter method of breathing is too deeply ingrained to require any special instruction. In order to give the lungs plenty of play I think there is no exercise equal to hill climbing; it calls all the respiratory muscles into play, and makes the individual expire as well as inspire; it has also a beneficial effect on the heart, and the amount of exercise can be easily regulated according to the capacity of the individual. In summer the patient can be sent to an elevated region up to 4,000 or 5,000 feet above sea level, provided the vital capacity be not below 150 cubic inches for the male and 100 for the female. A few years ago I saw an emphysematous man foolishly attempt the ascent of Mont Blanc, but he was very glad to turn back when he reached the Grands Mulets. For the injurious effects of very high altitudes on the circulation I cannot do better than refer you to the writings of the late Dr. Alfred L. Loomis¹ and Dr. Theodore Zangger.²

Numerous mechanical devices and pneumatic cabinets have been invented to assist respiration in these cases, but they have all practically gone out of vogue, except perhaps some simple elastic bands to assist expiration. Some patients find it more comfortable to sleep in the prone posture on account of the assistance afforded to expiration. Nasal and thoracic breathing should be encouraged; in this way the costal cartilages are not so apt to become rigid, but a watch should also be kept on any excess of lime salts in the blood. Cold sponging in the mornings, the Aix douche with massage, and other hydropathic measures can be commended, but the patient should not be left long in a moist chamber.

Drugs.

Their name is legion, but comparatively few of them are of much value; the most generally useful are the iodides and the various preparations of iodine. When there is any deficiency in metabolism or the patient is too stout, I frequently add thyroid to the iodine. If there be any excess of lime in the blood, the citrates of sodium and potassium should be freely administered, or lemonade made with citric acid and cream of tartar may be freely given. Of course, in such cases milk should be forbidden. The bowels should be kept open with a dose of sulphate of sodium in the morning. Bronchitis should be treated as previously directed; if there be much cough, it should be assuaged by some preparation of opium. In the later stages, when there is cardiac failure, special attention should be directed to that organ. If the costal cartilages have been allowed to get very rigid, the surgeon may be called in to perform the operation recommended by Freund.

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Arris and Gale Lectures

ON

THE PHYSIOLOGICAL AND PATHOLOGICAL EFFECTS WHICH FOLLOW EXPOSURE TO COMPRESSED AIR.

DELIVERED IN THE THEATRE OF THE ROYAL COLLEGE OF SURGEONS ON MARCH 18TH AND 20TH, 1908.

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LECTURE I.

No races of mankind—indeed, no species of terrestrial animals—are habitually exposed to an atmospheric pressure which exceeds by more than a few millimetres of mercury that normally obtaining at sea level. Accordingly, our experience of the phenomena produced by such exposure is comparatively modern, not antedating the sixteenth century (Sturmius, Halley), and has only become wide since technical improvements in the diving-dress and the invention of subaqueous work chambers, or caissons, have rendered possible extensive aquatic engineering and marine salvage feats.

EFFECTS OF RAPID DECOMPRESSION: CAISSON DISEASE.

Whether we consider caisson workers or divers, we are faced by the same condition—exposure to compressed air. Broadly, the distinction between the two classes is that while caisson workers are usually exposed to a lower total pressure than divers, they remain under that pressure for a considerable time, while the diver commonly returns to the surface after a relatively short exposure. In both cases disease and death have been, and unfortunately still are, of frequent occurrence. The following are some caisson statistics:

Douchy Mines: 63 cases.
Kehler Bridge: 133 cases.
St. Louis Bridge: 119 cases out of 352 workers; 50 cases of paralysis; 14 deaths.
Brooklyn Bridge: 110 cases, 3 deaths.
Toulon Dry Dock: 43 cases, 2 deaths.
Culsac Bridge: 104 cases, 3 deaths.
Elsler Bridge: 38 cases, 2 deaths.
Nussdorf: 320 cases among 675 workers.
Felesti Bridge: 55 cases and 5 deaths among 154 workers.
Hudson Tunnel: 3 deaths a month until the conditions were improved.

Since May, 1906, 10,000 exposures to compressed air have been associated with 16 deaths.

It would be unnecessary, even were it possible, to give a clinical description of the cases, varying from simple joint pain to severe paralysis and death. One of the best accounts we have of a rapidly fatal case is due to McKinlay (1900).

The subject was a man of 33, exceptionally strong, and in good health. He descended 24½ fathoms in search of a torpedo, being therefore exposed to a positive pressure of between 4 and 5 atmospheres. He remained below forty minutes, and ascended in half that time. He entered the boat without any trouble, and, when the helmet was removed, chatted unconcernedly about his work. He had suffered no inconvenience in the water, and only returned because the light was insufficient for his purpose; his comrades agree that he had never before come up in better condition. All was well for some eight or nine minutes, when suddenly he experienced pains in the stomach, and asked the attendants to get his dress off quickly, as he wished to go on board. In a few seconds he said, "Send for the doctor," and immediately collapsed. Fleet Surgeon McKinlay, who was in attendance at once, found the man cyanosed and breathing stertorously, the lips covered with foam; he was undressed and taken to the sick bay, but expired before arriving there, fifteen minutes after gaining the surface and six or seven counting from the first symptoms.

A careful autopsy revealed the following state of affairs: The surface of the brain was deeply engorged with dark fluid blood; many air bubbles were detected in the veins of Galen and choroid plexus, also in the superficial cerebral vessels themselves when the latter had been partly emptied of blood. The veins on the surface of the heart were beaded with air, while that organ felt like a bladder half full of water, gurgling loudly when pressed. On incision of the right ventricle air escaped with a puff; the left ventricle was empty. The mesenteric vessels were likewise filled with air and the liver frothed on section.