An Address

ON

THE THERAPEUTIC VALUE OF GASES INTRODUCED INTO THE PLEURAL CAVITY:

NITROGEN DISPLACEMENT (ARTIFICIAL PNEUMOTHORAX) AND OXYGEN REPLACEMENT.

DELIVERED BEFORE THE SOUTHAMPTON MEDICAL SOCIETY, MARCH 4TH, 1914.

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(With Special Plate.)

THE subject of the therapeutic value of gases introduced into the pleural cavity in the treatment of certain diseases of the lungs and pleura may be divided under two headings: (1) Nitrogen displacement; (2) oxygen replacement, the gas used in each case being indicated by the title.

The former of these two procedures is now passing beyond the first experimental stage, and is gradually gaining the recognition which it deserves. As must necessarily happen when a new method of treatment is introduced, the earlier results are not so satisfactory as those obtained when the lessons taught by experience have been learnt; and as there have been in the past some unfavourable results, and even a series of fatalities, this method has been looked upon with ill favour by many people. Now, however, that we appreciate more fully the type of case which is suitable for this form of treatment, and the means whereby the fatal results can be prevented, nitrogen displacement must, by the very excellence of its results, become more and more extensively used, and many lives which would otherwise have been lost can now be considerably prolonged, and many invalids who would have been unable to return to work can be made fit to resume their occupation.

The other method—oxygen replacement—has been used by me during the last two years as a means of rigidly controlling the intrapleural pressures while removing fluids from the pleural cavity. The results are eminently satisfactory, and I hope that I shall succeed in showing that oxygen replacement should become a routine procedure.

NITROGEN DISPLACEMENT.

Normally there is a potential cavity between the visceral and the parietal pleura, and between these two layers there is a negative pressure equal to about 7 mm. of mercury. This negative pressure is considerably increased by deep inspiration, and decreased by expiration. If the expiration is a very violent one—for example, coughing—the pressure may become positive. The negative pressure prevents the lung from being collapsed by the elastic tissue which it contains.

When nitrogen is run into the pleural cavity the lung slowly collapses, and in the majority of cases will eventually, if sufficient gas is introduced, lie flattened against the mediastinum, the intrapleural pressure changing gradually from negative to positive. If the lung is not extensively diseased it will be almost fully collapsed before the pressure during quiet inspiration is on the positive side of zero; but when the lung has undergone fibrous or pneumonic consolidation, or is filled with masses of caseating tubercle, the intrapleural pressure will give a positive reading some time before the collapse of the lung is complete. A reading taken, however, a few days later will show that the pressure is again a negative one. This is due not so much to the absorption of nitrogen, which is too slow to produce such a change, but to the accommodation probably of the intrathoracic structures, and to the yielding of the resistance in the lung. As the pressure increases and the lung collapses, the movements of that organ during respiration become less and less, till finally the lung is in a condition of complete immobility.

Other changes have necessarily been occurring at the same time. Not only the alveoli, but the bronchi and bronchioles also, have participated in the collapse, forcing out any contained secretions and preventing their re-accumulation. Cavities, when present, are obliterated by the approximation of their walls, and their contents are expelled. It follows naturally from this that if there is a vessel in the cavity which is eroded, the haemorrhage from that vessel will be arrested by the apposition and pressure of the surrounding tissues.

A consideration of these facts makes it at once evident that nitrogen displacement must be considered in the treatment of diseases such as phthisis, bronchiectasis, and putrid bronchitis.

NITROGEN DISPLACEMENT IN THE TREATMENT OF PULMONARY TUBERCULOSIS.

1. The Choice of Case.

Many people are of opinion that nitrogen displacement should be reserved as the last resort in the treatment of tuberculosis, an opinion formed probably from an erroneous impression that the collapse of the lung is permanent. This is a great misfortune, as it deprives the patients of the treatment while they are in a position to benefit by it, and tends also to bring discredit to the method. No case, so long as it is progressive, can be regarded as too early for treatment by nitrogen displacement. The acute early lesion will respond much more readily than the old fibrotic lung, and the proportion of failures in finding a free pleural cavity will be considerably smaller. The course that I would strongly urge is, that every case of well defined or suspected pulmonary tuberculosis should be radiographed as a routine procedure when first seen. If the case is then suitable for nitrogen displacement, the patient may receive medical treatment for the maximum period of three months. At the end of three months, at latest, the patient must be again radiographed and a comparison made of the plates and the clinical findings with those observed before treatment began. Unless the patient shows very definite evidences of improvement, the nitrogen displacement should be immediately attempted. Should it be suspected at any time during that period of three months that the disease is progressing, a second radiogram must be at once taken, and, if this is confirmative, the displacement should no longer be deferred. A sudden haemoptysis must be regarded, from the point of view of treatment, as evidence that the disease is progressive.

The disease need not necessarily be confined to one lung, although such cases are more satisfactory to treat. Provided that there is sufficient healthy lung tissue on the opposite side (about two-thirds) to carry on the functions of respiration, nitrogen displacement may be produced (Fig. 1). There are three types of cases, however, in which this form of treatment is particularly suitable:

1. In which there is a persistent rise of temperature.
2. In which there is profuse or repeated haemorrhage.
3. In which there is cavity formation in one lung.

Acute miliary tuberculosis when confined to one lung is no contraindication.

I have seen so many cases in which there has been a peribronchial infiltration which could not be detected by clinical examination, that I must once more emphasize the necessity of a radiogram being taken to determine the condition of the less affected side before treatment by displacement is begun. The radiogram has the advantage, in addition, of affording an accurate record for reference; it gives the details of the pathological changes in the deeper as well as in the more superficial parts of the lung, and allows of these changes being studied at leisure; while it not infrequently enables one to suspect the presence of pleural adhesions by showing deformities of the diaphragm.

2. Technique.

It is advisable that the patient should be kept in bed a week before the treatment is begun, except, of course, when the displacement is urgently necessary to check a haemorrhage. The reason for this is that it is advantageous to obtain a clear knowledge of the variations in the temperature and the rate of the pulse when at rest, and the amount also of the expectoration.

I make it a routine practice now to give a hypodermic injection of $\frac{1}{4}$ grain of morphine a quarter of an hour before the first attempt at finding the pleural cavity is made. This I consider necessary, as it may be very difficult to find the potential space between the visceral and parietal pleura and more than one puncture may be necessary. Morphine and the anaesthetization of the track of the needle, which latter is an absolutely essential part of the procedure, are the only sure means of averting the danger of a pleural reflex, which will be discussed later. The most suitable site for puncturing the chest wall is in the fifth, sixth, or seventh intercostal space, in the mid or anterior axillary line, in front of the anterior border of the latissimus dorsi, and below and behind the inferior border of the pectoralis major. If a free pleural cavity cannot be found in this region attempts may be made in the intercostal spaces on the front of the chest as high as the second or behind as high as the fifth. When the radiogram shows that one part of the lung is less implicated by the disease the attempt should be made over that area, if the puncture in the region first mentioned has failed to find the pleural cavity. The area selected should be painted with iodine.

The anaesthetization of the track of the needle is very simple. A Record hypodermic syringe and a needle, preferably 3 cm. long (it must be at least 2.5 cm.) and 10 c.cm. of 2 per cent. sterilized novocain are required. The needle is driven into the substance of the skin at the site of the proposed puncture, and a circular area 1 cm. in diameter is infiltrated with the novocain. The needle is withdrawn, and then driven vertically into the skin through the centre of the infiltrated area, and pushed steadily inwards along the upper border of a rib to the pleura, novocain being all the time forced into the tissues. With practice the passage of the needle through the various layers of fascia and muscle can be recognized. The progress of the needle is momentarily arrested immediately after traversing the internal intercostal muscle and an extra quantity of novocain expelled from the syringe. The needle is then advanced, and if the patient feels the point as it touches the pleura a further quantity of novocain must be injected, so as to ensure the complete anaesthetization of that membrane. The needle is withdrawn and a small puncture made through the skin with a double-edged tenotome. This will do away with the necessity of forcing the needle through the skin and the danger of conveying organisms with the needle from the skin to the pleural cavity.

The needle for introducing the nitrogen is connected with the apparatus, and some gas is run through into a bowl of water to make sure that all is in working order. The needle is put in direct connexion with the water manometer, and then slowly forced through the chest wall until it reaches the pleural cavity. I use a stilette in the needle only when there is some oozing of blood from the skin puncture, but always withdraw this while the point of the needle is still embedded in the intercostal muscles. The moment the point of the needle has reached the pleural cavity the manometer will indicate the presence of a negative pressure equal to about 7 mm. of mercury, and will show oscillations corresponding with expiration

and inspiration—the difference in pressure being equal to about 1 mm. of mercury (13.9 mm. of water). A long inspiration will produce a still further negative pressure, while a cough will considerably diminish the negative and may produce a positive pressure. In either case, however, the fluid in the manometer will rapidly return to about its former level. Should the needle have penetrated the visceral pleura and entered the lung, a negative pressure will still be registered by the manometer, but it will be of less degree, and the regular respiratory oscillations will be absent. A long inspiration may increase the negative pressure, and in such a case the fluid in the manometer will not return to its previous level. Occasionally, when the needle is pressing against the surface of the parietal pleura, very slight respiratory variations will be registered by the manometer. On most of the occasions that I have noticed this, the two pleural membranes were adherent.

As soon as the operator is satisfied that the needle is in the correct position, 100 c.cm. of oxygen are allowed slowly to run into the pleural cavity, and if this has been effected

easily without producing any marked variation in the pressure, 100 c.cm. of nitrogen are run in. The intrapleural pressure is noted, the needle withdrawn, and the small wound covered with a piece of zinc oxide plaster. This plaster must be removed on the next day, else the scab will remain moist, and will suppurate. The usual effect of the first injection at any rate, and sometimes of the subsequent ones, is to produce a rise of temperature and an increase in the pulse-rate. If only 200 c.cm. of gas have been run in on the first occasion the reaction is rarely serious, the temperature returns to its previous level in two or three days, and two days after this has occurred the

second injection of gas can be given. On this occasion 350 c.cm. of nitrogen are run in, and if there is no reaction the subsequent injections can be made at intervals of two days—500 c.cm. of gas being introduced on the third day and 750 c.cm. on the fourth and following days until the lung is completely displaced and so long as the pressure during inspiration does not rise above *plus* 1 (mm. of mercury).

The average pleural cavity will contain about 3,500 c.cm. of gas, but it is impossible to know when the lung is completely displaced except by means of the *x* rays. If the lung is not extensively diseased, a pressure varying from *plus* 1 to *plus* 3 (mm. of mercury) will indicate complete collapse, but if the lung is consolidated, or is very fibrous, this pressure may be reached with the lung only three parts or even less displaced, even when no adhesions are present. In such cases it will be found that after an interval of four or five days the intrapleural pressure will be again negative, and a considerable quantity of gas can be run in before the inspiratory pressure crosses the zero. (Fig. 5.)

The presence of adhesions between the pleural membranes, insufficient however to obliterate the cavity, converts the simple procedure above described into a very complicated one. The difficulties which arise in each individual case vary with the character and extent of the adhesions. It is possible for me to describe three variations only: (1) It is occasionally found that no dense adhesions exist, but that the two membranes are

Fig. 5.—Charts showing variations of intrapleural pressures during nitrogen displacement. The record in the column *a* is the pressure before, and the record in column *b* is the pressure after running in the nitrogen, the amount of gas introduced on each occasion being shown below. A, Pressure chart of L. S. (see Fig. 6); B, pressure chart of a patient in whom displacement was impossible on account of extensive adhesions.

universally, though not firmly, adherent to each other. The finding of the pleural cavity is a matter of great difficulty, and when found the pressure rises rapidly the moment the gas is introduced. When, however, it is equal to about 5 to 8 mm. of mercury, the gas continues to flow without causing any increase of pressure. Complete displacement can usually be effected. (2) In some cases it would appear that adhesions are present but stretch readily, or that owing to adhesions the various parts of the pleural cavity are in imperfect communication with each other. The pressure rises rapidly, but if a pause of five to ten minutes is made whenever the pressure is equal to *plus* 10 (mm. of mercury), the pressure will gradually fall, and with patience a considerable quantity of gas can be introduced on each occasion. It will probably be impossible to obtain complete displacement. (3) In other cases there are extensive and dense adhesions. When about 50 c.cm. of gas only have been introduced, the pressure may have reached as high as *plus* 10 and shows no tendency to drop. This is an indication that it is useless to persevere.

Displacement by nitrogen when required for the arrest of haemorrhage must be much more rapidly produced; 500 c.cm. are run in on the first occasion, and 750 to 1,000 c.cm. on each of the succeeding days. If there is much reaction and the haemorrhage has ceased after the first injection, the interval between the subsequent ones may be prolonged and the amount run in modified.

In all cases the patient should be kept in bed during the whole process of displacement, as if he is allowed to get up before the lung is completely collapsed there may be disturbing variations in temperature.

Brauer advocates the exposure of the pleura through an incision, and the puncture of the pleura only, by the needle. The object of this is to guard against wounding the lung. Compared with the other method, however, it is unnecessarily complicated. I have tried it on one occasion, when I was unable to find the pleural cavity by direct puncture, but it only proved to me that the pleural membranes were adherent.

3. Complications.

The two serious complications which have been responsible for almost all the fatal cases referred to at the beginning of this paper are pleural reflex and gas embolism. Pleural reflex or pleural syncope is a condition which may be described as one of acute shock, and the symptoms may appear at the moment when the unanaesthetized pleura is punctured, as a result of the rupture of adhesions, or even from the rapid production of too great a positive pressure. There are certain cases when a higher pressure would enable one by rupturing adhesions to produce a displacement. This is justifiable only when the treatment by displacement is a last resort, and the relations of the patient fully understand the danger of the procedure.

I have punctured the pleura on over 200 occasions, but I have only once seen the phenomenon, and that was in one of my earlier cases before I fully realized the importance of anaesthetizing the parietal pleura. The symptoms were fortunately of a very brief duration, and although I injected gas into that patient on several subsequent occasions, the reflex did not occur. The symptoms observed were sudden marked pallor, complete disappearance of the pulse at the wrist, extremely shallow rapid breathing, and momentary unconsciousness.

Gas embolism is due to the escape of gas into one of the veins of the lung, as a result either of puncture of the vein by the needle, or the tearing of the lung during the rupture of an adhesion and the exposure of an opened vein to the gas at high pressure. The precautions necessary to prevent the occurrence of these two dangerous complications may be repeated; they are: The administration of morphine before the first injection; the anaesthetization of the track of the needle before every injection; the observation of regular oscillations in the manometer before any gas is run in; the introduction of oxygen at the first injection before that of nitrogen; the interruption of the flow of nitrogen whenever the intrapleural tension reaches a positive pressure equal to 10 mm. of mercury.

A pleural effusion develops in about 30 per cent. of all cases treated by nitrogen displacement. It may occur during the displacement, but more frequently during the first few weeks after the displacement is complete. The effusion is rarely excessive and often gives rise to no

symptoms. It should always be suspected if during the production of the displacement the manometer reading shows a higher pressure* than was registered at the end of the previous injection. The effusion is a serious complication only when it develops within a week or two of the displacement, as the increase in pressure may then be sufficient to produce respiratory and cardiac embarrassment; at other times it is of no consequence provided it produces no symptoms, as the fluid helps to keep up the displacement of the lung and will eventually be absorbed.

If at the beginning the lung is too rapidly displaced, there may be a severe reaction owing to the excessive autoinoculation. This will be indicated by the temperature rising and remaining high, and may possibly end in an acute dissemination of the tubercle.

Two of my patients, both highly nervous women, have complained of pain in the upper part of the chest a few hours after the final injection of nitrogen required to produce complete displacement. The pain, which appeared to be severe, subsided after two or three hours, and on neither occasion, either by clinical or radiographic examination, could I detect any cause for it. In each case, however, the patient cried and the spasmodic movements of the chest increased the intrapleural pressure and led to an attack of dyspnoea, which passed off as soon as she became less agitated.

The great increase in physical well-being which a patient who has perhaps been confined to bed for a considerable time experiences when the affected lung has been completely displaced must not be allowed to obscure the dangers associated with a secondary myocardial degeneration.

4. Maintenance of the Displacement.

My experience is that the nitrogen is absorbed from the pleural cavity much more quickly during the earlier than during the later months of treatment, and it is essential therefore that the first few refills should be done at more frequent intervals than the subsequent ones. During the first six months of the treatment I run in a fresh supply of nitrogen every six weeks, but after that I allow an interval of eight weeks to elapse. There is no necessity for the patient to be confined to bed at these times, but I make him lie down for three or four hours after the injection. Those who are at work are allowed to return to it the next day, and suffer little or no inconvenience from the increase in pressure. The amount of nitrogen required to replace that which has been absorbed varies from 500 to 1,000 c.cm. At the end of a year I allow the lung to re-expand so as to be able to make fresh clinical and radiographical observations, but as a rule it is advisable to maintain the displacement for at least eighteen months. If, during the earlier period of time, the lung is allowed to expand and the two pleural membranes to come in contact with each other, there is considerable risk that they will become adherent.

5. The Immediate and Later Results.

The immediate results are the disappearance of the fever, the diminution or even disappearance of the cough and expectoration, the lessening of the pulse-rate, the improvement in the appetite and of the general condition, and the consequent increase in weight. If haemoptysis has been a troublesome symptom that will also cease. (See Figs. 6 and 7.)

A patient who has not been confined to bed before this treatment was begun may be allowed up on the third day after the last injection of nitrogen, and provided his general and physical condition is satisfactory, there is no reason why he should not resume work after three to six weeks' convalescence, during which time bracing air, nutritious food, and a steadily increasing amount of exercise are advisable.

The later results depend naturally, to a great extent, on the amount of lung affected by the disease. While the beneficial influence of the nitrogen displacement is most striking on the side which has been treated, there is no doubt whatever that a tuberculous lesion, when present, of the other lung will be, in certain cases, materially benefited also. It is even possible to forecast with a certain degree of accuracy those cases in which both lungs will

* That is a pressure which is less negative or more positive.

improve under the treatment, one only being displaced. When the tuberculous lesion of the second lung is a true peribronchial infiltration, more or less limited to the region of the hilum, it is probably an infection secondary to the

Fig. 6.—Temperature chart showing the effect of nitrogen displacement. Nitrogen was introduced at the points marked A, B, C, D, E, F, and G. After G the displacement was complete. At X and Y the patient was allowed up for radiographic examination. It will be noticed that the rise of temperature which followed on each occasion did not occur when the patient was allowed up at Z and on the following days, when the displacement was complete. Note the diminution in the amount of sputum and in the pulse-rate.

primary lesion in the lung which is displaced, and improvement may be hoped for on both sides, but when the undisplaced lung contains tuberculous foci scattered over or extending through part of one lobe, and specially if

Fig. 7.—Temperature chart showing the influence of nitrogen displacement on the temperature. This patient had had constant fever, ranging from 99° to 103°, during the preceding three months. Nitrogen was introduced at A, B, C, D, etc.

there is considerable fibrosis, no such definite improvement must be expected. In such cases there is no objection, once the tubercle on the one side has been arrested and the lung allowed to re-expand, to displacing the second lung while the former resumes the functions of respiration.

6. The Value of Other Forms of Treatment in Conjunction with Displacement.

The patients of whom I have shown you radiograms and charts this evening may be divided broadly into two groups—those whom I have treated at University College Hospital, and those whom I have either in part treated or observed under treatment at the Pinewood Sanatorium. For the opportunity of dealing with the majority of the patients who come in the first group I am indebted to Dr. Halliday Sutherland, Dr. Pitt, and Dr. Harvey, and these patients have all had the tuberculin treatment continued after the displacement of the lung has been effected.

The patients at the Pinewood Sanatorium have been under the care of Dr. Etlinger, who has himself been carrying on the work of nitrogen displacement with most encouraging results. To none of these patients has Dr. Etlinger given tuberculin after the displacement of the lung by nitrogen. I am indebted to him for allowing me to make use of one of his charts.

(a) Nitrogen Displacement in Conjunction with Tuberculin Treatment.

On this subject I do not consider myself competent to offer either suggestion or criticism, as my experience is much too limited. The early results in the two above-mentioned groups of patients have been equally gratifying. It has been found, moreover, that if a patient is extremely sensitive to tuberculin, he will be much more tolerant after the lung has been displaced.

(b) Nitrogen Displacement in Conjunction with Sanatorium Treatment.

The term "sanatorium treatment" may at the present day mean much or little, but when "sanatorium treatment" means treatment in a sanatorium with fresh air, good food, exercises, and efficient control, the combination is excellent.

(c) Nitrogen Displacement in Conjunction with Graduated Exercises.

A question that is often asked is, How can two such different forms of treatment as complete rest to the lung (which can only be obtained by immobilizing that organ by nitrogen displacement) and graduated exercises both be beneficial to patients suffering from pulmonary tuberculosis? There are two answers to this question. The first is, that a patient is never put on graduated exercises so long as the disease is sufficiently active to give rise to any definite fever; whereas the immobilization of the lung by nitrogen displacement is applicable equally to those patients who are febrile and to those who are afebrile. The second answer is the suggestion that the benefit derived from graduated exercises may to a very large extent be due to the improvement in the patient's general condition. Phthisis is a wasting disease, and we know that one of the signs of the progress of the disease is the constant loss of weight, and that, to check this wasting, substances like cod-liver oil are given. All health is associated with an interference with metabolism, and this leads to loss of weight. When the active cause of illness is controlled, the metabolism can be stimulated by exercise, but the exercise must be graduated to the capacity of the individual.

I am certain that in cases of pulmonary tuberculosis, once the disease is under control—as it undoubtedly is when nitrogen displacement has been done—the improvement in metabolism by graduated exercises is of the greatest benefit. When a patient, however, has been seriously ill, the ordinary forms of exercise—walking, digging, etc.—do not seem to me to be capable of really efficient graduation from the lowest to the highest scale,* and are not to be compared with the system introduced by Dr. Etlinger at the Pinewood Sanatorium. There the patient, beginning with a little gentle massage, passes on to the most elementary form of Swedish exercise, and proceeds thence through a carefully graduated series of exercises, increasing both in duration and in the energy required for the performance of them.

Partial Displacement.

It occasionally happens that, owing to the presence of adhesions, partial displacement only of the lung is possible. If the lung is reduced to about half its former bulk, this displacement should be maintained, as numerous cases have been recorded in which the partial immobilization has resulted in considerable benefit to the patient.

It must not be thought, because an adherent pleura prevents the introduction of nitrogen, that surgical possibilities in the treatment of pulmonary tuberculosis are at an end. There are several other surgical procedures whereby benefit can be obtained, but they are outside the scope of this paper.

Nitrogen Displacement in the Treatment of Bronchiectasis.

It was at one time hoped that nitrogen displacement might prove to be a satisfactory method of treatment for bronchiectasis, and this hope was encouraged by the amelioration in the condition of the patient immediately after treatment. While, however, the cough and the excessive sputum can be diminished and the fever

* Some exercises are carefully graduated—for example, carrying known weights known distances—but certain groups of muscles only can be brought into use in this form of labour, and the monotony of the performance must be very great.

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Fig. 1.—A radiogram of a patient with phthisis, showing a cavity in the right upper lobe and disease of the right middle and left lower lobes. A suitable case for treatment by nitrogen displacement on the right side.

Fig. 2.—E. B. Radiogram taken one day after 2,280 c.cm. of fluid had been drawn off by the ordinary methods of aspiration. The patient gives a history of empyema of seven years' duration.

Fig. 3.—E. B. Oxygen replacement. Radiogram taken a month later.

Fig. 4.—E. B. Oxygen replacement. Radiogram taken three months later. Note the approximation of the margin of the lung to the chest wall.

and stinking breath abated, these changes are present so long only as the displacement is maintained. It is clear, however, that, although no permanent good can be expected, the temporary diminution of the symptoms is of very great advantage, in that it will enable a patient who is exhausted by constant fever and cough and the continual formation of quantities of offensive sputum to improve considerably in his general health, and enable him to withstand the strain of the more severe operations, such as rib resection or the ligature of a branch of the pulmonary artery.

Nitrogen Displacement in the Treatment of Putrid Bronchitis.

Of the value of this form of treatment there is no practical evidence, but from our knowledge of the controlling influence of the nitrogen displacement on the pulmonary secretions, the fever, cough, etc., we have reasonable grounds to suppose that it might materially benefit a patient suffering from a unilateral putrid bronchitis.

OXYGEN REPLACEMENT.

Aspiration has been for so long a time the established treatment for serous effusions of the pleural cavity that the distress to the patient frequently witnessed towards the end and for some little time after the withdrawal of the fluid has come to be regarded as a more or less unavoidable incident in the procedure. Neither this distress nor the fear of the supervention of acute oedema of the lung is, however, the only objection to aspiration. If in a series of patients radiograms are taken before and after "tapping" a pleural effusion, it is very surprising and also disappointing to find how much fluid yet remains. Two radiograms will serve to illustrate this point. One was taken immediately before and the other twenty hours after the withdrawal of 1,000 c.cm. of fluid from the pleural cavity; yet the difference in the level of the fluid in the two cases is only one interspace. The aspiration in this case had been stopped because of the pain which had developed during the removal of the last 200 c.cm.

The proportion of the fluid which can be withdrawn by the ordinary method of aspiration varies to some extent with the size of the effusion. In a small effusion it is more, in a large effusion it is less, than 50 per cent. From the patient whose radiograms I have just shown you I withdrew by oxygen replacement all the fluid from his pleural cavity two days later. The amount was 1,500 c.cm. In another patient, also a chronic case, I withdrew by the same process 2,800 c.cm. two days after 2,280 c.cm. had been aspirated by the ordinary method.

As is well known, a large effusion causes not only displacement of the lung, but also displacement of the mediastinum and its contents, and there is no doubt that the products of inflammation (for example, thickening of the pleura and adhesions) tend partly, at any rate, to resist the return of these structures to their normal position. In a large effusion, as the fluid is aspirated the intrapleural pressure becomes gradually and increasingly negative. If the lung were capable of complete and

immediate expansion no symptoms would be produced until all the fluid had been withdrawn. Experience shows, however, that neither the lung nor the mediastinum is capable of rapid adjustment. The lung cannot expand quickly because of the infiltration and thickening of the visceral pleura; and if there is associated intrapleural disease and the displacement has been of some duration, because of the fibrosis; while the mediastinum is prevented from returning to its former position by the thickening of the pleura and the formation of adhesions. Although at first during the aspiration the change in the intrapleural pressure is slow, there soon comes a time when the intrathoracic organs can no longer compensate for the rapid loss of fluid. The intrapleural pressure now becomes rapidly and increasingly negative, and in response the inflamed or fibrosed tissues and adhesions are stretched in a passive attempt at further compensation. The results are pain, which may be extremely severe, cough, and even fatal oedema of the lungs.

It is obvious that if it is required to remove a pleural effusion some substitute must be found for the fluid, and that substitute must necessarily be one which can be readily absorbed, and cause by its absorption a steady yet not too rapid pull on the intrathoracic organs. The substance which answers all these requirements is oxygen. Oxygen is easily introduced into the pleural cavity. The rate and amount of flow can be under complete control. It is rapidly absorbed. This rapid absorption of oxygen does not, however, mean that if a large quantity has been run in, there will be no gas left in the pleural cavity at the end of a few days. If a radiogram is taken at the end of four days, it will be

seen that there is still some gas in the pleural cavity and possibly some slight reaccumulation of fluid, but the space occupied by these will be considerably less than that occupied by the oxygen immediately after the replacement, provided that the lung is not absolutely bound down by dense adhesions. If, further, the intrapleural pressure is taken it will be found to be still markedly negative and an examination of the intrapleural gas will reveal the presence of nitrogen and carbon dioxide only. These gases can be absorbed also but at a slower rate.

It is not my intention to describe in detail the technique of oxygen replacement—that I have already done in the *Lancet* of December 28th, 1912—but I would only remind you that the apparatus for introducing the oxygen is the same as that for introducing nitrogen; that the track of the two needles which are necessary should be anaesthetized by novocain, and that while the needle connected with the Potain's aspirator is entered into the lower part of the pleural cavity, that connected with the oxygen is entered some two or three spaces higher. The fluid is aspirated until the first symptoms of drag on the intrathoracic organs is noticed—discomfort, pain, or cough—when 100 c.cm. of oxygen are run in. The two processes are alternated until all the fluid is removed.

Oxygen replacement is not only of great value as a method of removing pleural effusions and exposing the

Fig. 8.—Chart showing variations of intrapleural pressure during oxygen replacement (a case of chronic empyema). The record in column a is the pressure before, and the record in column b is the pressure after withdrawing the fluid and replacing it by oxygen.

lung for radiographic purposes, but I believe it is the only efficient means we possess for the treatment of chronic cases of tuberculous pyopneumothorax. As the radiograms show, it is possible in these cases, by replacing the gas and fluid by oxygen, gradually to draw out the lung without producing any symptoms of distress or discomfort. The process must be repeated on three, four, or even five occasions at intervals of two or three weeks, leaving on each occasion an increasingly high negative pressure. If the lung is completely fixed by adhesions, it cannot be made to re-expand, but it will be found that the tendency for the fluid (at first pus, and then serum) to reaccumulate will rapidly diminish—a state of affairs which is greatly appreciated by the sufferer. One of my patients with a pyopneumothorax, who had been unable to work for five months owing to the discomfort and shortness of breath, occasioned apparently by the pus (which was sterile), has been at work for over a year since I treated him by this method. Although he has only one lung, he says he is inconvenienced by shortness of breath only when he has to climb a long ladder.

Two cases I would like to recount in greater detail.

CASE I.

A man, aged 24, developed, in January, 1913, acute pneumothorax, and was admitted to University College Hospital, under Sir John Rose Bradford, with a hydropneumothorax. The lung was collapsed against the mediastinum and the fluid reached the level of the angle of the scapula. At the beginning and at the end of June I withdrew the fluid by oxygen replacement, withdrawing on the first occasion 3,060 c.cm. and on the second 1,260 c.cm. When I saw the patient again in August there was some fluid but no gas in his chest. The upper part of the lung was expanding. I withdrew on this occasion by oxygen replacement 1,860 c.cm. of fluid, and an x ray taken immediately after showed that practically all the fluid had been removed, and that the lung had expanded still further. The patient returned to full work in October, and I was unable to see him until three weeks ago. The skiagram now shows complete expansion of the greater part of the lung, marked thickening of the pleura, and possibly a little fluid at the base.

CASE II.

A man, aged 39, seven years ago had an attack of pneumonia on the right side. After the attack had subsided, he was "tapped," according to his account, and pus was withdrawn from the right pleural cavity. He was again "tapped" on four subsequent occasions during the next two years, pus being obtained each time. When admitted to University College Hospital under the care of Dr. Bolton in August of last year, the right side of his chest was full of fluid. He was wasted, and suffered from breathlessness and general debility, but there was no cyanosis or clubbing of the fingers. He was aspirated on August 30th and 2,280 c.cm. of fluid drawn off. This fluid was thick and yellow, in appearance resembling pus. It was found to consist of masses of epithelial cells, pus cells, and lymphocytes, and contained two lipoids. It was, however, sterile. The following day this radiogram was taken (Fig. 2). On September 1st I removed by oxygen replacement 2,800 c.cm. of a fluid similar in character. The variations in pressure during the replacement are shown in the chart. At the end the needle became blocked by a solid plug, and I was in consequence unable to withdraw more fluid. This plug, I discovered later by radiographic examination, came from a mass of practically solid debris at the base of the cavity. On September 4th I withdrew 485 c.cm. of fluid; the negative pressure at the end was equal to 29 mm. of mercury, which was tolerated without pain. At the end of the month I withdrew a further 1,950 c.cm. of fluid, again by oxygen replacement, the negative pressure at the end being equal to 28 mm. of mercury. The radiogram taken after this showed that the lung was beginning to expand (Fig. 3). On October 6th, by the same method, 1,485 c.cm. of fluid were drawn off, and a negative pressure left in the pleura equal to 36 mm. of mercury. The fluid, which on the previous occasion had been somewhat less opaque, was now much more definitely serous in character, and the proportion of cells was strikingly less. On November 30th, after 1,455 c.cm. of fluid had been aspirated by oxygen replacement, the negative pressure left was equal to 40 mm. of mercury. The fluid had the appearance of an ordinary serous effusion, and the expansion of the lung was considerably increased. On January 4th of this year, after again an interval of five weeks, I could withdraw only 950 c.cm. of fluid; the intrapleural pressure was at the beginning 17, and at the end 42 mm. of mercury. The radiogram (Fig. 4) shows a further increase in the expansion of the lung, and it can be seen, in addition, that the high negative pressure in the pleural cavity is causing some displacement of the mediastinum. That this displacement of the mediastinum is not injurious is shown by the fact that the patient was able to return to work four months ago, and has continued without intermission since then. Further, Dr. Lewis, who has kindly taken electro-cardiographic tracings, tells me that there is no evidence of any impairment in the heart's action. By February 16th less than 600 c.cm. of fluid had reaccumulated,

and I again withdrew this by oxygen replacement. On this occasion I obtained a negative pressure as high as 54 mm. of mercury without producing pain, and left him with a pressure of -50. At the end the lung was drawn out to the lateral wall of the chest, and could be felt with the point of the needle. The improvement in the man's general health is most gratifying. He has lost his breathlessness, has put on flesh, and says he now feels perfectly strong and well.

It is very surprising with what comfort the patient tolerates the high negative intrapleural pressure, and suffers apparently no inconvenience from it (Fig. 8). The only precaution that I have taken with him on the last four occasions has been to keep him in bed during the day on which the replacement is done. He gets up the next morning and returns to his home at Cambridge, resuming work the following day.

I believe that there are still other intrapleural conditions which can be treated by this simple process, and I hope in the future to be able to show that it is a satisfactory substitute for the open operation for certain types of acute empyema, especially the post-pneumonic in children.

THE NEED FOR RESEARCH IN ANTE-NATAL PATHOLOGY.*

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OBSTETRICIANS have recently been considering whether more cannot be done to lessen the enormous loss of life which goes on during gestation.

If I am right in my estimate¹ that there are about four times as many abortions as stillbirths (that is, 2.2 stillbirths and 8.8 abortions to 100 live births), it means that in England and Wales about 100,000 fertilized ova die annually before they are born, and that about as many infants die in their nine months of intrauterine life as during the survivors' first year of life. These estimates are much below those given by Priestley,² who said "there is one abortion to every three or four full term deliveries, and that from three to six out of every ten women abort at least once during their married life." Can this Section suggest how statistics regarding abortion can be obtained from a large series of average married women, so that the relation of abortions to live births can be definitely known? Even then there would be numerous unrecorded, because unrecognized, very early abortions.

We know very little of the causes of this terrible mortality. I have tabulated them roughly in the address to which I have already referred, but if my colleagues have been as lax as I have been in my past efforts to ascertain the causes of abortions or stillbirths, they will realize how many opportunities have been missed in not having the expelled products of conception systematically and scientifically examined with a view to discover the cause of death. Theoretically at all events, with the methods now at our disposal, we ought to be able to determine the cause of an intrauterine death as correctly as of an extrauterine death. Probably we are all agreed that a few of the abortions and premature births are due to primary uterine contractions, the fetus being normal. Maternal and perhaps paternal malnutrition may be a large etiological factor both as regards early abortions and births of sickly children. I understand that Dr. Darwall Smith has been investigating the subject of malnutrition, and will give us his conclusions. All the other spontaneous premature deliveries are due ultimately to some pathological condition of the fetus and its membranes, or of the maternal or fetal placenta.

DIFFERENCE BETWEEN EMBRYONIC AND FETAL PATHOLOGY.

Ante-natal disease and death must be considered in its early embryonic stages, as well as in its later or fetal stages. Ballantyne³ points out the great differences between the results of pathological activity in the embryo and in the fetus, and he shows that whilst in the fetus diseases are more or less of the same nature as the maladies of post-natal life, "in the embryo the morbid

* Read at a meeting of the Obstetrical and Gynaecological Section of the Royal Society of Medicine on April 2nd, 1914.