THE TREATMENT OF PULMONARY TUBERCULOSIS BY NITROGEN COMPRESSION.

[WITH SKIAGRAMS ON SPECIAL PLATE.]

BY

GEOFFREY LUCAS, B.A.CANTAB., L.S.A.LOND.,

SENIOR ASSISTANT PHYSICIAN, NORDRACH-ON-DEE SANATORIUM,
BANCHORY.

So much has been written in this and other journals on the technique of the operation of nitrogen compression for pulmonary tuberculosis that I propose only to touch upon a few practical points of importance before proceeding to a recitation of the illustrative cases.

With regard to the preparation of the patient for operation, when possible I prefer the patient to be at absolute rest in bed for two days previously. On the second evening an aperient is given, and if the bowels have not acted satisfactorily, a simple enema is given early the following morning. The chest is then carefully examined, and three likely sites for puncture are chosen and painted with tincture of iodine to the size of half a crown. Three-quarters of an hour before the time appointed for operation 0.5 c.cm. of omnopon-scopolamine is given hypodermically. Half an hour later, if the patient is not well under the influence of the drug, a further dose of 0.25 to 0.5 c.cm. is given. This procedure is found most beneficial in diminishing reflex sensibility, whilst the rate and depth of respiration is not materially affected. The proposed site of operation is again painted with tincture of iodine and a hypodermic syringe containing 15 minims of a solution of novocaine and adrenalin is taken, and the needle plunged through the skin at right angles into the intercostal space. It is pushed onwards gently until it is judged to have reached the parietal pleura (this can often be felt by a sudden feeling of resistance to the needle). Two or three minims of the anaesthetic are next injected, and the needle is gradually withdrawn, injecting slowly the while. In this way a track is anaesthetized along which the Saugmann's pneumothorax needle is subsequently passed. It is a practical point of some importance that no excess of anaesthetic fluid should be left immediately under the skin, as if it is there is a probability that the Saugmann's needle may be blocked and the manometric oscillations consequently interfered with. For the same reason it is of great importance that the Saugmann's needle should be absolutely dry, and for this purpose it is my practice to keep the needle in absolute alcohol until required for use. When taken out of the alcohol it is thoroughly dried over the flame of a spirit lamp, and hot air is blown through the needle by means of a small air bellows attached to the proximal end. At the first operation I prefer to use a needle with an opening at the end, but when a potential space has been established between the parietal and visceral layers of pleura I use a needle with a solid point and a lateral aperture at the end of the shank immediately above the point.

With reference to the amount of nitrogen to be introduced and the alteration of intrapleural pressure to be brought about at the first sitting, some diversity of opinion exists. The two principal factors governing this question are—firstly, the presence or absence of pleural adhesions, and, secondly, the size of the chest. A point to remember is that one can proceed more rapidly with safety on the right than on the left side of the chest, as the normal position of the heart is less interfered with by introducing gas into the right than the left pleural sac. The use of *x* rays is of inestimable value in affording information as to the relative position of the intrathoracic organs, as percussion of the cardiac area becomes increasingly difficult and unreliable as the introduction of nitrogen proceeds. It has been my practice, in cases free from adhesions, to aim at producing a slight positive pressure (2 to 3 cm. of water) at the third of three successive sittings, which have taken place at intervals of a day between each. I have found that in chests of average capacity three separate injections of about 500 c.cm. of nitrogen each will bring about this result.

SELECTION OF CASES.

As regards the selection of cases for treatment, it is desirable, though not essential, that one lung should be sound. In the event of both lungs being affected, there must, of course, be sufficient healthy substance in the "sound" lung to carry on the functions of respiration. It is quite astonishing how little apparently healthy lung is necessary for this purpose. Fig. 1 well illustrates this point; it will be seen from this skiagram that an artificial pneumothorax has been produced on the right side. The left lung is largely involved by fibroid phthisis. Yet this patient has been able to walk from ten to twelve miles a day, and has just indulged in a winter's hunting.

It was formerly thought that by producing a pneumothorax in a bilateral case with quiescent limited disease on the "sound" side one ran a great risk of lighting up the disease afresh by throwing additional work on the "sound" side. This has not proved to be the case in practice. Indeed, if the pathological state of affairs is considered, it is seen that rather than exciting the disease to fresh activity one is in reality bringing about a condition of affairs which is conducive to arrest. When one lung is extensively involved and the other slightly so, a considerable amount of compensatory emphysema takes place in the "sound" lung. The alveoli are enlarged and the capillaries dilated; subsequently the capillaries become thrombosed and atrophied and *post mortem* the lung presents a swollen and anaemic appearance. Now when an artificial pneumothorax is produced the emphysematous lung is subjected to pressure, and consequently compression is brought to bear on the vessels and some obstruction to the circulation in that lung is produced. This in turn produces venous stasis, a condition which obtains, but to a much more marked degree, in a case of mitral stenosis. Thus there is produced in the "sound" lung the essentials aimed at in Bier's treatment of tuberculosis by passive hyperaemia, a condition of the lung which tends to bring about arrest rather than excitation of the disease. The radiographic appearance of an emphysematous lung is well illustrated in Fig. 2, while Fig. 3, from the same case, taken after the production of an artificial pneumothorax on the left side, shows the compression to which the right lung has been subjected.

The position taken up by the collapsed lung is naturally dependent on the presence or absence of adhesions in the pleura. Figs. 5 and 6 well illustrate two common positions taken up by the lung in the absence of adhesions. Fig. 5 shows the lung collapsed in the shape of a ball round the pulmonary root, whilst in Fig. 6 it is seen lying along the bodies of the vertebrae.

The sudden drop of temperature claimed to be brought about by the production of an artificial pneumothorax in pyrexial cases has only occurred once in my practice (Chart A). I have found in my cases that the temperature usually for two or three weeks after the operation remains on much the same level as before, with frequently a tendency to a lowering of the morning temperature. After this period it gradually subsides, with an occasional temporary exacerbation, and by the end of six weeks or so the temperature has once more assumed a normal curve. In nearly all my cases there has been an immediate and permanent drop in the daily amount of expectoration, after an initial rise of three or four days' duration due to the squeezing out of secretion from the compressed lung.

There is one other point, but by no means the least in practical importance. It is never possible to predict before making the attempt whether or not one will be able to produce an artificial pneumothorax. Whilst I have failed after repeated attempts in cases which to the ordinary methods of investigation seemed to be entirely free from adhesions, I have likewise failed in cases in which I regarded such a possibility as remote. The moral of this is not to be too encouraging to the patient as to the results to be attained by this method of treatment, as if one is too optimistic and fails in the performance of the operation on account of adhesions, the disappointment and sense of depression produced by failure will require all the tact and ingenuity at one's command to counteract the ill effects produced and prevent a detrimental influence being brought to bear on the progress of the disease.

I will now relate a few selected cases, each of which presents some points of interest.

CASE I.

A young lady, aged 22, who developed phthisis in the summer of 1911. Lassitude and loss of weight were the initial symptoms. She was admitted to this sanatorium on October 11th, 1911. The condition then present was infiltration, with crepitations, over the whole of the right upper lobe and apex of the right lower lobe. Left lung healthy, tubercle bacilli abundant in sputum. Improvement took place during the winter of 1911-12, and in June, 1912, the patient returned to her home to continue treatment under home conditions for the summer months. She returned in October with a recrudescence in the right lung. There was also evidence of the disease having com-

CHART A.—Case I. B = 500 c.cm.; F = 600 c.cm.; G = 750 c.cm. N.

menced in the left lower lobe. She remained under treatment during the winter of 1912-13. The disease on the left side became arrested, and improvement took place in the right lung. During the summer of 1913 the patient was able to take exercise to the extent of walking four miles daily, but in December an exacerbation of the disease occurred in the right lung, accompanied by pyrexia, sweating, and loss of weight. Matters did not improve. Accordingly, at the beginning of March, 1914, it was decided to attempt collapse of the right lung by means of artificial pneumothorax. On March 5th the intrapleural space was found showing a pressure of -16-10 cm. of water; 600 c.cm. of nitrogen were introduced, raising the pressure to -4-0. Refills were given on subsequent days (vide Chart A), until on March 10th the intrapleural pressure had been raised to +12+7. Physical examination of the chest revealed the total absence of breath sounds on the right side, and a skiagram taken the following day (Fig. 5) shows the lung well collapsed. During this period the temperature and pulse-rate had both fallen to within normal limits.

On March 16th the patient was allowed to sit up in bed for an hour. On March 18th a further refill was given, and the following day the patient was allowed to sit up in a chair for two hours. All seemed to be going on well, and a successful result from the treatment was anticipated. During the night of March 21st-22nd the patient complained of restlessness and

CASE II.

A man, aged 34, weighing 13½ st. and standing 6 ft. 2 in., was admitted on February 13th, 1914, with a history of hæmoptysis six weeks previously. Examination of the chest showed a limited lesion in the right upper lobe with evidence of softening. The left lung was normal. As the temperature was slightly raised, the patient was kept at rest in bed. Cough was a prominent symptom, accompanied by muco-purulent sputum, to the extent of 150 c.cm., daily. Ten days after admission the temperature began to rise steadily (Chart B), sweating at night commenced, and the patient soon became acutely ill. Auscultation of the chest revealed evidence of increased activity in the right upper lobe.

This state of affairs continued without much change for a fortnight, when, on March 13th, a severe hæmorrhage occurred, which continued at intervals for the next two days, in spite of

treatment on the usual lines. A particularly severe hæmorrhage occurred on March 15th, and as the patient's condition was rapidly deteriorating and the hæmorrhage showed no signs of abating, it was decided to attempt the production of an artificial pneumothorax as a means of checking the hæmorrhage by pressure; 1,000 c.cm. of nitrogen were introduced at the first sitting, raising the intrapleural pressure from -10 to -2 cm. of water. This had the effect of controlling the hæmorrhage to a great extent, but as bright red expectoration persisted the following morning, a further 1,000 c.cm. of nitrogen were introduced, raising the intrapleural pressure to +4. This had the desired effect of stopping the bleeding. The patient showed no signs of distress as the result of these two large injections of nitrogen, and, as the disease appeared to be running such an acute course, it was decided to continue the treatment and produce a complete pneumothorax. Accordingly refills of 500, 950, and 1,000 c.cm. were given within the next five days (vide Chart B), and the intrapleural pressure was eventually raised to +18 cm. of water, at which it has been kept by periodical refills for upwards of a year. The progress of this case has been most satisfactory. The temperature and pulse-rate quickly dropped, and the patient was soon able to be up and about, and has been taking daily walking exercise to the extent of six miles without fatigue, whilst the physical signs and symptoms indicate quiescence of the disease, and it

CHART B.—Case II. A = 1,000 c.cm. N; B = 500 c.cm. N; C = 950 c.cm. N; D = 1,000 c.cm. N; E = 900 c.cm. N.

increasing shortness of breath. Some cyanosis was present, and the pulse-rate had increased to 120. Examination of the chest revealed a diffuse commencing pneumonia of the left lung, fine crepitations being audible from apex to base. The dyspnoea rapidly increased, and as the patient's condition was rapidly becoming critical I decided to release the right lung in an attempt to give the patient more aerating surface with which to carry on respiration and also to relieve any mechanical embarrassment of the heart. Accordingly a Saugmann's needle was introduced, one limb of which was connected by rubber tubing to a Potain's aspirator pump, the other to the manometer. The clip was released, and the tap shut. The nitrogen was in this way extracted from the right side of the chest. Readings were taken on the manometer between every two or three strokes of the pump by clipping and opening the tap. The intrapleural pressure was reduced until a negative pressure of -4-2 was produced. During these operations the pulse was not materially affected, and no additional distress was manifest. The Saugmann needle was withdrawn and the chest auscultated. The breath sounds were immediately audible over the whole of the right lung, and in character were much the same as before the initial collapse sixteen days previously. Unfortunately the hope of improvement entertained was not realized, and the patient died from heart failure twenty-four hours later in spite of the free use of oxygen.

The points of interest in this case are: First, the almost immediate improvement in the temperature and pulse-rate subsequent to the collapse of the diseased right lung (Chart A). Secondly, the performance of the operation for decompression under conditions of emergency; and, lastly, the rapidity with which the collapsed lung expanded under the release of the pressure.

It is hoped that ultimate permanent arrest will be obtained. In this case there are evidently some adhesions on the posterior surface of the upper lobe, as, although breath sounds were inaudible in front, there was evidence of feeble tubular breathing over a small area behind. This is borne out by the appearance in the skiagram taken but not shown in this paper. The case is interesting as showing the value of this form of treatment in cases of alarming and uncontrollable hæmorrhage.

CASE III.

A young man developed phthisis in the left lung in January, 1910, at the age of 26. He received sanatorium treatment for a period of six months, during which time considerable improvement took place, and in the autumn of the same year he was recommended to take up residence in Australia. He spent four years abroad, during which time he had many ups and downs, and towards the end of that period had several severe hæmoptyses. He returned to England in 1914, and on the voyage home had a severe hæmorrhage on board ship. In November, 1914, he was sent to this sanatorium by a London physician with the request that an attempt should be made to produce an artificial pneumothorax, though the physician did not hold out much hope of a successful issue on account of the duration of the disease and the undoubted presence of pleural adhesions. On examination it was found that the left lung was involved throughout by tuberculous disease, with evidence of cavitation in the upper lobe. The right lung showed evidence of a quiescent lesion at the apex of the upper lobe. Fig. 2 shows the radiographic appearance on admission—extensive left-sided disease with much compensatory emphysema in the right lower lobe. On November 10th three attempts were made, at different sites, to find a free pleural space, but without success. On November 12th a further trial was made, and at the second

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FIG. 1.—Showing a complete pneumothorax on the right side with extensive disease on the left.

FIG. 2.—Shows Case III immediately before operation. Note the emphysematous condition of the right lung.

FIG. 3.—Shows Case III during the process of the production of complete pneumothorax. Note the altered appearance of the right lung owing to the compression of the left lung.

FIG. 4.—Shows Case III with the lung finally collapsed. Note the shadows of the two lines of attachment of the pleura to the chest wall—'a', to the anterior wall; 'b', to the posterior wall.

FIG. 5.

FIG. 6.

Figs. 5 and 6 show two common positions assumed by a collapsed lung free from adhesions.

attempt the needle encountered a free space in the sixth intercostal space in the posterior axillary line. The manometer oscillations were small but definitely negative -4 , -2 , accordingly 300 c.cm. of nitrogen were introduced, which had the effect of producing positive pressure of $+3$ $+1$. It was therefore obvious that one had entered a pleural sac. However, hope was entertained that one might be able to gradually break down pleural adhesions and eventually produce an effective pneumothorax. Frequent refills of small amounts were subsequently given until eventually a very satisfactory pneumothorax was produced without any pain or untoward symptoms. A pressure of $+18$ was reached and has been maintained. Figs. 2, 3, and 4 show three stages in the course of treatment. Fig. 3 shows extremely well the effects of compression of the left lung on the emphysematous right lung. A careful scrutiny of Fig. 4 shows that the left lung is partly bound down by adhesions to both the anterior and posterior chest wall. The faint line of shadow indicated by the arrow (a) marks the line of attachment to the anterior wall, whilst the arrow (b) shows the line of attachment to the posterior wall. The lung is evidently compressed inwards towards the mediastinum in the shape of a gutter between the two lines of adhesion. This case is at present doing well. The general condition is excellent, and the patient is living an ordinary life under home conditions, the compression being kept up by means of refills at varying intervals of approximately a month. The case is interesting as illustrating how even adhesions of long standing may be harmlessly broken down by the exercise of caution and perseverance in the treatment.

In conclusion I should like to express my indebtedness to Mr. T. R. Watson, radiographer to this sanatorium, for the production of the skiagrams illustrating this paper.

THE PATHOLOGY AND TREATMENT OF GUNSHOT WOUNDS OF THE SMALL INTESTINE.

By OWEN RICHARDS, M.Ch., F.R.C.S.,

PROFESSOR OF CLINICAL SURGERY, EGYPTIAN GOVERNMENT SCHOOL OF MEDICINE; TEMPORARY CAPTAIN R.A.M.C., BRITISH EXPEDITIONARY FORCE, FRANCE.

FOUR months' work in a casualty clearing station have given me the opportunity of observing a number of these cases, and though my experience is not wide enough to warrant any final conclusions, yet some points have struck me which may be of interest to others who have to deal with cases of this class.

PATHOLOGY.

Whereas in wounds of the colon and duodenum the chief feature is the progressive escape of contents followed by a more or less localized peritonitis, in the small intestine such an escape usually does not occur at all, and if it does is so slight as not to be an essential feature in the case. The cause of death in wounds of the small intestine is more frequently intestinal obstruction due to paralysis and distension spreading upwards from the injured coil. To make this contrast clear, I have appended notes of examples of cases of both classes.

In Case I there was a wound of the colon; the chief symptoms were general distension and localized pain. On opening the abdomen a large collection of faeces was found localized under pressure in a cavity formed by adherent intestines. Faeces continued to be discharged from this wound for five days.

Similarly, when the colon is wounded extraperitoneally, faeces are expelled under pressure into the surrounding connective tissue spaces to an almost unlimited extent. I have seen a wound of the colon on the left side (verified *post mortem*) cause faecal infiltration extending into the right loin and discolouring the skin on the right side of the abdomen before death.

In Cases II and III similar localized collections of intestinal contents under pressure followed wounds of the second and third portions of the duodenum respectively.

In wounds of the jejunum and ileum this progressive escape of contents has not occurred in those cases which I have seen.

Of course, if a full coil of intestine is widely opened, it stands to reason that the contents in it at the time must escape. Some undoubtedly escaped in Case IX. But where the coil is empty or the perforations small there is no escape even at the time; in any case, there is no progressive escape later, such as is seen in wounds of the duodenum or colon. The neighbouring coils and the omentum

rapidly become adherent, the injured portion becomes inflamed, immobilized, and shut off, and, although it becomes full, it is only when it is lifted from its bed at operation that the contents are discharged.

Case IV illustrates this point. The injured coil had retracted from the wound area, and was missed at the time of operation. At the autopsy, although it presented four fairly large wounds, it was so securely wrapped in omentum that no general coiling or inflammation of the peritoneum had occurred, although the coil itself was then quite full.

In Case V, where there were three wounds due to a fragment of shell, the condition was the same. In Case VI there was no escape till the coil was lifted from its bed, when the fluid contents spouted out freely.

In Case VII the injured coil was full of liquid faeces which had not escaped, and the same is true of Case VIII.

In Case IX, where the intestine was nearly divided, there was a limited escape, but no general fouling such as would be present after the rupture of an ulcer. A week later there was found a fairly widespread adhesive peritonitis with no suppuration.

So that it seems that the usual course of gunshot wounds of the small intestine, even when multiple and fairly large, is that they are sealed off at once in such a way that there is only a very small escape of contents, not enough to produce a general peritonitis or even a localized collection of faeces. All that is produced at the time is a very limited local peritonitis.

After this has occurred the intestine at and between the wounds becomes inflamed, immobilized, and paralysed; it slowly fills up and constitutes a functional block. The bowel above it becomes progressively distended, paralysed, and poisoned, just as it does in any other form of intestinal obstruction. The patient after a time develops distension and bilious vomiting, and ultimately dies.

This point is illustrated by Case VII, where the bowel was resected two and a half days after the injury, when bilious vomiting had already set in. The dilated intestine above the injury was emptied at the time of operation, and this gave temporary relief, but the symptoms recurred, and the patient died. *Post mortem* the bowel above the junction was found very much distended, that below it empty and contracted, and this although there was no physical obstruction whatever.

In Case IV, where the injured coil was not dealt with, the same condition was found at the autopsy.

The clinical course of these cases is in agreement with this view. Many of them—by no means all, but a large proportion—remain free from symptoms and apparently well for two or three days on starvation and morphine. At the end of this time, which often coincides with their transfer to a clearing hospital, they rapidly develop distension and vomiting, and ultimately die.

If these symptoms were due to an immediate escape of contents, one would expect immediate pain, rigidity, rising pulse, and vomiting. But the course they actually follow and the symptoms they present are rather those of an obstruction of gradual onset than those of a prompt general peritonitis.

The explanation of these differences in the behaviour of wounds of the duodenum and colon on the one hand, and of those of the mobile part of the small intestine on the other, is probably twofold. In the first place, the small intestine is so situated that it is very readily sealed off by omentum and adjacent coils. In the second place, it seems to be the case that the movements of the small intestine are more readily paralysed by injury and inflammation than those of either the colon or the stomach. So that although a passive dilatation occurs, there is no driving force behind it which could lead to escape of contents under pressure. If such a force were present recent adhesions would not prevent leakage, though they might limit its extent.

TREATMENT.

If the course of events is usually such as is suggested above, the bearing of it on treatment is obvious. For it is clear that simple suture of wounds of the duodenum and colon is both necessary and sufficient. If this is efficiently done the contents will be driven along their proper course; if it is not done, they will be continually forced either into the peritoneum or into the extraperitoneal tissue. In