was done by placing a small balloon on the chest and another on the abdomen, held in place by broad binders, and with each was connected a Marey's recording tambour. The tension was adjusted to give the best range of movement. The tracings thus obtained show extensive and rapid movements of the chest, but only a very slight movement, a mere tremor, of the abdomen, which subsequent investigation clearly demonstrated was propagated from the chest. As controls, tracings were taken in the same

FIG. 1.—Thoracic and abdominal respirations in Case I. Rate 100. FIG. 2.—Thoracic and abdominal respirations in Case II. Rate 88.

way from normal men. It will be observed that it is possible for a normal person to breathe at the rate of 75 a minute or more without showing any sign of irregularity of respiratory movements, or any decrease in the action of

FIG. 3.—Thoracic and abdominal respirations from a normal man taken in the same way as Figs. 1 and 2. Rate 20.

the diaphragm as indicated by the amplitude of the movements of the abdomen. The tracings taken from the two patients, then, seem to indicate a failure in the action of the diaphragm, and it is probable that the acceleration

FIG. 4.—Thoracic and abdominal respirations from a normal man breathing 60, 136, and 176 times per minute, to show the absence of any decrease in the abdominal movements.

in rate was due to this. We found no discomfort was caused by breathing at 75 a minute, but when the rate was increased to about 170 a minute it could not be maintained for any length of time. So long as it was maintained it showed no decrease in the abdominal movements.

G. Gerhardt¹ has recorded some cases diagnosed as having paralysis of the diaphragm. Of one case he said that the patient breathed shortly and insufficiently for the needs of the voice, and that he could not breathe deeply. In some of his cases improvement or cure was said to have followed faradic or galvanic treatment of the phrenic nerves. Dr. Cowan,² reporting on a case diagnosed as peripheral neuritis in a fat, flabby woman, possibly an alcoholic subject, says:

In the first week of December the diaphragm was found to be paralysed. The costal arch was unduly widened, the epigastrium fell back with each respiration, and the hernia no

longer descended into the sac; cough was feeble and imperfect, and largely replaced by choking fits. There was, however, no difficulty in breathing or cyanosis, and the respirations only numbered 26 per minute. The apex beat had swung outwards into the axilla, and the left chest was everywhere dull to percussion, and the respiratory murmur was extremely feeble, and accompanied by a few fine râles.

It would seem from this case that paralysis of the diaphragm is not necessarily accompanied by any marked increase in the rate of breathing.

Our thanks are due to Captain Torrance, R.A.M.C., for giving us permission to publish, and to Dr. A. G. Waddell for the clinical notes.

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A PNEUMOTHORAX PARADOX.

BY

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THERE is a fact very familiar to all who have experience in the treatment of phthisis by artificial pneumothorax which will probably appear very surprising to other clinicians. It would naturally be supposed that, when one lung is put quite out of action by the induction of pneumothorax, the patient would be very short of breath. Curiously enough the opposite is ordinarily the case. I was very much struck with this in a recent case of my own. The patient, a girl aged 16, had signs of extensive cavity in the left upper lobe, with abundant crepitations in the lower lobe. Some weeks after the induction of a left-sided pneumothorax she remarked to me quite spontaneously: "When I came to the hospital it was all I could do to get upstairs; now I can go up and down stairs quite easily."

The same thing is conspicuous in a patient whom I have now under treatment, by the kindness of Sir Thomas Oliver, in the Royal Infirmary, Newcastle-upon-Tyne. On admission she was able to talk but little on account of shortness of breath. Great improvement followed the induction of artificial pneumothorax on the left side. Some time after this she was allowed to go to a concert which was held at Christmas time in one of the wards. When I asked her if she had breath enough to laugh, she replied with a very pleasant exhibition of her capability in that way.

These are given merely as instances of what is an ordinary experience in successful cases. The explanation of the paradox is, no doubt, that the dyspnoea in phthisis is not mainly mechanical and due to the amount of lung tissue which is disabled by structural disease. This is the case to some extent of course. But mainly the dyspnoea is toxic. The same thing has been generally recognized by clinicians in pneumonia. A patch of consolidation in that disease may produce a degree of dyspnoea quite out of proportion to its extent. And conversely, when the crisis takes place the dyspnoea quickly abates, although physical signs may still show that a large part of the lung is not yet capable of functioning.

That an artificial pneumothorax does have the effect of reducing the output of toxins is proved by the fact that in a patient whose temperature has continued high in spite of prolonged rest in bed it may become normal within a few days of the introduction of air or gas into the pleura.

I have desired to give prominence to this effect of artificial pneumothorax in relieving shortness of breath because I apprehend that many practitioners, having patients with advanced phthisis afflicted with severe dyspnoea, are deterred from considering the induction of a pneumothorax by the fear lest the distress of the patient should be aggravated by it and the fatal termination precipitated. So far from this fear being well grounded, the truth is that provided the case is otherwise suitable for treatment by pneumothorax, that very dyspnoea, which was thought to be a contraindication, is really a condition which it is capable of relieving in a degree which cannot be expected from any other method. And it may be added that the more extensive the disease in the lung which we propose to compress with air in the pleura the less will be the strain of extra work thrown by the pneumothorax on the other lung. To immobilize

entirely a lung which is already almost incapable of function is physiologically a *vile damnum*; but from the point of view of restraining the output of toxins it may be an enormous gain.

A CASE OF ARTIFICIAL DOUBLE PNEUMOTHORAX.

BY

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IN a recent edition of a popular textbook on physiology the following sentence appears in the chapter on the mechanism of breathing: "A perforation of the chest wall would mean that the lung on that side would no longer be of use; a similar injury on the other side (double pneumothorax) would cause death." This statement, along with the impression so many, as well as myself, have always entertained, that the so-called vacuum in the parietal "space" must be retained on at least one side of the thorax, seemed at variance with the continued viability of the patient in the following case.

From the use of artificial pneumothorax in the treatment of pulmonary tuberculosis it is well known that one lung may be very greatly collapsed with little or no embarrassment to respiration. Through a fluoroscope the lung, which is under great collapse, can be seen to undergo a limited respiratory movement, and even when there is a decided positive pressure of gas this lung does not entirely cease to function. I was, however, not a little surprised to find a patient breathing quite comfortably and moving about the grounds of the institution with no embarrassment, although, in the routine treatment of a case of pulmonary tuberculosis by artificial pneumothorax, I had produced a double pneumothorax of some considerable size, through a patency existing between the right and left pleural spaces.

Mr. C. was considered a suitable patient upon whom an artificial pneumothorax should be produced in order to rest the right lung, which was extensively involved and gradually becoming worse, whilst no definite active lesions were found in the left lung.

The first four introductions of nitrogen were of small amounts, averaging about 250 c.cm. each time; and these probably went into pockets between adhesions, judging from the oscillations and readings of the water manometer. But on the fifth introduction a definite pocket of gas between the base of the lung and the diaphragm was discernible through the fluoroscope.

Pressures (Water Manometer), and Accompanying Amounts
of Gas Introduced.

Treatment.	Interval.	Gas.	Pressure at Start.	Pressure at Finish.
Initial	—	Oxygen 100 c.cm. Nitrogen 250 c.cm.	-8	+10
Second	1 day	N 300 c.cm.	-3	+10
Third	6 days	N 235 c.cm.	-20	+10
Fourth	4 "	N 260 c.cm.	-8	+20
Fifth	3 "	N 450 c.cm.	-9	-4
Sixth	4 "	N 425 c.cm.	-6	0
Seventh	8 "	N 700 c.cm.	-4	+6
Eighth	2 "	N 800 c.cm.	-4	+8
Ninth	8 "	N 1,000 c.cm.	+5	+12

After the eighth treatment the area above the diaphragm on the left side showed a slight transparency with the fluoroscope, but after the ninth treatment a very distinct pneumothorax was seen on both right and left sides, lifting the bases of both lungs away from the diaphragm. The highest part of the bases of the lungs was at the level of the fourth intercostal space in the mid-clavicular line. The patient said he was "a little short of breath," and felt that he "had gas on the stomach," but, as stated before, being an "up" patient, he continued to walk about the grounds with little discomfort.

It was obvious that the continuance of the artificial pneumothorax was not advisable; so the next day I withdrew the gas by reversing the method of its introduction.

Quantity of Gas Removed and Changes in Pressure.

Quantity of Gas Removed.	Pressure after Removal.
Initial pressure ...	... + 12
400 c.cm. ...	... + 6
600 c.cm. ...	... + 4
915 c.cm. ...	... + 1
1,175 c.cm. ...	... + 1
1,325 c.cm. ...	... - 2

As soon as a negative pressure was obtained the operation was discontinued, but, as is shown in the x-ray plate, a considerable quantity of gas still remained on the right side. The gas was withdrawn at the same site as it was introduced in previous operations—namely, the eighth intercostal space in the anterior axillary line on the right side.

A study of the two x-ray plates showing the conditions before and after the withdrawal of the gas was instructive. The patency between the right and left pleural spaces was quite well defined at the time, when viewed through the fluoroscope. The extent of the involvement of the right lung was shown by the mottling of the organ. The adhesions in both lungs were well marked, and probably accounted for the marked depression of the diaphragm on both sides. Even after withdrawal of the gas the left lung showed a triangular transparent area indicating some retention.

Besides the physiological features mentioned and the anatomical peculiarity of a patency between the pleural spaces, which, of course, may have been the result of a tuberculous focus perforating the "partition" between the spaces, there is the further lesson conveyed of the indispensability of the fluoroscope, or x rays, in conjunction with the use of artificial pneumothorax, if the dangers of compressing a healthy lung at the same time as compressing the diseased one are to be avoided.

EPIDEMIC PNEUMONIC INFLUENZA AS SEEN IN MALAYA.

BY

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PNEUMONIC INFLUENZA broke out during the month of October, 1918, amongst the Tamil labour force on a rubber estate of which I was in medical charge, and was very fatal.

Lobar pneumonia is endemic in Malaya, and causes a fairly large proportion of the deaths among all classes of the community. Thus it may easily happen that the first few cases in an epidemic of this kind are regarded merely as an unusual incidence of ordinary lobar pneumonia. In this way valuable time may be lost ere precautions against the spread of the epidemic are taken.

While the disease produces a true pneumonia, there are many symptoms which serve to distinguish it from the ordinary lobar type of that disease.

The onset is usually heralded by a rise of temperature with hacking cough and a pain, which is almost invariably located by the patient in the front of the chest, and not at one side or another as in lobar pneumonia. The onset of this pain is preceded by sore throat and a nasal discharge; the latter persists during the illness. The coughing is painful, but the cough is not of the kind usually found in ordinary pneumonia. It is deep and full, and pain excited is much more generalized. The coughing occurs in long bouts, and in the intervals there is great exhaustion with the vocal breathing or groaning present in most grave illnesses. The facies is marked by an anxious look, which persists even after delirium has set in. The complexion is a yellowish-grey. The nostrils are dilated, and the reddened mucous membrane coated with encrusted haemorrhagic discharge is a notable feature in severe cases. The sputum is rusty and of the nummular form at first, but very speedily becomes more profuse and greenish-yellow, though remaining glutinous. Profuse epistaxis is common and of unfavourable import. Haematemesis and haemoptysis have both been seen in very severe cases, always as a terminal phenomenon.

The temperature exhibits a much greater diurnal variation than in lobar pneumonia, varying between 99° and 103° F., and usually touching both these limits in