were 320,161 cases and 81,289 deaths, or 25.3 per cent. In the last three years of this period, however (these being war years as it happens), prevalence was much less and the fatality rate low—2,126 cases and 57 deaths, or 2.6 per cent.

Small-pox in Germany will be considered in the next lecture.

In Austria up till 1900 there was heavy prevalence with fatality rates of 20 to 40 per cent. From 1901 to 1914 inclusive the disease was at a low ebb, with fatality rates much below the previous level, but in 1915 and 1916 there have been nearly 50,000 cases, though the deaths are not stated.

Hungary, like Austria, had much small-pox, but there has been very regular diminution from 15,470 deaths in 1887 to 81 deaths in 1912, the last year for which figures were obtained. It is probable that the disease has had some prevalence during the war, though Budapest is the only town of which that can be said with certainty.

In Russia there is a great deal of small-pox, with apparently a very high fatality rate, though there is probably some fallacy owing to incomplete returns of cases.

The above facts as to the continent of Europe, taken from Dr. Bruce Low's report, show that while in some of the countries there has been much less small-pox in recent years, in others there is still a high prevalence, and the constant intercourse between the United Kingdom and the Continent maintains a degree of risk which prevents any sense of security against successful invasion by the disease. Also, there is always a minor risk of small-pox by cotton from America, and by paper or other textile fabrics, as exemplified in Dr. Macewen's reports on outbreaks in cotton mills in Lancashire.

But, reverting to our own country, during the last half-century there has been, with the exception of the metropolitan epidemic of 1901-2, a great diminution in the fatality and the infectivity and the prevalence of small-pox. This diminution has progressed at an increasing rate of speed. In the latter part of the half-century, and in particular since the mild epidemic of 1901-5, the country has shown unparalleled freedom from the disease, not merely in respect of fatality, but also in respect of prevalence.

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FUNCTIONAL PARALYSIS OF THE DIAPHRAGM IN TWO CASES WITH ACCELERATION OF RESPIRATION.

BY

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ATTENTION has been directed recently to the rapid, shallow type of breathing in late cases of gassing, and Haldane has associated the condition with an exaggeration of the Hering-Breuer reflex. The condition has also been observed in cases in which nervous symptoms have developed as the result of shell shock or other disturbance.

The observations made upon two cases at Oakbank Military Hospital seem to indicate that a paresis or paralysis of the diaphragm, the result of functional disturbance of the centre of the phrenic nerves, may account for the acceleration in breathing in some cases.

CASE I.

Pte. J. (nine months' service), admitted to hospital with a diagnosis of debility. He was a thin, undersized man, easily excited and nervous when spoken to. The respirations were rather rapid (28), the breath sounds somewhat feeble all over the chest, with a few moist râles at bases of lungs; he had neither cough nor sputum, the pulse rate was 80, and, beyond a localized basal bruit, there was no other abnormality of the heart. On the seventh day after admission he showed marked tremors of the hands and legs, specially severe in the right sartorius muscle. The knee-jerks were slightly exaggerated,

with occasional slight ankle clonus in the right leg. Tactile and thermal sensation and eye reflexes were normal. On the eighth day it was noticed that the respirations were becoming gradually more rapid and fluctuating very much in rate (50 to 80). Ten days later the respirations were still very variable and usually extremely rapid. The breath sounds were much more harsh than formerly, but no râles were audible. The pulse was also very variable in rate (usually about 100), and he developed a transient albuminuria, which lasted three days. Observations on the thirty-fourth day show that the legs as a whole were now extremely tremulous, and the tremor of the right sartorius was merged in the more general one. The knee-jerks were much exaggerated, but no ankle clonus could be elicited, and the plantar reflexes were sluggish. No change in the condition of the chest was observed, but the respirations were more continuously rapid and less variable in rate (80 to 90). Careful charting of the rate showed that the increased rate was only present during the day, and practically disappeared during sleep.

CHART I.—Respirations in Case I.

By the 106th day, although the patient was allowed up and could go about the ward quite freely, the high respiratory rate with a fall to normal during sleep still continued; the tremors of the hands and legs were still very marked, and the former condition of reflexes was maintained. As the patient showed no tendency to improve, he was transferred to a special hospital for neurasthenics, and unfortunately no further information has been obtained.

CASE II.

Pte. Y., admitted to hospital from France, where he had been for five weeks, gave a history of fits as an infant, intermittent otorrhoea since childhood, acute rheumatism and acute pneumonia six years ago during the same winter. He had been rejected for the army in 1915, owing, he says, to neurasthenia, but was accepted in 1917.

On admission he complained of difficulty in breathing, especially when lying down, discomfort in the cardiac region, and of occasional attacks of pain in the arms and legs. The condition was of gradual onset, and became noticeable about three weeks before admission—that is, about two weeks after going to France. He was described on that date as a very pale man of moderate physique with enlargement of the area of cardiac dullness—apex beat in fifth space in nipple line. No thrill. No bruit. Second sound slightly accentuated. Rhythm regular. Tachycardia, but not extreme—96. Respirations very laboured, numbered 50 per minute, and in consequence had great difficulty in expressing himself in speech. There was a slight prolongation of respiratory movements in a small area close to the edge of the sternum in the third right inter-space, and the sounds in this area were rather moist, but no dullness on percussion could be appreciated. There was no sputum and no pyrexia. There were no tremors. The pupils were moderately dilated. Reflexes normal. Patient was a very heavy cigarette smoker, and his smoking was drastically curtailed on his first admission to hospital. During his residence little change was noticed in his condition; the rapid breathing persisted, and was present both while awake and asleep. The tachycardia still continued, and the following chart shows graphically the state of matters:

CHART II.—Respirations in Case II. Plain line = respirations. Dotted line = pulse.

He was transferred to a special hospital for neurasthenic cases, and has since been discharged from the army on account of "functional nerve disease." The respiratory movements are still rapid, but he is able to move about slowly.

At the request of Professor Noël Paton graphic records of the chest and abdominal movements were taken. This

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