Baster, 1910, he went for a golfing holiday, forgetful of his age and of the age of his arteries, and played three rounds a day. A few mornings later he awoke with violent vertigo, retching, and vomiting. His body felt very warm. The vomit was bilious, and the direction of objects left to right. He recovered completely. His blood pressure was 150. Again, in July of the same year the same series of events led to a second attack. They were always in the morning. Yet the patient did not take warning, and late one night he fell, seized with sudden vertigo. He sweated freely and vomited (note sea-sickness). He could not be moved for three hours. When I saw the gentleman in question he was still going ahead as if he were in the prime of life. He had occasionally a severe attack. He got quite over his attacks by taking his life less strenuously, and by the addition of a little alcohol to his dietary.

2. A female, aged 62. A great sufferer from rheumatoid arthritis, and almost complete deafness in the right ear. The patient, despite her severe handicap, was a most energetic and hard-working person, busy with good works. Sept. 3rd, 1912. For about three years she had suffered with left Eustachian obstruction, gradually becoming more and more deaf. On Jan. 1st, 1912, the patient had a severe attack of vertigo in bed in the morning, with objects rotating clockwise in a vertical plane. She has had four or five severe attacks since, and many mild ones. The attacks are heralded by flushing and by a feeling of surface warmth, and at times this is accompanied by perspiration. The patient has, however, had three attacks of vertigo without these symptoms. She could hear a whisper on the left side 4 inches, and C.32 to C.4096 double vibrations. Her blood pressure was 100.

RÉSUMÉ.

Chronic progressive middle-ear deafness and arteriosclerosis are thus, according to my investigations, the most frequent causes of aural vertigo, and fortunately one finds that a large amount of benefit can be derived from the use of drugs in these cases, though by no means all of them are capable of this relief. We have also seen that operative interference is justified, and where it is used it should be uniformly successful.

There are two points in my paper which I expect will have struck the reader; one is the small reliance placed on the rotary and caloric reactions, and the other is the total omission of that so-called group in which increased labyrinthine pressure is said to exist. It is not, perhaps, quite within the scope of the paper to give at all at length my reasons for not employing the tests, nor for placing more reliance upon them. I am convinced in my own mind that one is able to make one's diagnoses and one's prognoses in the class of case under consideration equally well without their employment. I must, however, admit that, especially in cases of low arterial tension, the results were most extraordinarily interesting. With regard to increased intralabyrinthine pressure, although I have operated on at least 20 cases of labyrinthine vertigo where there had been no perforation into the labyrinth—14 of them where there had been no previous suppuration, and the rest where there had—I have only twice seen any fluid on opening the labyrinth. In these two cases there was a considerable amount of fluid; one of them also had an extremely large external semicircular canal, apparently four or five times greater than normal. But even then I am not prepared to say that these cases contained fluid under pressure, and I have consequently thought it better, as the matter is treated entirely clinically, to omit this possible factor. But it is rather difficult, indeed, to understand, from a purely mechanical point of view, how fluid can be retained under pressure in such a non-vascular bony cavity as the labyrinth.

The use of ernutine was suggested by my senior clinical assistant, Mr. A. F. Penny, of London. I found it impossible, even in so lengthy an analysis, to contrast and apply Dr. Kerrison's classification to that I adopted, so preferred to leave it, hoping at some future time to give it the place it deserves and derive from it the benefits it possesses.

Harley-street, W.

MANCHESTER UNIVERSITY.—Dr. T. Wingate Todd, lecturer in anatomy in this University, has been appointed Henry Wilson Payne Professor of Anatomy in the Medical Department of the Western Reserve University, Cleveland, Ohio.

TUBERCULOSIS DISPENSARY FOR BIRKENHEAD.—Dr. J. R. Hutchinson, Local Government Board inspector, held an inquiry recently at the Birkenhead town hall into an application by the town council for powers to borrow £620 for a tuberculosis dispensary under the Insurance Act. It is proposed to purchase premises in Duncan-street, and convert them into a dispensary. At the conclusion of the inquiry the inspector visited the premises.

FURTHER OBSERVATIONS ON THE PNEUMOTHORAX TREATMENT OF PHTHISIS.

BY CLAUDE LILLINGSTON, M.B., B.C. CANTAB.

SINCE the publication of three papers on the subject in THE LANCET of July 15th, 1911, Forlanini's treatment has steadily gained favour in England, where its popularity suffered from a negative phase long after its establishment abroad. Now it is suffering here from a positive phase which is equally unfortunate, as a careful selection of cases is not always made, and the operation is not always performed with that detailed knowledge and observance of the technique which are imperatively necessary. The induction of an artificial pneumothorax by one whose knowledge of the technique is culled from the brief perusal of a few treatises on the subject shows deplorably vicarious courage. It may, therefore, be advisable at this stage to take stock of this method, to deal with the broad outlines of the rationale, indications for and against, and complications, and to describe in detail some of the steps in the technique which, if misunderstood, may cause sudden death.

Rationale.

Is phthisis benefited by an artificial pneumothorax, and if so, why? All who have observed a certain number of patients thus treated give an affirmative answer to the first question. But it is more difficult to explain why the results are beneficial. There are many more or less plausible explanations forthcoming.

Collapse and compression of the diseased lung must rest it as effectively as a well-applied splint rests a tuberculous knee; in either case auto-inoculation is reduced to a minimum. Another beneficial factor is the expression and drainage of fluid from the lung which, when completely collapsed, scarcely exceeds a normal kidney in size. Even when adhesions prevent complete collapse, a partial pneumothorax may greatly aid the drainage of cavities and the expression of fluid from the whole lung. I have seen several patients the apices of whose lungs were firmly adherent to the chest wall, and whose disease was most evident in the upper half of the lung. Yet compression of the lower half was strikingly beneficial, the daily expectoration diminishing to about one-tenth of the former amount in a few weeks. It is therefore probable that, even if a cavity in the apex is not compressed, the compression of the neighbouring lung aids the drainage of pus which, before compression, flowed over from the cavity into the more dependent portions of the lung. That fluid is squeezed out of the lung is shown by the increase of the expectoration which occurs during the first few days of the treatment.

Another favourable factor is the extensive formation of fibrous tissue around the diseased areas, which thus become securely cut off from healthy tissues. It is important to note that healthy pulmonary tissue does not necessarily become disorganised or permeated by scar tissue even after several years of consistent compression; and when a lung containing both healthy and tuberculous tissue is allowed to re-expand, the expansion is practically limited to the tissues which were originally healthy or but slightly cedematous. This has been repeatedly demonstrated post mortem. It has been recently urged against this treatment that healthy portions of the lung are wasted by compression which fails to act on diseased portions because pleural adhesions prevent their collapse. But this speculative objection is negated by the post mortem observations already referred to. The assumption lately advanced by a consultant in London that tuberculous foci in the lung are invariably accompanied by adjacent pleural adhesions is indeed extraordinary, for the induction of an artificial pneumothorax has frequently shown that comparatively healthy lung is adherent to the chest wall, while another portion of the lung, occupied by cavities and pneumonic infiltration, is covered by free pleura.

Two other explanations of the beneficial action of an artificial pneumothorax may be briefly mentioned. Carl Goebel¹ has pointed out that as an aerobic organism, the tubercle bacillus grows best in a free supply of oxygen, in

¹ Zeitschrift für Tuberculose, 1910, vol. xvi.

the absence of which its virulence is reduced. This accounts for the preference shown by the tubercle bacillus for organs, largely composed of fibrous tissue, in which the process of oxidation is low, and in which a considerable quantity of oxygen is therefore available for the tubercle bacillus. Hence, also, the rarity of phthisis in cases of mitral stenosis in which the lungs are congested with venous blood. The nitrogen, therefore, which is injected into the pleural cavity under a positive pressure may act beneficially through its gradual absorption by the diseased lung, to which the supply of arterial blood is largely reduced by the partial compression of the pulmonary arteries. Other observations which support this view are the benefit to tuberculous peritonitis derived from exploratory laparotomy, which favours venous stasis and absorption of atmospheric nitrogen occur; and the benefit obtained by Kuhn's suction mask, and by Bier's treatment by passive congestion. Several other arguments are advanced by Goebel in support of his view.

The other explanation, for an account of which I am indebted to Dr. Ernest P. A. Mariette, is advanced by Molle,² who attributes the success of treatment by an artificial pneumothorax to a trophic excitation of the pneumogastric nerves. He holds that there is a danger of "transferring" the disease to the opposite side in patients whose reflex irritability is abnormally great. He has observed in certain phthisical patients a neuromuscular hemihyperæsthesia at a spot where other physical signs of disease shortly appeared, and which was symmetrically opposite to the original lesion on the other side. He also points out that counter-irritation by the cautery, blisters, or tincture of iodine may cause migration of nervous complications from one side of the body to the other. Patients who react thus to counter-irritants are not, in his opinion, suitable for an artificial pneumothorax, the stimulus of which is likely to "transfer" the disease to the uncompressed lung. Patients, on the other hand, whose reflex irritability is found by experiments with counter-irritants to be low, are suitable for the pneumothorax treatment, provided the disease is more or less unilateral. But, he adds, counter-irritation alone will prove as effective as a pneumothorax in this class of patient. He concludes that the new surgical methods of treating phthisis, and especially an artificial pneumothorax, constitute a special form of counter-irritation whose effects are akin, for good or evil, to those of ordinary counter-irritation.

This theory is ingenious, almost fantastic. But Molle's arguments are too numerous and carefully weighed to be straightway dismissed. I have seen two patients whose disease was practically unilateral, and on whom several attempts to induce an artificial pneumothorax failed on account of pleural adhesions. The apparent failure of the operation greatly depressed the patients, who thought their last chance of recovery was lost. But both improved rapidly, and one is now walking ten miles a day two years after the attempt to induce an artificial pneumothorax. Both these patients were desperately ill.

Though not prepared to dismiss the two latter explanations as mere speculative conjectures, I hold that immobilisation and drainage of the diseased lung are the chief causes of arrest of the disease. The objection has been raised that if collapse and rest are beneficial to the diseased lung, the extra work thrown on the other lung must injure it. But, as Saugman has pointed out, the two lungs are simultaneously treated by different methods. Under ordinary sanatorium treatment patients with active disease are kept strictly at rest, while patients with slight and afebrile disease are exercised. Similarly, with the pneumothorax treatment, the severely diseased lung is given absolute rest, while the slightly diseased lung is exercised.

Indications for and against the Treatment.

The present attitude of many general practitioners and consultants towards the treatment is one of blank ignorance or of horror and contempt. To them an artificial pneumothorax is identical with a spontaneous, accidental pneumothorax with its concomitant distress and risks of sepsis. Yet the former can be induced so gradually that the patient is put to little discomfort, dyspnoea, or pain; and, if necessary, the gas can be withdrawn from the pleural cavity, and *status quo ante* restored in a few

minutes. Patients are frequently either warned against the treatment, or they are recommended it when moribund, and when the disease is extensive in both lungs. To induce a pneumothorax when moist sounds are audible everywhere over both lungs is to hasten the patient's death and to bring the treatment to disrepute. Yet half the patients in whom I have been requested to induce an artificial pneumothorax belong to this category.

The condition most suitable for the treatment is extensive disease of one lung, little or no disease of the other. But there are also many other suitable conditions. For instance, the disease may be limited to the apices of both lungs, but compression of one lung becomes an urgent necessity on account of recurrent and severe hæmoptysis. Again, one lung may be practically healthy, and the disease in the other may extend only from the apex to the third rib in front, and to the spine of the scapula behind; yet if this condition has persisted for over a year in spite of sanatorium and tuberculin treatment, and the patient is incapacitated, an artificial pneumothorax may be the best treatment. Sometimes half of each lung may be involved, and the patient may be going downhill in spite of ordinary treatment. The experienced physician who has watched such a case for several months can often give a shrewd estimate of the activity of the disease in each lung, and if he finds it practically stationary in one, the compression of the other may give strikingly good results. Dr. Jane Walker has kindly shown me a patient who developed a spontaneous pneumothorax which she is maintaining by injections of nitrogen. The patient is rapidly improving, although more than half his uncompressed lung is affected; here, therefore, the disease is evidently stationary.

Often only after a pneumothorax has been induced is it possible to estimate the degree of activity of the disease in the healthier lung. Whenever the disease in this lung is slight but active, the pneumothorax on the other side must be gradually induced, so as not to throw a sudden excess of work on the uncompressed lung. It should also be remembered that slight movements by a patient breathing with one lung only are equivalent to violent exercise by the possessor of two healthy lungs. It is, however, extraordinary how much exercise can be taken by patients accustomed to harbouring a pneumothorax, and I have sprinted to catch a train when I boasted a large pneumothorax. But till the healthier lung has proved its capacity to do the work of two, it must be watched with the vigilance of a cat at a mouse-hole, and absolute rest must be prescribed as soon as danger signals, as a rise of temperature or other physical signs, appear. If the healthier of the two lungs shows recent signs of disease, such as a pleuritic rub combined with fever, the induction of a pneumothorax on the other side should be deferred till this condition has subsided, even if the healthier lung appears in other respects to be normal.

It is the exception for the pleural surfaces to be adherent throughout, even in old-standing cases complicated by repeated attacks of pleurisy; and I have only in 15 per cent. of my cases failed to induce a pneumothorax on account of adhesions. But it is often difficult to find a point where the pleural surfaces are free, and from which the intrapleural pressure may be raised till soft and recent adhesions elsewhere give way. When once a small pocket of nitrogen has been formed between the pleural surfaces, it is often astonishing how, with frequent small injections of nitrogen, this pocket may be enlarged till almost all the adhesions have given way.

So far from being a contraindication, tuberculous laryngitis is, *ceteris paribus*, an indication for the treatment which favours its arrest by reducing cough and expectoration and by improving the general condition. Dr. S. V. Pearson's success at Mundesley Sanatorium with a case of severe laryngeal disease combined with almost unilateral pulmonary tuberculosis has been most striking with this treatment. It is unlikely that the life of a patient suffering from intestinal tuberculosis and persistent diarrhoea can be appreciably prolonged, even if the pulmonary disease be arrested by a pneumothorax. It is deplorable how many patients ultimately die from tuberculosis of the intestine, kidneys, meninges, and other organs whose pulmonary disease was found at the necropsy to be completely arrested by an artificial pneumothorax. In most of these cases the disease had lasted for several years before the pneumothorax was induced, and it is certain that when this

treatment is adopted earlier in the disease, the tale of fatalities due to tuberculosis in organs other than the lung will be appreciably diminished.

Complications and their Avoidance.

Much has been written about pleural reflex and gaseous embolism, the two causes of sudden death during the operation. With care, both risks are negligible. The risk of pleural reflex is reduced by injecting eucaine or stovaine into the pleura. Two points in this connexion are important. The solution of anæsthetic must be concentrated so that only a little fluid escapes into the tissues; otherwise fluid in the track of the pneumothorax needle will fill its lumen, and thus prevent the registration by the manometer of changes in the intrathoracic pressure. If the operator holds the hypodermic needle lightly he can also probe with it to ascertain the pleura's exact depth from the surface and its state, whether it be thickened or not. I usually inject eucaine at three or four different points in the chest, and select for my first puncture with the pneumothorax needle that point at which the hypodermic needle has encountered least resistance. Unfortunately, however, firm pleural adhesions sometimes exist even where the exploring hypodermic needle has encountered but slight resistance.

Sudden death from gaseous embolism continues to occur, as recent publications show. But, as indicated by the following case recently published by Balvay and Arcelin,³ these deaths may be the operator's—and not the operation's—fault. The patient had acquired a fairly large artificial pneumothorax, but her lung was still adherent to the chest wall in places. On introducing the pneumothorax needle at a different point from that at which the first injections had been given, the operator failed to obtain characteristic manometric oscillations. In spite of this ominous warning the gas was introduced under considerable pressure. The patient immediately became unconscious. She coughed up blood, and in 48 hours she was dead. The needle had been introduced over the site of an adhesion which held a portion of the lung to the chest wall, and thus gas was forced into a pulmonary vein.

A similar accident may occur even if the pneumothorax is complete and no pleural adhesions exist, if the operator is careless enough to ignore a motionless manometer. For the needle may catch in a blood-vessel superficial to the parietal pleura, and with this vessel impaled on its point, it may invaginate the parietal pleura without puncturing it, and may reach to the depth at which the operator on previous occasions has penetrated to the pleural cavity, and has obtained characteristic oscillations. He may now argue that he is working at the right depth, and that no manometric oscillations appear because the needle has become obstructed by a small particle of tissue in its transit through the chest wall. He therefore proceeds to blow out this hypothetical particle by forcing gas through the needle. The result is more instructive to himself than advantageous to the patient.

Gaseous embolism may happen in yet another way. During a first injection the operator thrusts the needle through the chest wall till characteristic manometric oscillations show that its point is in the pleural cavity. He now holds the needle securely at this depth lest it should pass deeper and penetrate the visceral pleura. But though neither the patient nor the needle has apparently been moved, the oscillations may suddenly cease, owing, as a rule, to the parietal pleura having slipped over the point of the needle, which consequently is again superficial to both layers of the pleura. The operator who, because he has once obtained typical oscillations, and because he thinks the needle has not moved since, injects nitrogen in the face of a motionless manometer, will sooner or later have a sudden death to explain.

It follows that however secure the operator may feel, his only safe course is to watch every movement of the manometer, and to cut off the gas as soon as satisfactory oscillations cease. When, hour after hour, he has tried in vain to obtain satisfactory oscillations by puncturing in several places, the temptation to "shove in gas and see what happens" is great. Cases have, in fact, been recorded in which no oscillations were obtained till a little gas had been forced into the pleural cavity, after which

typical oscillations appeared. By this procedure hopelessly ill patients have regained their health; but the risk entailed is considerable, even when the needle is aspirated to ascertain the presence of blood in its lumen before any gas is injected. No novice should attempt this method.

Results.

In such a chronic and capricious disease as phthisis an estimation of the value of any form of treatment is worthless unless the number of patients treated is considerable, and the improvement effected has stood the test of time. My experience, therefore, with 18 patients treated for periods ranging from 6 months to 3½ years, is insufficient *per se* to build pretentious claims on, and I will but briefly touch on the following points. The disease was in the third stage and active, and the prognosis was very bad in every case. Yet the disease is either arrested or is undergoing arrest in 13. Five patients are either dead or dying; but I sincerely believe that the treatment has prolonged their lives. I have seen no fatal accident from the treatment.

More valuable are Dr. L. Spengler's results, for they have stood the test of a longer probation period.⁴ He has excluded all recent cases, confining himself to those in which a pneumothorax had been induced nine months to four years earlier. As evidence of arrest of disease he adopted the following conditions: absence of pyrexia, cough, and expectoration; if the latter be present, it must not contain tubercle bacilli. Fifteen patients fulfilled these conditions, and were also capable of a full day's work. In 12 of these the prognosis before the operation was almost hopeless, and in the remaining 3 it was very bad. Dr. Spengler considers that these results are a satisfactory answer to the objection that though a pneumothorax may cause temporary improvement, it does not often enough produce permanent results to warrant the risk of operation. A useful review of recent literature on the treatment is given by Dr. W. T. Ritchie,⁵ who has collected the results of several workers in this field. Interesting original articles on this subject have also lately been published by Dr. S. Vere Pearson⁶ and Mr. Hubert Chitty.⁷

Gorleston-on-Sea.

A CASE OF COMPLETE ERADICATION OF EXTENSIVE RECURRENT CANCER AFFECTING BOTH BREASTS AND AXILLÆ;

DEATH 15 YEARS LATER FROM HEART DISEASE AND DROPSY
AT THE AGE OF 82 YEARS.

BY R. CLEMENT LUCAS, B.S. LOND., F.R.C.S. ENG.,
CONSULTING SURGEON TO GUY'S HOSPITAL AND TO THE EVELINA HOS-
PITAL FOR CHILDREN; MEMBER OF THE COUNCIL OF THE ROYAL
COLLEGE OF SURGEONS AND OF THE EXECUTIVE COMMITTEE
OF THE IMPERIAL CANCER RESEARCH FUND.

It is not only by the consent, but also by the desire, of the relatives of this patient that I publish the details of her case, and its publication may perhaps assist other surgeons to persuade their patients to avail themselves of the only known means of cure for cancer, rather than gamble away their only available chance of safety by resorting to useless methods of treatment so temptingly put forward by designing persons, who play upon the fears and encourage the natural dread that everyone feels at the idea of having an operation performed.

E. J. was a patient of Mr. J. A. Hosker, of Boscombe, Bournemouth, and she had been operated upon some years before I saw her by a Bournemouth surgeon, who had amputated her left breast for cancer, but had not carried his incision into the axilla to clear away the lymphatics in that situation. In the interval between the operation and the recurrence of the disease the operating surgeon had died, and this fact may have made the patient hesitate some time before consulting her own medical man as to her tumours when recurrence took place. She, however, had a nephew in our profession, Mr. W. Watkin Leigh, of Treharris, who at one time had been my dresser at Guy's Hospital, and

⁴ Münchener Medizinische Wochenschrift, Feb. 28th, 1911.

⁵ Edinburgh Medical Journal, July, 1912.

⁶ Brit. Med. Jour., Oct. 12th, 1912.

⁷ Medical Press and Circular, Nov. 13th, 1912.

³ Archives Générales de Médecine de Lyon, May 29th, 1911.