A pair of larger rabbits was infected by the subcutaneous instead of by the parenteral route. Both were decidedly out of sorts in seventeen days. Treatment was commenced on the twenty-sixth day. The first had already begun to decline seriously eight days before, owing to the infection having been intentionally accelerated by massage of the infected glands. Tuberculin of human origin was administered to both animals, but the first died thirty-five days after infection, without having made the slightest sign of rallying.

The second rabbit of this pair was seriously ill within sixteen days of infection, and treatment was commenced ten days later, and continued to the fifty-sixth day after the original infection. Only seven doses of tuberculin were given altogether. This animal increased in weight long after treatment was stopped, for nearly five and a half months. Eventually it was seized with nasal catarrh and signs of pneumonia. Nasal catarrh had never been observed in any of the other animals; it was probably a superadded infection by one of the pasteurilla group of organisms, and on this supposition the animal was promptly killed. *Post mortem* large healing tubercles were found in both lungs as well as fresh haemorrhagic patches. The site of infection was completely healed, but there was a large subscapular tuberculous gland, circumscribed by healthy tissue, though otherwise certainly capable of rekindling the infection.

This case and the last one of the other series were both decidedly encouraging, because rabbits at the best are difficult to manage under experimental conditions. Many die from accidental causes, and it says much for the care and skill of my assistants that the death rate of both rabbits and guinea-pigs is the lowest that I ever heard of. All these animals were very severely infected before treatment commenced, and they were all treated with very excessive doses of tuberculin. Not one reacted fatally to this excessive dosage.

Man has a much greater natural resistance to tuberculosis than the guinea-pig or rabbit, and his treatment is accordingly more satisfactory. Still, I have little fear about the results of my next animal experiments, if I think it necessary to resume them. As soon as it was thoroughly ascertained that the use of this tuberculin was perfectly safe for animals, I took advantage of the courtesy of Mr. Gemmill, and later of Mr. Naughton Dunn, who enabled me to treat cases of lupus vulgaris and of surgical tuberculosis. These cases responded very favourably.

Lupus erythematosus has responded to treatment even more rapidly than lupus vulgaris. But I consider an apparent cure not to be beyond danger of relapse, and would advise further treatment for eighteen months. I am indebted to Mr. Yates and Mr. Heath for cases of this disease.

I am indebted to Dr. Taylor of Syston, Leicester, for the first reports on the treatment of pulmonary tuberculosis with this tuberculin. I deliberately avoided treating this disease until I had studied the effect of treating the other forms of tuberculosis. Encouraged by Dr. Taylor's reports on advanced chronic cases, I undertook the treatment of a very advanced case of phthisis. I am indebted to Dr. Castellain for an introduction to this case, and to Dr. Garbutt for permission to attend and give the necessary treatment.

There were physical signs in both lungs and large cavities in the upper lobe of the right lung. The prospects were as hopeless as could be desired for the trial of a new method of treatment, and in that opinion we all concurred. The patient was aged 23, and was herself a good example of the "spes phthisica." She was quite sure she was recovering. She had three doses of the bovine tuberculin, followed by a course of autogenous tuberculin, beginning with a dose of a millionth of a gram. All anorexia rapidly ceased, the appetite became excellent, and sleep was restored; sweats ceased, sputum was reduced from about two pints a day to about a quarter of a pint. The pulse became regular and much slower (144 at first, afterwards 88), respiration was calm, tubercle bacilli were comparatively scanty in the sputum, and the physical signs greatly improved. The temperature became more equable, but was slower in coming under control than any other symptom. Flesh was put on quite obviously.

Later the patient had a complete pneumothorax of the right side. The temperature rose to nearly 103°, but it afterwards returned to normal, and she made some progress. Nineteen days after the occurrence of the accident the lung apparently collapsed again suddenly and death quickly ensued. She had been examined regularly all the time, and the lung was found to be expanding.

The principle I have enunciated is probably applicable to the treatment of leprosy and certain other diseases. The nastin treatment of leprosy by Professor Deycke of Constantinople is akin to it, but nastin is very toxic.

I am indebted to the Director-General of Medical Services, Ministry of Pensions, for permission to publish this paper.

PLEURAL REFLEX ASSOCIATED WITH FRACTURE OF THE TWELFTH RIB.

BY

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THE following case seems worthy of record as a striking and unusual complication of fractured rib.

An unmarried woman, aged 41, fell from a stool on the night of July 15th, 1924, striking her back against a chest of drawers. There was an abrasion of the skin extending vertically upwards from a point below the level of the last rib about four inches from the right border of the spinal column. The twelfth rib was fractured about midway between its junction with the vertebra and the abrasion.

Strapping was applied, and the patient remained in bed, experiencing, for the first few days, considerable discomfort and pain even on slight movement. Four days after the accident she had an attack of hiccup lasting for some hours; both before and after this she had an occasional single hiccup. Progress was otherwise uneventful, and on July 28th the patient got out of bed for the first time. The next morning, when out of bed, she stooped for something and was suddenly conscious of a feeling of faintness, breathlessness, and palpitation, accompanied by severe pain in the upper part of the sternum. She felt very ill and thought that she was going to die. When seen an hour later she was pale, anxious, and dyspnoeic. The pulse (136 a minute) was feeble, and small, but regular. The heart was not enlarged. Harsh, rapid, and shallow breath sounds were heard all over the lungs. There were no areas of diminished resonance and no adventitious sounds. The temperature was 98° F. Adrenaline and cardiac stimulants were administered and the breathing improved; the general condition, however, remained serious during the day. In the evening the pulse rate was still 136, and the temperature had risen to 100.8° F. There were no physical signs in the chest and no pain at the site of fracture.

After this improvement was steady. The pain in the upper part of the sternum was continuous until the third day; it was not increased by breathing or movement. The temperature fell gradually, and was normal on July 31st (fourth day). On August 2nd the pulse rate was 72, the respiration rate 20, and the general condition good. The patient was allowed to get up. From about August 1st onwards, when all acute symptoms had subsided, involuntary sighs occurred from time to time, seeming to the patient to arise from the mid-sternal region. They continued two or three times a day for about a week, ceasing gradually. There was no further interruption to convalescence.

It seems probable that the sudden movement of stooping irritated the nerves of the pleura, either by tearing an adhesion or by friction, and so gave rise to a pleural reflex of the vasomotor type.

A consideration of the anatomy of the part shows that the pleura and diaphragm are in close proximity to the twelfth rib at the site of fracture, the external arcuate ligament of the diaphragm being attached to the tip and lower margin of the twelfth rib and to the transverse process of the first lumbar vertebra. The lung is above, reaching only to the eleventh rib, while the pleura extends to below the twelfth. In the normal subject the kidney would be liable to injury through the pleura and diaphragm, but in this patient it was movable and well below the level of the rib.

Pleural reflex or pleuro-pulmonary reflex has usually been recorded in connexion with operative thoracotomy. Stivelman¹ concluded that in over 75 per cent. of cases there was injury to the visceral pleura and underlying lung tissue. Capps,² however, has shown experimentally that the reflex can be occasioned by irritation of the pleura alone, the healthy pleura being much more tolerant than inflamed pleura. It is probable that, in the case reported, the pleura had been injured. The patient experienced more pain and discomfort than usually attend simple fractured rib, while the hiccup during the first week points to involvement of the diaphragm and therefore of the diaphragmatic pleura. The inflammatory reaction may well have persisted till the thirteenth day, when the cardio-respiratory symptoms occurred.

The sighing which arose during convalescence calls for some comment. Physiologists explain sighing as a deep inspiration following, and compensating for, a temporary cessation of breathing.³ Various causes which depress the respiratory centre may give rise to sighing—for example, cerebral anaemia and the excessive use of tobacco. It also occurs when the mind is deeply engaged, so that people unconsciously sigh at their work.

In the case under review a cause for suppression of respiration might be found in the injured pleura and

diaphragm; or, since the patient was up and about, the sighing may simply have resulted from cerebral anaemia connected with the recent vasomotor disturbance.

REFERENCES.

¹ Stivelman: *Amer. Journ. Med. Sci.*, June, 1923, 876. ² Capps: *Oxford Medicine*, 1920, vol. ii, 163. ³ Hutchison: *Applied Physiology*, Arnold, 1908, p. 221.

ARSENICAL POISONING TREATED BY SODIUM THIOSULPHATE.

BY

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The following case is of importance as a sequel to the communication from Dr. H. C. Semon in the *BRITISH MEDICAL JOURNAL* of April 12th, 1924.

A woman, aged 30, became infected with syphilis towards the end of her fourth pregnancy. She had been treated by intravenous injections for ten months, and had then been advised to take six weeks' rest from treatment. Three weeks after the last injection, and five weeks before she came under our care, she became jaundiced, and noticed loss of power in the arms and general weakness; she passed dark urine and pale stools. For seven days prior to admission she had vomiting, anorexia, constipation, insomnia, and failing vision.

On admission there was deep generalized jaundice, the mouth was dry and dirty, and answers to questions were correct but monosyllabic and very slow. There were no physical signs of neuritis, but the hands could not be lifted, and the vision was bare perception of light. The liver dullness was diminished; the urine contained albumin and bile.

While awaiting a supply of chemically pure dehydrated sodium thiosulphate we gave the patient 30 grains of the stock salt in

solution by the mouth every four hours, and continued this method of administration throughout the first week. There were no resultant toxic symptoms (colic or diarrhoea), but there was no arrest of the arsenical symptoms. By the third day the patient was apparently moribund; she vomited frequently and in excess of intake; she was restless, apathetic, and doubly incontinent; the bowels acted only after enemata.

At this stage the first intravenous injection was given, and for ten days the patient had 0.45 gram daily, followed by seven similar doses on alternate days, making a total of 7.65 grams spread over a period of twenty-six days. In addition, during the first week she took about 80 grams by the mouth, but probably much of this was lost by vomiting. From the first intravenous injection the downward trend was arrested. After five doses the mental condition was perfectly clear, muscular power of the arms was good for all movements, and the patient could read small print. She was still jaundiced, incontinent, and vomiting. There was still bile in the urine, and leucine was seen in the deposit. At the end of the third week she was convalescent and able to be up for half the day, eating well, and not vomiting; the jaundice was almost gone, and the urine normal.

The levulose tolerance was tested and seemed to show a definite derangement of liver function during the height of the illness, with rapid recovery under treatment. The deterioration in her general condition during the first two days clearly demonstrated the progressive nature of the disease and emphasized the remarkable improvement following intravenous administration of the specific remedy.

Levulose Tolerance.	April 25th.	May 13th.
Fasting blood sugar	0.131	0.100
Half an hour after levulose	0.137	0.086
One hour after levulose	0.218	0.103
One and a half hours after levulose	0.206	0.106
Two hours after levulose	0.206	0.100

We should like to express our thanks to Dr. Thackray, Medical Superintendent, for permission to publish this case.

Memoranda:

MEDICAL, SURGICAL, OBSTETRICAL.

MALIGNANT CERVICAL ADENITIS SIMULATING ANEURYSM.

THE case here recorded, though disappointing from a diagnostic point of view, is interesting as illustrating how very closely cervical adenitis may simulate aneurysm. An exploring needle would have settled the diagnosis, but as nothing was to be gained by the procedure it was not carried out.

A woman, aged 79, was admitted to the Tooting Bec Hospital in August, 1923. She showed evidence of specific disease, and her history confirmed this. On October 14th, 1924, she suddenly developed a tumour about the size of a hen's egg above the right clavicle. As far as can be ascertained this took only about a week to become apparent. The tumour was slightly irregular in shape and was distinctly expansile. Two little flags, one on either side of it, separated about a quarter of an inch with each beat of the heart. The heart rate was 66 and the blood pressure 130. There was no appreciable difference in the two radial pulses, but the right radial pulse gave a tracing exactly similar to that of a subclavian aneurysm portrayed in Hutchison and Rainy's *Clinical Methods*. There was a distinct systolic bruit over the aneurysm and over the aortic area. This is interesting as an illustration of a contribution I sent to the *JOURNAL* some time ago entitled "An aortic murmur."

A month later the tumour was noted as much larger—the size of a small orange—and the radial pulse as being much altered. The patient was treated with potassium iodide, being rapidly advanced to 40 grains every four hours, and showing no unpleasant symptoms from the drug. The size of the tumour in a week's time was distinctly less and there was a constricting band at the back of the mass. The following week the mass increased in size and the potassium iodide was discontinued. The blood pressure was 135, and the radial pulse tracings were similar, with no morbid characteristics. All this time there was little or no complaint of pain, and the patient died on December 5th from a low form of terminal pneumonia.

At the necropsy a large mass of cancerous glands was found completely encircling the subclavian and carotid arteries with secondary deposits in the mediastinal glands and deep in the liver.

The diagnosis made was a carotid aneurysm involving the subclavian and possibly innominate arteries, and several persons of wide experience saw the patient and found no

fault with the conclusion arrived at. When the change in size was brought about by administration of potassium iodide it was thought that that clinched the diagnosis. The systolic bruits were, of course, caused by compression of the artery, and not by the constriction at the entrance or exit of an aneurysmal sac.

I am indebted to Dr. E. H. Beresford for permission to publish the case.

Tooting, S.W.

THOMAS LINDSAY, M.D.,
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IMMUNITY, SPECIFICITY, AND NON-SPECIFIC FACTORS.

IN the issue of December 13th, 1924 (p. 1098), Dr. R. A. O'Brien says: "It may be that the administration of *coli* vaccine does materially influence the course of typhoid fever and of arthritis, but we feel that we must have more evidence before accepting the claim." In view of this the following case of colitis and arthritis cut short by an attack of typhoid fever, and the recovery from the typhoid fever, is of interest.

A girl, aged 18, suffered since the age of 13 from attacks of subacute arthritic rheumatism. She had suffered for some years before this from five or six loose motions a day. An examination of the stools showed enormous number of *B. coli*. The arthritis was most probably caused by the colitis. At 18 years of age she contracted typhoid fever. The fever was complicated by severe haemorrhage on the twenty-third day of the disease. Vidal's test was positive. The temperature fell to normal on the forty-second day of the illness, and health and strength gradually returned. The arthritis and stiffness of joints disappeared and have not returned, though it is now more than three years since recovery from the typhoid fever. The motions are now one or two a day.

One case proves nothing, as it may be entirely coincidence, though even coincidence has a cause; but the recovery from the typhoid fever and the cutting short of arthritis and of colitis appear to me to be sufficiently interesting to record, especially to those who successfully treat typhoid fever by *coli* vaccine, or those who successfully treat arthritis by intravenous injections of *B. coli* or typhoid bacilli.

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