Although it is true that opium and arsenic, e.g., are poisons when given in overdoses, still we do not, merely on that account, taboo them as valuable remedial agents. Similarly, because alcohol is a poison to the amoeba when it bathes in saturated solutions of it *ad libitum*, or to the dipsomaniac when he "sups" it in bucketsful, it does not necessarily follow that, when given medicinally and in properly measured and limited medicinal doses, it will kill or even injure white blood corpuscles or beneficent phagocytes standing sentinel against the inroads of the tubercle bacillus. On the contrary, there is perhaps sufficient evidence to say that by inhibiting injurious intestinal fermentative changes, which probably favour auto-toxæmia, alcohol when thus administered may be a valuable aid in defending the system against infection. The difficulty for the amoeba or for the white blood corpuscle is to know what is the proper medicinal dose. For when even a scientific "Band of Hope" lecturer seems to imply, if we may judge from his answer to an anxious inquirer at the close of a lecture delivered in Liverpool, that a tablespoonful of whisky can be taken with impunity after every meal, there may be some excuse after all for the dipsomaniac amoeba—for the white corpuscle which does not know where to stop. When attending the post-graduate course at Edinburgh in September, 1891, I found that Dr. (now Sir) R. W. Philip, in the clinique at the Victoria Dispensary for Tuberculosis, advised alcohol to be given in cases with fever temperatures, where it was not contraindicated by renal or cerebral complications.

As to tuberculin, I did not use any before 1901. Ten years previously I had asked Professor A. Fraenkel, when I was studying in his post-graduate course at the Moabit Hospital in Berlin, to show me cases of phthisis treated by tuberculin. He told me that he had none to show me, because he was not using tuberculin, having been disappointed, a few months previously, at the time of the first rush for tuberculin treatment. At Edinburgh, also, in the same year (1891), no tuberculin was being used by Dr. Philip. Subsequently, when the initial difficulties with Koch's tuberculin had been overcome, when the product had been standardised, and the safe initial doses for both old and new tuberculin found, I commenced employing the remedy in my practice here, at first in one or two cases of surgical tuberculosis—cervical glands, &c.—and afterwards also in phthisis pulmonalis. The tuberculin used was in all cases Koch's new tuberculin, T.R. The initial dose was a small one (1/100000 mgm. or 1/80000 mgm.) in the majority of cases. This was followed three or four days later with from 1/80000 to 1/60000 mgm. according to the amount of the previous dose, and provided all general reaction had ceased if there had been any. Three or four days later the dose was increased by another 1/20000 mgm. and so on, the temperature being invariably taken on the same evening as well as in the afternoon and evening of the two following days, and if there was the slightest rise above the normal the dose was not increased on the fourth day. Subsequently the temperatures were taken every two or three hours.

The intralaryngeal injections of antiseptics were given up after a year or two, because the results obtained with this method of treatment did not appear to be as favourable as those following the guaiacol treatment.

After August, 1912, the method of tuberculin treatment above described has been almost entirely replaced by the German intensive treatment of doubling the dose every four days whenever the temperature observations, made every two hours, indicated such a course of procedure—i.e., when no rise of temperature had followed the previous dose—and ultimately going on to the larger doses of 1, 2, 4, &c. mgm. up to half a cubic centimetre of T.R. The results have been better than by the old method of treatment.

Southport.

UNIVERSITY OF LONDON: UNIVERSITY COLLEGE UNION SOCIETY.—The Foundation oration on Lister and his Work will be delivered by Sir Rickman J. Godlee, Bart., P.R.C.S., in the Botanical Theatre, on Thursday, March 13th. The reception by the President will take place at 8.30 P.M., and the oration will be given at 9 o'clock. Academic dress will be worn.

Clinical Notes:

MEDICAL, SURGICAL, OBSTETRICAL, AND THERAPEUTICAL.

A CASE OF SYRINGOMYELIA, WITH A NOTE ON THE CAUSES OF WEAK SHOULDER.

BY V. ZACHARY COPE, M.S. LOND., F.R.C.S. ENG.,
SURGEON TO OUT-PATIENTS, ST. MARY'S HOSPITAL, W.

THE patient, aged 40, was sent by Dr. Galway Murray to my out-patient clinic on Dec. 11th, 1912, complaining of an increasing loss of power to raise the left arm. He gave no history of accident or illness. The symptoms had begun six weeks previously.

On examination he was found to be a man of average nourishment, without any special signs of muscular wasting over the affected region. He was unable to raise the left arm above a right angle in the abducted position. Fibrillary tremor was noticed in the deltoid, which was evidently weaker than normal, though the fibres were seen to be contracting to the voluntary stimulus. There was some slight creaking felt in the shoulder-joint. There was no tactile anæsthesia detected, though careful test was made, especially in the area of exclusive supply of the circumflex nerve. In flexion and extension at the shoulder the scapula appeared to move with the arm. The serratus magnus was acting, as also the trapezius. No abnormality was discovered in the triangles of the neck. The patient gave no history of syphilis, and his occupation did not bring him into contact with lead. The pupils reacted to light and the knee-jerks were brisker than normal. There was no ankle-clonus or plantar extensor response.

The above investigation excluded the surgical causes of weak shoulder, so I sent the patient to Dr. Wilfred Harris, my colleague who is in charge of the neurological department at St. Mary's Hospital, and his report was as follows: "There is paresis of the left deltoid and infraspinatus, and a wide area of analgesia from the region of the second cervical down to the fifth lumbar nerves, escaping the arms to a certain degree. There is partial reaction of degeneration of the deltoid of which the reaction to faradism is diminished, and the anodic closure contraction is equal to the cathodic closure contraction."

The diagnosis was clearly syringomyelia, and confirmation of this was forthcoming in the fact that the patient could not distinguish between heat and cold when applied over the cutaneous areas presenting the analgesia, especially in the shoulder regions. In the dorsal region and over the arms (with the exception of the deltoid area) the sensory abnormalities were but slightly marked. No other abnormal symptoms were present in this case, which illustrates syringomyelia in a comparatively early stage. The interest lay in the diagnosis from other causes of weak and stiff shoulder. The causes of a chronic weak or stiff shoulder may be roughly classed under five headings: 1. Intra-articular causes. This includes the various forms of chronic arthritis, especially tuberculous, rheumatoid and osteo-arthritis. Also adhesions following traumatic synovitis. 2. Juxta-articular causes. This includes subacromial bursitis, old unreduced dislocation at the shoulder-joint, and upward subluxation of the clavicle at the acromioclavicular joint. 3. Muscular causes. 4. Neuritic causes. 5. Central nervous causes.

In the elucidation of this case the first group of causes were excluded by the absence of pain and the free passive movement permitted at the joint. The second group were negatived by the absence of any previous injury. Of the causes under the fourth heading a mixed neuritis would have been accompanied by pain owing to the involvement of the sensory fibres, whilst a pure motor neuritis is only found in lead poisoning, of which there was no indication. Injury to the circumflex nerve was excluded by the absence of any history of trauma. In deciding between intrinsic muscular causes and central nervous causes the electrical reactions of partial degeneration pointed to a spinal lesion, which was also suggested by the increased knee-jerks. The dissociated

anæsthesia served to exclude progressive muscular atrophy and pointed definitely to syringomyelia. Such a case as this shows the great importance of a careful neurological examination in any doubtful case of weak shoulder.

Electrical treatment has resulted in very marked improvement, for the man can now easily lift up his left hand well above the head.

Weymouth-street, W.

NOTES ON A CASE OF TYPHOID FEVER, WITH PARTIAL PARALYSIS OF THE THIRD NERVE AS A COMPLICATION.

BY JOHN LAMBIE, M.D. GLASG., D.P.H.,
ASSISTANT MEDICAL OFFICER OF HEALTH, RHONDDA URBAN DISTRICT.

HAVING read an account of a case in which paralysis of the third nerve occurred as a complication of typhoid fever, in THE LANCET of Jan. 25th, p. 261, I have decided to publish these notes on a case presenting many similar features.

The patient, a girl aged 16, was admitted to the Rhondda Isolation Hospital on Oct. 29th, 1912, this being about the fourteenth day of illness. Previous to admission there had been shivering, headache, and anorexia, but no diarrhœa. On admission the patient appeared fairly well nourished, the tongue was covered with a coating of white fur and was moist, and nothing abnormal was detected in the abdomen. Rhonchi were heard all over the lungs, and occasional moist crepitations at the bases. The heart sounds were normal.

From the day of admission till Nov. 15th the progress of the case was uneventful, but no sign of defervescence appeared. During this time the temperature never fell below 100° F., and varied between that limit and 102·8° which was the highest temperature recorded. The temperature fluctuated daily, usually about one degree, but sometimes as much as two degrees. The pulse-rate varied from 104 to 116 per minute. The patient's mind remained perfectly clear during this period. The bowels were constipated throughout, and were opened regularly by enemata. The Widal reaction was positive, but no other definitely positive sign of typhoid fever was manifest throughout the illness. On Nov. 15th the patient vomited several times, and during the following day there was a good deal of retching, but no further vomiting. On the 17th very severe headache was complained of, and from that time on the patient suffered from considerable mental derangement, unlike the usual typhoid delirium, but resembling the form of mania which one occasionally sees in typhoid patients, especially in young subjects. She was perverse and childish, and slept but little. On the 20th an erythematous rash appeared on the face and arms, which was possibly accounted for by the fact that bromides had been administered.

On Nov. 21st photophobia was noted, and there seemed to be a general hyperæsthesia, the patient calling out loudly when touched. On the 22nd ptosis of the right eyelid appeared. Both pupils were dilated, but reacted to light and to accommodation, and no squinting was observed. There appeared to be paralysis in some degree of all four limbs, and the action of the diaphragm seemed to be partially in abeyance, but no pulmonary symptoms supervened. The knee-jerks were absent. There was retention of urine, necessitating the use of the catheter, and an enema given the day before having failed to act another was now given, but this again failed to produce the desired result. Two grains of calomel were then given, and this dose was repeated four hours later. On the 23rd the patient was in a condition of stupor, but had passed urine several times incontinently, and several small loose stools also incontinently. On the morning of the 24th ptosis of the left eyelid was noted, and both pupils were widely dilated and fixed. Difficulty in swallowing was noted during the night, and up to the time of death. The patient died at 2 P.M. on the 24th. There was no rise in the pulse-rate till the 22nd, when it began to rise and continued to rise till the end came. The temperature was on the average lower after the onset of headache on the 17th than before, and at no time did it reach the previously mentioned limit—viz., 102·8°. Permission to make a post-mortem examination was not obtained.

Observations.—A post-mortem examination in this case

was very desirable, both for the purpose of confirming the diagnosis of typhoid fever, and for ascertaining the nature of the lesions giving rise to the nervous symptoms observed. The parents were unwilling, however, and it was not considered advisable to press the matter. With regard to the diagnosis, up to the onset of the nervous symptoms I regarded the case as one of typhoid fever, and that of a favourable type. The nervous symptoms as described, however, I am unable to satisfactorily account for. There was no retraction of the head, and Kernig's sign was absent. The mental condition of the patient rendered it difficult to determine the exact amount of paralysis present. There were, however, flaccidity and entire absence of motion in both legs and in the left arm, but some power of motion was observed in the right arm.

I have to thank Dr. J. D. Jenkins, medical superintendent of the Rhondda Isolation Hospital, for permission to publish these notes.

Pentre, Glamorgan.

Medical Societies.

ROYAL SOCIETY OF MEDICINE.

CLINICAL SECTION.

Discussion on Cervical Rib.—Exhibition of Cases and Specimens.

A MEETING of this section was held on Feb. 14th, Sir WILLIAM OSLER, the President, being in the chair.

Dr. F. WOOD JONES opened a discussion on Cervical Rib. Speaking from the anatomical point of view, he said that a review of the vertebrate series warranted the assertion that the rib series, which was complete in limbless animals, tended to become incomplete with the development of limbs. A snake had no limbs, and possessed neither neck nor waist; but with the rising functional importance of the limbs in lizards a neck and a waist were developed, and ribs became limited to a definite portion of the body. The ribless neck and ribless loins were the outcomes of the functional perfection of the pectoral and pelvic limbs, or, in other words, the ribs were stripped from the vertebral column opposite those segments from which the limb buds arose. The reason for this was seen in the developing embryo. At first, segmental nerves ran to a limb bud which was coterminous with the body segments from which it arose. But as growth proceeded the segmental nerves grew apart at their origin from the spinal cord, and became focused in the limb bud which ceased to keep pace with the antero-posterior growth of the body segments from which it arose. In this manner the mesoblastic intersegments became aborted between the converging nerves which formed the limb plexus, and mesoblastic intersegmental ribs were shorn from the neck and loins by the outgoing limb nerves. There was thus an antagonism between the development of a limb plexus and the development of ribs extending over that area from which the limb bud arose. The normal antagonism was manifested by the aborted costal elements of the normal cervical vertebrae, by the deep nerve groove which abbreviated the seventh cervical rib, and by the groove (so-called sulcus subclaviæ) which marked the upper surface of the normal first thoracic rib. A limb bud might arise nearer to the head or nearer to the tail than was normal, and its limb plexus might be of the prefixed or the post-fixed type. With either variation costal anomalies might result. Pre-fixation might proceed so far that the plexus readjusted itself at a higher level with the development of a seventh cervical rib. Post-fixation might proceed so far that the plexus readjusted itself at a lower level with the abortion of the first thoracic rib. Or there might be a disharmony between nerve movement and rib development, and brachial neuritis might be caused by an abnormal pressure produced by a somewhat post-fixed plexus on a first thoracic rib or by a somewhat prefixed plexus on a seventh cervical rib. The antagonism between plexus formation and rib development accounted, by the pressure exerted on the lowest cord of the plexus, for the sensory and muscular symptoms displayed in such cases. It also accounted for the vascular symptoms; for the sympathetic vascular nerves ran for the most