absorption from, the intestine. Pre-operative and post-operative gastric lavage should be the rule in cases of obstruction, and spinal anaesthesia was the method of choice for operation. Evisceration was to be avoided, and was seldom necessary if collapsed bowel was at once sought for and traced upwards. Opening and draining the intestine was necessary only in cases with marked distension, and then it was usually advisable to leave a valvular tube jejunostomy for some days.

Closed loop obstruction was seen in its purest form in the human subject in the obstructed appendix. It was not yet sufficiently appreciated that the locking up of faecal matter in the vermiform appendix by the impaction of a concretion in a stenosed area or at a kink led inevitably to gangrene. Experiment had shown conclusively that this was an inevitable sequel. It was only by regarding such cases as examples of intestinal obstruction demanding the immediate surgical help which was the accepted treatment for this emergency that we could lower the death rate from the perforated gangrenous appendix—the end-result of such obstruction. Of the 3,000 deaths occurring in this country every year from so-called appendicitis; Professor Wilkie believed that over two-thirds were examples of primary obstruction of the appendix which had been allowed to go on to perforation.

Occlusion of the blood supply to a segment of intestine led to a potential obstruction, but death in such cases resulted from a combination of peritonitis and poisoning from the absorption of autolytic products.

In *strangulation*, the most urgent because the most fatal form of obstruction, there were many factors—obstruction of lumen, closed loop, interference with blood supply, and, lastly, compression or twisting of the mesentery with associated shock. Early operation under spinal anaesthesia offered the best hope of success in these desperate cases. Adynamic ileus was a term which must be used with caution, because it was liable to be confused with true obstruction. Abdominal distension occurring in cases of peritonitis, particularly if associated with colicky pains, was frequently due to a definite organic obstruction of the ileum from plastic adhesions in the pelvis, and for this a jejunostomy was a life-saving operation. For true adynamic ileus, usually found in generalized peritonitis, active surgical intervention could do little, and probably fomentations, subcutaneous saline, morphine, and possibly hypertonic intravenous saline and enemata of bile offered most hope of relief.

Reports of Societies.

THE TREATMENT OF URAEMIA.

A MEETING of the Section of Therapeutics and Pharmacology of the Royal Society of Medicine, on February 11th, was devoted to an informal discussion of the treatment of uraemia, Dr. GEORGE GRAHAM occupying the chair.

Professor F. LANGMEAD, introducing the subject, said that, so far as the physician was concerned, uraemia as met with in Bright's disease was a late symptom of an incurable disease. There were different views as to what ought to be included in the term "uraemia," since one school would include all uraemia which could be put down to toxaemia, even renal retinitis, while the other would restrict it to cases with cerebral and gastro-intestinal symptoms, including vomiting and diarrhoea. At present the etiology was shrouded in hypothesis, and it was necessary to unravel the part played by toxins and the part played by supernormal blood pressure and acidaemia. His chief lines of treatment of uraemia consisted of purgation, diaphoresis, and venesection, the last-named being the most important. Care was necessary in prescribing purges for the uraemic patient; the speaker often preferred jalap, since it acted quickly as a cathartic. The evil effect on the kidneys of saline solutions had to be remembered. With diaphoresis he did not hesitate to combine hot packs or radiant heat baths with pilocarpine, if there was no respiratory trouble or heart failure. When twitching and convulsions were present he used morphine. He had seen patients benefit from lumbar puncture, though some physicians objected to it. Diuretics containing mercury were dangerous. In a case of large white kidney, with considerable oedema, 5 per cent. glucose solution resulted in making the patient much brighter, and largely reducing the oedema. Professor Langmead asked whether members had found that giving parathyroid extract and calcium lactate had any effect other than increasing the blood-calcium content. Also, had members employed the system of blood-washing which had been recommended by some Continental authorities—namely, removing 500 c.cm. of blood at a time, and after dialysing it through a collodion membrane (taking care to prevent coagulation) returning it to the circulation? Thyroid extract had been used for diuresis, but since it enhanced the metabolism it did not seem desirable to use it in uraemia.

Dr. E. P. POULTON described a case in which the administration of 15 grams of urea three times a day caused the blood urea to rise to 40 or 50 mg., and the daily urine output to be increased from 500 or 600 c.cm. to 1,500 c.cm. during all the time the urea was continued. It was important to ensure that the amount of albumin lost was made up in the diet taken. He reported various instructive cases.

Dr. W. LANGDON BROWN said the vomit in cases of uraemia contained a large quantity of ammonium salts; he regarded such symptoms as an alternative method of excretion. Skin rashes also indicated such an alternative method, and he had seen them occur in patients with a high blood urea figure. He would include under uraemia three groups: cases with convulsions and amaurosis, those with uraemic asthma, and those with other cerebral manifestations. The first group he attributed to some general toxic condition, in which the kidney might not share, because such symptoms were met with in eclampsia and in the case of the degenerative toxaemic kidney; though dramatic, convulsions were most amenable symptoms. Cases in the third group were associated with a rise of the blood urea content. Uraemia was not due to the urea in the blood; it might be caused by some toxic amino-acid. The speaker did not like using pilocarpine, which, he thought, hastened oedema of the lungs, nor were the newer diuretics containing arsenic and mercury safe in uraemia. In these cases the protein should be cut down at an early stage; fruit juice and sugar were good substitutes for meat. A septic focus often played a very important part in precipitating disaster in uraemic patients. He did not think diaphoresis was wise unless definite oedema was present, since it was a trying process for the heart.

Dr. IZOD BENNETT did not regard the vomiting which was prominent in cases of uraemia as a compensatory or physiological mechanism. He considered that in deaths from uraemia the factor directly responsible for the death was dehydration following excessive vomiting. He had checked vomiting by using barbituric acid preparations, and he had even used morphine for the purpose, since sometimes it seemed to save the life of a patient apparently dying from uraemia. He endorsed the value of venesection in acute cases. Dr. O. LEYTON agreed that the amount of urea in the blood was not necessarily a measure of so-called uraemia. He had seen patients with a high blood urea figure who had no symptoms at the time; there needed to be a very high figure of concentration before urea caused symptoms. Administration of the lactic-acid-forming bacillus was beneficial as regards control of the intestinal flora, but a reliable preparation must be employed. Dr. A. A. OSMAN said that in cases of chronic parenchymatous nephritis, with considerable oedema but without much evidence of nitrogen retention, treatment with large doses of alkalis resulted in a satisfactory clearing up of the oedema. All these patients had had symptoms more than a year before the commencement of the treatment. In patients suffering from definite uraemic attacks he had found lumbar puncture extremely useful, and he had never seen harmful effects follow the procedure. Sometimes a patient appeared to have come out of coma because of hypertonic saline injections which had been given; at any rate such patients had been able to live at home for six to nine months before having another attack.

The CHAIRMAN expressed his belief that venesection was very good, but it should, he considered, be restricted to early cases; he had seen patients in late stages who had only 30 to 40 per cent. of haemoglobin, and in them it would be a risky procedure. They stood in more need of blood transfusion, if it was judged wise to give it. When a patient had uraemia it was important to determine the hydrogen-ion concentration of the blood and the patient's reserve of blood. Such tests as he had mentioned occupied considerable time, and so were less often employed than was desirable, but he considered them necessary. Vomiting was apt to increase the alkali reserve. Dr. A. G. PHEAR agreed with Dr. Langdon Brown that pilocarpine was a dangerous drug in these cases, but it was useful when patients were oedematous and needed to have sweating induced. In some instances a hot bath caused the temperature to rise, but there was no sweating, the skin remaining dry and stubborn. The giving of 1/10 or 1/20 grain of pilocarpine to such patients often acted on the heat centre, causing the desired sweating. Lumbar puncture was a valuable proceeding in a limited number of cases, including acute cases with cerebral symptoms, considerable oedema, and convulsions; its effect in reducing the intracranial pressure was chiefly mechanical. Sir WILLIAM WILLCOX emphasized the importance of the physician making sure that he was actually dealing with a case of uraemia, for very similar symptoms were present in cases of advanced morbus cordis, impairment of liver function, and arterio-sclerotic changes in the vessels of the brain. Vapour baths to promote the action of the skin were dangerous in these cases unless the heart was in good condition; he had seen patients die from the effect of them. Elimination by the bowel could be greatly helped by colonic irrigation. Minute doses of morphine could be risked in a case of uraemia if a fair quantity of urine was being passed. For the headache of uraemia large doses of bromides were valuable.

THE RELATION OF NERVOUS SYMPTOMS TO NERVOUS LESIONS.

AT a meeting of the Section of Neurology of the Royal Society of Medicine on February 13th, with Dr. GORDON HOLMES in the chair, a short paper was read by Dr. S. A. KINNIER WILSON on the relation of nervous symptoms to nervous lesions.

Dr. Kinnier Wilson said that at one time he had imagined that clinical conceptions based on clinical differentiations were sufficient for all neurological purposes, but it did not take him long to discover that a purely clinical arrangement was obviously unsatisfactory because a whole group of symptoms of the same kind could arise at different levels in the central nervous system, and as a result of entirely different pathogenic action. It might be thought that epidemic encephalitis was one and indivisible, but its clinical manifestations were innumerable. The clinical-anatomical method had given rise to discoveries and advances of the first importance, but it was rather out of date; it was called, rather contemptuously, the necrological or "mortuary school" of medical thought. He dated his own awakening in the matter largely to the late Sir James Goodhart's paper entitled "The passing of morbid anatomy." Neither clinical nor pathological classification offered any advantage from the standpoint of nosology; the only scientific one would be based on etiology, and further research was necessary. The time had passed when clinical syndromes and pathological complexes could be collated and termed a disease. Some might go as far as Sir Humphry Rolleston and say that there was no such thing as disease, or, rather, that a disease was nothing else than a mental picture, a conception in the mind. At any rate, insuperable difficulties would be found in establishing morbid entities unless it could be proved that a given causative agent produced the same reaction, the same clinical course ensuing, with amenability to the same form of treatment.

Physiology knew only three classes of stimuli: those which excited, those which depressed, and those which first excited and then depressed; clinical symptoms should be considered, therefore, as either excitation or depression of function, or as a series in which depression followed excitation. Without exception all were functional. A symptom was the expression of physiological reaction, and thus it could not be specific unless reactions themselves were imagined to be specific, an impossibility when the diversity of agencies and systems was considered. In itself a symptom had no constant relationship to underlying structural relations; a patient with disseminated sclerosis might have ankle clonus one week and not the next. Symptoms *per se* represented dynamic alterations in function, which might or might not coincide with structural change. Further, symptoms themselves, whether due to dynamic or static (structural) change, could not be differentiated. What exactly was expected to be found in a case of neuralgia? It was universally accepted that neuralgia was an extremely prominent neurological symptom, but what alteration was to be found to correlate with neuralgia? The pain must be produced by the stimulation of a normal sensory mechanism, which must be in a state of health, and not of depression. What was expected to be found in a case of tetanus? Recent post-mortem examinations of cases had shown that there was no change at all in the spinal cord cells; that was only to be expected, because in tetanus there was overfunction or release of function of these cells. No constant relationship could hold between the size of a lesion and the amount or mass of functional disturbance. In epilepsy, for example, there was an extreme disorder of function, but with what lesion could it be correlated? Twenty minutes after an epileptic attack the patient was normal. If the cells were then examined nothing abnormal would be found. He did not deny that lesions could be found in epilepsy; he only maintained that there was no correlation between a specific lesion and a specific discharge of function.

The most serious error in the clinical-anatomical method was the omission of the time factor. A few years ago there were many cases of post-encephalitis with inversion of sleep rhythm; but where was this inversion localized? It was not localized anywhere in space, but in time. Since no specificity attached in a general way to nervous symptoms, there arose the homologous question whether pathological changes could be considered specific. His own view was that such changes were reactions to noxious agents. There was a pathological reaction which was common to a number of different conditions, and therefore could not constitute a disease in the scientific sense. The bearing of these problems would not escape the student. They would impress him again with the desirability of regarding disease processes, whether of activity or depression, as indications of vital effort to cope with noxae. Many symptoms were signs of health, and many lesions were attempts at repair.

After a brief discussion Dr. GORDON HOLMES, the president, said that what Dr. Wilson had brought forward was a very close repetition of the teaching of Hughlings Jackson brought up to date. When Hughlings Jackson published most of his work, little was known of modern neuro-pathology, and many of the finer lesions associated with symptoms were not recognized, but his teaching still held good.

Radium and Nervous Tissue.

Dr. E. A. CARMICHAEL brought forward, on behalf of Mr. J. Paterson Ross and himself, an experimental study conducted at St. Bartholomew's Hospital, with a view to ascertaining the effect of radium on cerebral tissue. Radon seeds, with a 5 mm. platinum filter, were placed on the dura mater of rabbits, and the animals were killed at intervals of from three to twenty-four hours. The first thing to occur was a definite change in the staining properties of the cells, followed by congestion of the vessels, fatty degeneration of their endothelium, and finally haemorrhages. In another series of experiments radon seeds, with a 3 mm. platinum filter, were employed, and the animals were killed after periods extending from 68 hours to 60 days. Up to 212 hours, which corresponded to the time of activity of the seed, the size of the lesion was definitely progressive. At the end of 40 days the lesion changed in character, the haemorrhage disappearing and

being replaced by a necrotic area. A further series of experiments, not yet completed, was undertaken with radium needles, which were left in position in the cerebral cortex for long periods. At the end of seven days no effect whatever could be discovered macroscopically or microscopically. Later a lesion appeared, which slightly increased. A point to be noted was that glial tissue actually proliferated under the influence of radium; this raised the question whether it was advisable to use radium in gliomata.

CORONARY THROMBOSIS WITH CARDIAC INFARCTION.

AT a meeting of the Aberdeen Medico-Chirurgical Society on February 6th, with the president, Dr. CHARLES FORBES, in the chair, a demonstration of clinical cases was given. Dr. W. F. CROLL showed a case of coronary thrombosis and cardiac infarction in a man aged 55; the symptoms were as follows.

In September, 1925, the patient began to have what he described as a gasping feeling on negotiating a fairly steep incline, and later pain, definite across the chest, developed, which ceased if he stood still. Under medical advice the patient took things easily, and he was without pain for several months. In December, 1926, while walking more quickly than usual, he was seized with very severe pain in the lower part of the chest and along both arms. The pain over the sternum persisted for a week, in spite of complete rest in bed. On examination the pulse rate was found to be about 120 and regular, the blood pressure 105/60, and the vessel walls normal. The apex beat could not be palpated, the heart sounds were almost inaudible, there were no cardiac murmurs, and no pericardial rub was heard. There were crepitations at the bases of both lungs; the liver and splenic dullnesses were normal, and the Wassermann reaction was negative. The urine contained no albumin. At a later examination in March, 1927, the pulse rate was still fast (101), and the blood pressure 110/20; no apex impulse could be felt. In all areas the first cardiac sound was very faint and the second fairly good. There were no murmurs and the cardiac rhythm was normal. An electro-cardiogram showed inversion of T in leads I and II, a QRS of low voltage, and a left-sided preponderance. At a recent examination, the electro-cardiogram showed increased inversion of the T wave in lead I, an indefinite T in lead II, and an increased voltage of the QRS in all leads. The diagnosis of coronary thrombosis with cardiac infarction was made from the following symptoms and signs: the character of the pain, which was over the lower part of the sternum and adjoining parts of the chest, and radiated to both arms; the long duration of the pain; the persisting rapid pulse; the low blood pressure; the distant cardiac sounds; and the characteristic electro-cardiogram.

Dr. Croll said that the prodromal pain which the patient experienced in 1925 indicated the beginning of the thrombosis, while the attack of severe pain in December, 1926, pointed to complete occlusion of the artery. He showed electro-cardiograms illustrating the characteristic changes which were diagnostic of cardiac infarction, and referred briefly to the prognosis and treatment of this condition.

Treatment of Bronchiectasis.

Dr. R. J. DUTHIE showed a man, aged 31, who was suffering from bronchiectasis. He described the history and treatment, and gave the following clinical details.

The patient said that a tooth had passed down the windpipe in January, 1928; cough and sputum had commenced gradually and had persisted. In November, 1928, he had a moderate haemoptysis followed by severe haemorrhage on three occasions in June, 1929. On admission to hospital in August, 1929, he was coughing up about a cupful of purulent blood-stained sputum every morning. No tubercle bacilli were found after repeated examination, and there was clubbing of the fingers. An *x*-ray examination showed what appeared to be a foreign body in the left lung; no foreign body could be recognized, however, by bronchoscopic examination on account of bleeding from the left bronchus. A few days later he had repeated copious haemoptyses, and the physical and radiological signs pointed to the bleeding coming from the right lung. This lung was partially collapsed by artificial pneumothorax; since the collapse there had been no further haemoptysis, the sputum was greatly reduced, and the general condition much improved.

Dr. Duthie suggested that the lung collapse should be pushed further by increasing the intrapleural pressure. If pneumothorax successfully stopped cough and expectoration, and if an injection of lipiodol revealed no severe pathological changes in the left lung, permanent collapse of the right lung, either by phrenic evulsion or paravertebral thoracoplasty, might be indicated at some future date.

Other Clinical Cases.

Dr. JOHN CRAIG showed a case of spinal tumour in a girl, aged 10, who had been seen at a previous meeting of the society. In the interval the tumour had been exposed by Mr. A. Mitchell, and diagnosed as an intramedullary glioma, a finding which was confirmed microscopically. It had been impossible to remove the tumour, but the spinal decompression had greatly relieved the symptoms, so that the child could now walk about without support. Deep *x*-ray therapy was being applied to the growth.

He also showed a long-standing case of melaena neonatorum in a girl, aged 3, who was now in perfect health. When 3 days old she had lost much blood by the bowel and by the mouth. An intramuscular injection of 25 c.cm. of maternal blood into each thigh had an immediate and good effect, the infant remaining well until the age of 2 months, when jaundice appeared. This symptom persisted for two months. Towards the end of the first year the child became very anaemic, although no blood was being lost by the bowel. A blood examination showed 20 per cent. haemoglobin, red cells 2,000,000 per c.mm., and white cells 17,000, of which 10 per cent. were myelocytes. Transfusion of maternal blood by the longitudinal sinus was performed on two occasions, with an interval of three weeks; the child made an uninterrupted recovery and was now a normal healthy girl.

A third case shown by Dr. Craig was of chronic duodenal ulcer in a boy aged 5.

PROGNOSIS AND TREATMENT IN NEPHRITIS.

AT a meeting of the Manchester Medical Society on February 5th, with the president, Professor A. RAMSBOTTOM, in the chair, Dr. H. MORLEY FLETCHER of London gave an address on nephritis, in which he dealt chiefly with prognosis and treatment.

Dr. Morley Fletcher reviewed briefly certain points connected with the structure and functions of the kidney, indicating the channels by which toxins and organisms might reach the kidney—namely, the blood vessels, lymphatics, and ureters. The importance of lymphatic conveyance seemed to have been underestimated until comparatively recently. He called attention to that form of acute nephritis which was associated with severe haematuria, and occurred in children and young adults, being known as acute haemorrhagic nephritis. In this type, oedema, if present, was slight, and the urine contained fewer leucocytes than in the ordinary forms of acute nephritis. It usually accompanied acute tonsillitis, also pneumonia and glandular fever, and the prognosis was generally favourable. This condition must be distinguished from simple haemorrhage from the kidney, sometimes called "essential haematuria," and from the form of nephritis which was associated with purpura. The speaker also referred to the rare form of haemorrhagic nephritis of hereditary, familial, and congenital origin which was nearly always associated with deafness, and was probably due to some form of streptococcal infection. As regards the treatment of acute nephritis, stress was laid on the importance of giving, during the first few days, a moderate amount of fluid in the form of water, lemonade, and fruit juice, but no milk, in order to rest the inflamed organ as much as possible. In a few days the diet might be increased by the addition of more fluid and carbohydrates, with fruit and vegetables, but the amount of milk must not exceed half a pint. Further increase of the diet would depend upon the condition of the urine, the amount of oedema, and the results of renal function tests. In discussing chronic nephritis Dr. Morley Fletcher referred to the comparative rarity at present in the large London hospitals of chronic parenchymatous nephritis, or the hydraemic type of renal disease associated with anasarca, as compared with its frequency twenty years ago, and asked whether similar observations had been made in Manchester. The prognosis and treatment of chronic nephritis was dealt with in a consideration of the hydraemic and azotaemic forms. The condition known as nephrosis was described, and recent views with regard to its pathology were discussed. The paper concluded with remarks bearing on the treatment of uraemia.