similar symptoms, their illness, however, not resulting in paralysis. He also referred to epidemics of the disease which had been described by several observers. The poison usually falls with greatest force on the anterior nerve cells, but is not always confined to them, for convulsions occasionally occur, pains in the limbs are not infrequent, and spasticity may result. He then referred to another disease—insular sclerosis—and said the view enunciated by Pierre Marie was apparently gaining ground—viz., that this disease is really an infectious one. Patches of myelitis undoubtedly do occur as a sequel to infective fevers. But while not claiming that this was the same condition he thought that the periods of remission in disseminated sclerosis, so similar to those occurring in constitutional syphilis, indicated the infective character of the disease. Tabes was another disease the close connexion of which with syphilis no one could doubt. It was an interesting question how far tabes was a result of specific infection and he suggested that not syphilis merely but also chancroids might be the infection preceding tabes. We know that neuritis may follow different kinds of microbic infection and probably myelitis also may do the same. There seems no reason why tabes should not similarly follow different kinds of microbic infection.

Dr. DERCUM (Philadelphia), after thanking Dr. Buzzard for his suggestive address, remarked that the action of toxins was probably a general one on all the tissues, with a local expression at some very susceptible part.

Dr. JOSEPH COLLINS (New York) had found in the examination of cases from the Vermont epidemic that the nervous system generally was affected, yet no specific organism was found. He did not agree with the view that disseminated sclerosis is infective. He thought a more probable explanation was that it was a result of congenital abnormalities. The nutritional balance of the cells of the nervous system was also of much importance in reference to nervous disease.

Dr. F. J. SMITH (London) related two cases of apparently some infective nervous affection after a septic wound and suggested that in such cases where the symptoms pointed to peripheral rather than central affection the transference of symptoms from one side to the other should be regarded as the result of nerve influence.

Dr. JOHN M. MACCORMAC (Belfast) remarked on the great divergence of opinion as to the connexion between tabes and syphilis and expressed the opinion that the changes in the nervous system in disease are chemico-physical in nature.

Dr. J. A. ORMEROD (London) thought that in tabes there must be more than mere microbic infection, probably personal susceptibility. For it is well known that tabes occurs ever so much more frequently in men than in women and cases of Friedreich's ataxy became much worse after an illness. Indeed, in some cases the illness seems to evoke the first symptoms and he would be inclined to think the microbic origin of disseminated sclerosis and even of infantile paralysis was "not proven."

Dr. R. A. FLEMING (Edinburgh) thought that in peripheral neuritis the vaso-motor nerves probably suffer and so there is exudation into the sheaths with pain and paralysis.

Dr. MOTT (London), in detailing the changes which he had found in two cases of beri-beri, remarked on the direct effect apparently exerted on the cardiac muscles by the poison of this disease and on the severity of the clinical symptoms as compared with the amount of change found in the nervous system.

Dr. FERRIER (London) remarked on the great suggestiveness of Dr. Buzzard's address, but was inclined to regard the infective theory of the origin of disseminated sclerosis as seductive but not proven. Of the close connexion between syphilis and tabes dorsalis and also general paralysis of the insane he had no doubt.

Dr. ALEXANDER BRUCE (Edinburgh) had found in a case he had examined recently, in fresh foci of disseminated sclerosis, a blood-vessel in the centre and he thought that the change probably spread from this vessel and was caused by some toxic substance which had the effect of dissolving the myelin. The change occurred symmetrically round the vessel and evidently spread from it.

Dr. E. F. TREVELYAN (Leeds) described a condition of disseminated myelitis found by him in dogs which died after canine chorea and distemper and he described a case in which death occurred eighteen days after exposure to cold. There was suppuration in the back muscles, empyema, and myelitis.

The PRESIDENT thought that toxins in many instances gave rise to changes, but he thought that toxins were not always of microbic origin but were produced sometimes inside the body. He referred to the fact that second attacks of infantile paralysis were practically unknown as evidence of infective character. He thought herpes zoster was probably microbic and facial paralysis seasonal. He referred to muscular atrophy (myopathy) with little change in the nervous system as occurring as a sequel to sprue and he was inclined to regard disseminated sclerosis as a result of intoxication.

Dr. BUZZARD replied on various points raised, and the PRESIDENT, in conclusion, thanked him for his introductory address.

Dr. BEEVOR (London) read a paper on

The Action of the Latissimus Dorsi in Hemiplegia.

He said that this muscle had three different actions—in moving the arm, in coughing voluntarily, and in coughing reflexly. In hemiplegia the voluntary action in moving the arm was usually lost on the paralysed side, the voluntary action and the reflex action in coughing both usually remaining. Sometimes, however, the voluntary action in coughing was also lost but the reflex action remained. He remarked on the possible usefulness of the condition as a diagnostic sign.

PATHOLOGY.

WEDNESDAY, JULY 27TH.

Dr. G. SIMS WOODHEAD (London) read a paper on

Post-Diphtheritic Paralysis.

He began by quoting statistics from a large number of cases of diphtheria or suspected diphtheria in which a bacteriological examination had been made at the Conjoint Laboratories, with the object of showing the large proportion of cases in which paralytic phenomena developed. The distribution of the primary paralysis was as follows. Out of 494 cases 185 were primary paralysis of the fauces, 197 of the oculo-motor muscles, 10 of other muscles, and 102 of primary paralytic heart failure. In regard to the time of onset important observations were made. Of the paralyzes of the palate the great majority occurred between the fifth and fifteenth days; none before the fourth. Of the oculo-motor paralyzes the bulk occurred between the fourth and seventeenth days; none before the fourth. Of the paralyzes of other muscles about one-half occurred between the tenth and fourteenth days; none earlier than the tenth. Of the cardiac paralyzes the bulk occurred between the fifth and tenth days, and in a few instances as early as the second day. In each of these groups the onset of the paralytic symptoms might be delayed for many days. It was obvious, however, that in the human subject the bulk of the paralyzes occurred at a comparatively early date and cardiac paralyzes earliest of all. The fact that the heart might be affected so much earlier than the other muscles appeared to indicate that the primary lesion probably occurred in the nerve-cells. These lesions were easily overlooked, because the changes were transient except in those cells which were completely degenerated. Sidney Martin, Meyer, and others had described segmental degeneration of the motor nerves, but in all probability such changes were of a Wallerian type. The toxin acted primarily on the nerve-cells which either degenerated or recovered at an early date; later the nerves for which these cells acted as trophic centres showed secondary degenerative changes, and only then was the paralysis revealed. After experimental inoculations of guinea-pigs for the purpose of testing antitoxins, paralyzes occurred more frequently than before, probably owing to the larger doses of toxin and antitoxin now employed. Madsen, who used the original method of testing—i.e., neutralising with one-tenth of an antitoxin unit—found that most of the cases of paralysis in guinea-pigs developed at the end of the third week. He pointed out that the paralysis, when it did occur, followed the local infiltration which resulted from the injection of an incompletely neutralised dose of toxin. It began in the posterior part of the trunk and in the hind limbs. Later the symptoms became more pronounced and the paralysis gradually extended forwards to involve the fore limbs also. The muscles of the head and neck were the last to be affected. In spite of the extreme muscular weakness the animals were still very lively and continued to eat as long as there was food within reach. Ehrlich, on the

other hand, observed that the bulk of the paralyses in guinea-pigs appeared at the end of the second week, but his observations were made on animals in which the neutralising amount of serum was one unit. In this connexion it was important to note that the diphtheritic poison was a highly complex body and appeared to include several substances possessing different properties: (a) a substance possessing lethal activity; (b) a locally irritating body; (c) albumoses which produced a marked reaction on the body temperature; and (d) a substance which had a special action on nerve-cells. Now, some of these substances combined with the antitoxin only after the lethal and locally acting toxins had been neutralised, and this was especially the case with the substance which produced paralysis. Hence the larger the total doses of toxin and antitoxin employed the greater would be the risk of some of the paralysis-producing substance being left over. In other words, there were more cases of paralysis when a theoretically ten times lethal dose was neutralised by the corresponding amount of antitoxin than when unit doses were used, and still more cases of paralysis when 100 times the theoretically lethal dose was used. The author's observations also showed that the amount and severity of the local swelling was no indication either of the probability of the occurrence of paralysis or of the rapidity of its onset.

Dr. MOTT (London) congratulated Dr. Woodhead on his valuable contribution to a subject of great interest and importance. He suggested that the diphtheria toxin acted in the first instance on the most vulnerable parts of the neuron—i.e., on the fine branching filaments of the dendron and on the terminations of the axis cylinder process. These parts were bathed in lymph and therefore specially exposed to the action of irritants diffused throughout the body. He had examined cases of diphtheritic paralysis in man and had found degenerative changes in the heart without degeneration of the vagus nerve. He thought the early cardiac failure was due to the effect of stress. The weaker the heart became the faster it had to work and it would therefore more readily succumb to the action of the toxin.

Professor BAGINSKY (Berlin) said that he had been led by his own observation to believe that cases of post-diphtheritic paralysis were less common than before. He had found advanced degenerative changes in all parts of the nervous system, including both nerve-cells and nerve-fibres. The muscular tissue of the heart and diaphragm showed also extreme degeneration. He thought that cerebral embolism was the most common cause of post-diphtheritic hemiplegia and said that such cases should not be included among the true diphtheritic paralyses. He thought that antitoxin treatment should be practised as early as possible in order to reduce the number of cases of paralysis in diphtheria.

Dr. GOODALL (London) said that cases of post-diphtheritic paralysis were undoubtedly more common than before, because under the antitoxin treatment the patients either recovered or lived longer. The number of fatal cases, however, had greatly diminished. Moreover, if antitoxin be given at a sufficiently early stage, the number of cases of paralysis would be lower instead of higher than before. In a number of cases of post-scarlatinal diphtheria in the Metropolitan Asylums Board's Hospitals the proportion of those affected by paralysis was considerably less than among the diphtheria cases admitted from outside in the same time. The former series of cases were under skilled observation from the beginning and in them the antitoxin treatment was commenced at an early stage.

Dr. SIMS WOODHEAD, in reply, said that he was now convinced that antitoxin by itself had no power of producing paralysis even in large doses. He believed that the heart failed most readily and earliest because it was the organ which had least rest. He had advocated this cause in his article on Tetanus in Clifford Allbutt's "System of Medicine." Overwork and malnutrition were the great factors even in those paralyses which appeared later. The poison did its work, but it was only when muscle and nerve were called into functional activity that the full extent of the damage was revealed. The practical outcome of the whole discussion was that the antitoxin should be used early, before degenerative changes had occurred, and that a sufficient dose should be given to neutralise not only the lethal action of the toxin but also its local and its paralysis-producing action.

Dr. J. LORRAIN SMITH (Belfast) read a paper on the

Pathological Effects of Breathing Oxygen at a High Tension.

Previous observers had noted that when animals were compelled to inhale oxygen at a greatly increased tension

a series of changes were produced which were partly functional—e.g., respiratory and circulatory disturbances—and partly of the nature of tissue degeneration. Bert in particular found that special effects might be produced in the nervous system resulting in well-marked convulsive or tetanic symptoms. In his own experiments on mice and small birds Dr. Lorrain Smith investigated the tissue changes which develop prior to the strychnine effect. At an oxygen tension between 70 and 80 per cent. of an atmosphere about 50 per cent. of the animals died in about a week. At 130 per cent. mice invariably died after an average exposure of 64 hours with a pneumonic condition of the lungs characterised by intense congestion and an exudate into the alveoli which was either fluid or granular, but not fibrinous, and which affected the whole lung simultaneously. At 180 per cent. death occurred very uniformly in about 24 hours, and at 300 per cent. the duration of life might be reduced to 5 hours. It was also noted that the absorptive power of the lungs was seriously impaired by exposure to high oxygen tension. At 300 per cent. small birds became very excited and in about ten minutes showed violent tetanic symptoms. It was found that this convulsive effect was not produced in birds by an oxygen tension of less than 270 per cent., whereas mice did not begin to show it until a tension of 450 per cent. was attained. The strychnine effect in birds was further found to depend upon the integrity of the lung tissue; for birds with damaged lungs react like normal mice, so that the onset of tetanic symptoms did not appear until a tension of 450 per cent. was reached. That this effect depended entirely on the tension and not on the quantity of oxygen in the blood was also shown by mixing carbonic oxide with the oxygen at different tensions, when it was found that the tetanic symptoms developed at the same tension as before. Hence it was concluded (1) that an oxygen tension of 80 per cent. and upwards would produce a pneumonic condition in the lungs of small birds and mammals and (2) that tetanic symptoms were developed at a tension of 270 per cent. in birds and at 450 per cent. in mice, and that their onset was independent of the quantity of oxygen in the blood but depended on the tension alone.

PHARMACOLOGY AND THERAPEUTICS.

WEDNESDAY, JULY 27TH.

Diseases of the Stomach.

After Dr. AFFLECK'S introductory address (which was published in THE LANCET last week) a discussion was commenced upon the Treatment of Diseases of the Stomach as relating especially to—(a) general treatment and diet; (b) the influence of drugs upon the gastric chemical processes; (c) the influence of the treatment of coexisting maladies; and (d) the indications for surgical treatment. It was opened by Dr. HERSHELL, who mentioned that perhaps the most difficult disease to treat was atony. The indication was to improve nutrition. Electricity was valuable especially if employed as shocks from an induction coil having a very thick primary wire and a very slow make and break. Hydrotherapy was useful, and so was lavage and douching of the stomach with alternately hot and cold water. Care must be taken in washing out or douching the stomach to avoid over-distension. This was to be attained by using a double tube with the efferent tube of much larger calibre than the afferent. The douche should be continued for 5 minutes, varying the temperature from hot to cold every 60 seconds. As for dietetics in hyperchlorhydria theoretically a proteid diet was indicated, but it did not succeed in practice, for the very fact that hydrochloric acid was necessary for the digestion of proteids in the stomach seemed to increase the already too free secretion of that acid. Therefore he recommended a carbohydrate diet predigested and neutralisation of the acid with alkalis. He did not much believe in artificial digestive ferments, with the exception of taka-diastase. As to drug treatment he had found very weak solutions of tannin useful in hyperchlorhydria.

Professor EWALD (Berlin) followed. After thanking the Association for the honour they had conferred upon him, he said that rational therapeutics were impossible without exact diagnosis. Purely local treatment was not enough. Referring to modern methods of diagnosis he mentioned the gastro-diaphane as being of great value. In cases of neurasthenic dyspepsia he relied chiefly upon treatment calculated to improve nutrition generally, such as electricity and massage. Passing to the subject of gastric ulcer he said that on the