The report of the radiologist has been identical in twenty-two cases. He states: "The stomach empties satisfactorily, and there is no delay in emptying. The food passes the duodenum normally, and there is no obstruction." Some irregularity of the duodenum was noticed in several of the cases. In two cases he reports: "The stomach was atonic and the duodenum was not satisfactorily filled." The first of these cases has been free from indigestion since his operation. The second keeps well unless he eats a heavy meal; then he feels some discomfort and flatulence.

A study of the after-histories of cases of perforated duodenal ulcer treated by simple suture shows that in the series under review 90 per cent. of cases required no further operative treatment. Apart from the cases already mentioned, no patient had reported himself as having relapsed up to the time of this record. In 10 per cent. of cases a secondary gastro-enterostomy was necessary on account of recurrence of gastric disturbance; the results of this operation have been completely satisfactory.

These four cases all gave a history of indigestion extending over a prolonged period, and at the operation were found to have chronic ulcers. The symptoms of indigestion recurred within a few months after perforation. It would appear from this that cases giving a long history of indigestion are less likely to show a permanent cure after simple suture than cases with a short history and mild symptoms.

Subsequent Treatment in Cases of Perforated Gastric Ulcer.

The after-treatment in these cases requires special consideration. It is undoubtedly a fact that perforated gastric ulcer is a more serious condition than perforation of a duodenal ulcer, both as regard its immediate and remote results.

Of the five cases of gastric perforation which came under my care two were proved to be malignant at necropsy and microscopic examination. A third case appeared malignant at operation and was in poor condition when he left hospital; he was alive two months after operation, but has not been heard of since that time. The fourth case, a female with perforation of a large chronic ulcer in the anterior stomach wall, still suffers from pain after food, but refuses further operation.

The last case has written to me recently from Birmingham, where he was in hospital under treatment. He had a perforation of the anterior stomach wall about one inch in diameter, and had suffered from indigestion for years. He states that he still suffers from flatulence and vomiting, and it would appear as if some further operative treatment will be necessary. It is not within the scope of this paper to discuss the relationship of ulcer and carcinoma of the stomach, but it must be realized that the types of ulcer found in the stomach and duodenum differ widely.

The duodenal ulcer is often a simple, small, punched-out ulcer, whilst the gastric ulcer may be a large chronic and spreading area. Further, the fact that a malignant ulcer of the duodenum is almost unknown, whereas the malignant ulcer of the stomach is of comparatively common occurrence, must be remembered. It is suggested, therefore, that it might be safer to regard all large and chronic ulcers of the stomach as potentially malignant, and treat them as such at a later date by some form of excision wherever practicable. This would be more likely to produce a permanent cure than a gastro-enterostomy alone, which operation is often unsatisfactory for this condition.

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¹ *Annals of Surgery*, November, 1910. ² Paterson, *Surgery of the Stomach*. ³ Moynihan, *Abdominal Operations*, vol. i. ⁴ Jacobson, *Operations of Surgery*, vol. ii, sixth edition. ⁵ *Lancet*, 1922, vol. i, p. 270. ⁶ *Annals of Surgery*, April, 1921. ⁷ *Annals of Surgery*, March, 1921.

IN the United States during 1920 lethargic encephalitis was given as a cause of death on 1,505 death certificates, a death rate of 1.7 per 100,000 of the population; in New York State 364 deaths from this cause were reported, a death rate of 3.5 per 100,000 of the population.

A RECENT report issued by the United States Public Health Service announces the demonstration of an anaerobic organism producing a soluble toxin which affects animals in a manner similar to that of the botulism organism, but is not neutralized by polyvalent botulinus antitoxin. The organism apparently differs markedly from the botulinus isolated in the United States, and is possibly more nearly related to the European type described by von Ermengem in 1912, although it differs from this in certain respects.

HEREDITARY MICROPTHALMIA.

BY

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In presenting the following history of a family affected with hereditary microphthalmia I will refer to statistics in order that the prevalence of congenital blindness may be appreciated. The most recent reference is in an article on "Cause and prevention of blindness," by Mr. Bishop Harman, in the *BRITISH MEDICAL JOURNAL*.¹ Of the total number of cases of blindness between 3 and 4 per cent. are congenital; Magnus, in Germany, found 97 cases in 2,528 cases, or 3.83 per cent.; Trousseau, in France, 3.04 per cent.; Dumont, in the Paris Blind Asylum, 3.7 per cent.; Oppenheimer, in New York City, 3.84 per cent. In the United States of America, according to the census of 1890, of 50,411 blind, 4,267, or 8.46 per cent., were recorded as congenitally blind.

Heredity and Consanguinity.

In the eleventh U.S.A. census 43,326 blind persons reported 8,342 blind relatives—that is, 19.25 per cent. had such relatives; whilst of 4,156 congenital blind, 1,608, or 38.7 per cent., had blind relatives. From this it appears that the proportion of blind relatives was twice as great amongst the congenital blind as amongst the blind from all causes.

Mr. Bishop Harman, referring to congenital defects, says: "A few are hereditary—that is to say, the same defect is found repeatedly in the same family for two or more generations."

The influence of consanguinity was investigated by Trousseau,² who reached the conclusion that, "independent of heredity, consanguinity does not play any rôle in the production of blindness." Magnus also holds that there is no proof that consanguinity is a cause of blindness.

That heredity is a cause of blindness seems certain, as the cases which I am reporting go to prove. The diseases of the eye which are most frequently transmitted from parent to offspring are:

Malformation—for example, coloboma of iris and choroid.
Microphthalmia.
Persistence of the pupillary membrane.
Congenital cataract and amblyopia.
Nystagmus.
Albinism and retinitis pigmentosa.

These may be transmitted in the same or like form. It is, perhaps, not out of place to mention here that myopia is frequently hereditary. Motais, quoted by Trousseau,³ stated that 67.5 per cent. of cases of myopia are hereditary.

The accompanying genealogical tree of the "H." family,

which goes as far back as the great-grandmother, shows that amongst thirty-four traced members there are eleven cases of congenital blindness. I was unable to obtain any definite history of members of the family farther back than the great-grandparents. All the affected members are males, the condition being transmitted through healthy females, as in haemophilia; the children of male members of the family are normal. In that branch of the family in which I have been particularly interested it will be noted that of four boys three are blind from congenital microphthalmia, whilst the twin brother of one of the boys is normal. The one female child is normal. The father and mother of this branch are normal persons. In fact, the parents of all the blind children throughout the family are apparently normal individuals. There is no evidence of consanguinity in the family and no history or evidence of syphilis. One of the boys of the "H." family is an epileptic; all the other children are free from congenital or acquired disease apart from microphthalmia.

One of the boys in the "H." family has been for some time in a blind institution. His report for last term states:

"Distinct speaker. Intelligent. Excellent memory and no mannerisms. Self-control good. Intends to study music. Eye report: Congenital microphthalmia."

Recently the other two boys were admitted to the same institution, but the epileptic, on account of this defect, had to be discharged.

Prevention.

From the genealogical table it will be seen that the tendency to transmit blindness by the females shows no sign of diminishing, but rather of increasing. Through how many generations the condition may be handed on it is impossible to conjecture. Marriage and parenthood should be avoided by the members of this family, especially by the females. The male members show no tendency to transmit the disease as far as can be ascertained.

I am indebted to Dr. J. J. Butterworth, County Medical Officer of Health, Lancashire, for permission to publish this case, and to Mr. W. H. Illingworth, of Henshaw's Institution for the Blind, Old Trafford, for allowing me to make use of the report on one of the cases.

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¹ BRITISH MEDICAL JOURNAL, November 9th, 1921. ² Norris and Oliver: *Sym. of Diseases of the Eye*, vol. ii, p. 441. ³ *Hygiène de l'oeil*, p. 134.

THE INTERPRETATION OF SYMPTOMS IN DISEASE OF THE CENTRAL NERVOUS SYSTEM.

ABSTRACT OF THE GOULSTONIAN LECTURES, DELIVERED BEFORE THE ROYAL COLLEGE OF PHYSICIANS,

BY

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LECTURE II.

IN his second lecture Dr. Feiling dealt particularly with alterations in muscle tone and changes in reflexes. Alterations in muscle tone were one of the most common and most important symptoms of disease of the central nervous system; therefore the recognition and interpretation of the more characteristic of these changes was very necessary. He did not propose to discuss the exact mechanism by which these changes were produced; he restricted himself to emphasizing the clinical value of an appreciation of the different changes of tone.

Hypertonus or Increased Tone.

With regard to hypertonus, the terms "spasticity" and "rigidity" were applied more or less indiscriminately to states in which the musculature of the limbs exhibited an increased tone. The textbooks stated that it was lesions of the upper motor neuron which caused such spasticity. This was true, but was far from being the whole truth. The upper motor neuron was a very complex mechanism, by means of which many different systems of fibres acted on the lower motor neuron. It was possible to distinguish broadly between the hypertonus of pyramidal lesions—that is, lesions interrupting the cortico-spinal tract—and that of extrapyramidal lesions, not merely by any differences in the reflexes, but by the intrinsic characters of the hypertonus itself. Thus in hemiplegia due to a vascular lesion of the internal capsule or its neighbourhood, after the preliminary stage of flaccid palsy, if the limbs became spastic, the characters of this spasticity exemplified the particular changes in muscle tone which occurred in lesions of the pyramidal tracts. There was a marked difference between the arm and the leg. The leg was stiff in a position of extension, but in the arm it was the flexors which showed increased tone, the characteristic attitude of the arm being adduction at the shoulder and flexion at the elbow and wrist joints. Cases due to cortical or sub-cortical lesions did not usually show so great an increase of tone. In the arm the hypertonic muscles would yield to firm passive extension at the elbow-joint. They often gave way somewhat suddenly, so that, unless contractures had occurred, the arm could be almost fully extended. If the arm were then flexed again at the elbow, and the patient told to extend it himself, there might be extension to some point, and then the flexor muscles became tonically contracted and so prevented further extension.

But in disease of the extrapyramidal motor system the position was different. Paralysis agitans, for example, began in many cases with a hemiplegic distribution of symptoms, and might remain in this state for a considerable time, the onset of the tremor being delayed perhaps for many months.

Abdominal and plantar reflexes, especially in old people, were not always easy to elicit. The character of the hypertonus was different here from that in hemiplegia due to lesions of the pyramidal tract. Instead of finding hypertonus in the flexors of the arm and extensors of the leg, there was a diffused rigidity affecting all muscles more or less equally, and not altered by voluntary or passive movements of the limb. This was often found in cases of encephalitis lethargica, apart from the cases which exhibited the Parkinsonian syndrome.

Wilson, in his original description of progressive lenticular degeneration in 1912, clearly distinguished between the universal diffused stiffness of muscles in that condition and that found in the ordinary cases of hemiplegia. In spastic paraplegia due to disease of the spinal cord a careful study of the nature and distribution of the rigidity present could give a valuable insight into the degree of damage to the cord. These differences in the character of the hypertonus has been emphasized by the study of cases of gunshot injuries during the war, and notably by the work of Head and others. Lesions of the cord which, on the motor side, interfered with the function of the pyramidal tracts only, would cause paraplegia in extension. More severe lesions produced paraplegia in flexion. In some cases this study might also help in localizing the anatomical site of the lesion, and aid in determining the nature of the pathological process, for the more peripherally placed lesions would involve more the pyramidal tracts, with the production of paraplegia in extension; and the more centrally placed lesions would tend to involve other tracts as well, with the production of paraplegia in flexion. This was of real value in the matter of prognosis.

Decerebrate Rigidity.

Dealing next with decerebrate rigidity, the lecturer said that numerous observers had recently claimed to have found in certain disorders of the central nervous system a clinical picture under this name. Wilson had reported cases in which, with impairment of consciousness, there was tonic rigidity of trunk and limbs, with exacerbations in the form of tonic or postural fits. The typical posture was with arms rigid, adducted at the shoulder, strongly extended at the elbow, with internal rotation and marked pronation of the wrists. In Wilson's cases the pathological conditions found were very various, including cerebral tumour, cerebral haemorrhage, and tuberculous meningitis. Recent papers, however, did not fully agree with Wilson's views, and it was pointed out that only in the matter of posture did the picture resemble that of decerebrate rigidity, and that there was lacking the other characteristics, particularly those described by Sherrington. As to whether conclusions of clinical value could be drawn from these cases, the lecturer said that this phenomenon represented disturbance of function only; it did not necessarily indicate permanent structural disease. He quoted cases in which, although the posture was almost exactly similar, the pathological cause varied greatly. One was that of a child aged 2½ years, who was admitted on account of optic neuritis, and during the two months of his illness lay unconscious, flat on his back, with the extensor muscles of the neck and trunk stiff, the eyes being in a position of spasmodic convergence. There was the typical meningeal cry. The positions of the limbs were those already described. After death there was found an infiltrating tumour of the brain, of sarcomatous type, occupying the whole mid-brain, and extending into the crura on each side. The picture, indeed, gave no indication of the pathological process at work; it was merely a sign of disordered function: a lesion had upset the normal balance between the many different systems of fibres which acted on the motor neuron, allowing some particular system to exert an abnormal effect.

Ramsey Hunt had recently elaborated a theory of the dual nature of the motor system. This supposed the existence of two systems: (1) kinetic, (2) static—the first associated with movement, the second with the maintenance of posture. Movements and postures were regarded by Hunt as of three varieties—reflex, automatic associating, and isolated synergic. Also he postulated three divisions of the nervous system: segmental, paleo-encephalon, and neo-encephalon, each of these containing the two different motor systems—that is, kinetic and static. There was not yet available sufficient accurate knowledge of the functions of the extrapyramidal system alone to warrant the acceptance of this hypothesis in its entirety.

Hypotonia was present in some cases, such as severe instances of acute anterior poliomyelitis. Lesser degrees of