lentils, peas, rice, oatmeal, bananas, skimmed milk, coffee, tea, and cocoa; fluids 1,200 to 1,500 c.cm. (1½ to 2 pints); salt to taste. Starchy foods are limited in order to promote the maximum assimilation of proteins and to lessen the retention of water. Fat is limited to lessen the amount of lipoids.

We have found this diet to be most efficacious in reducing nephritic oedema, and, further than this, it has resulted in a very marked improvement in the patient's general condition and in reducing the amount of albumin in the urine.

A FAMILIAL FORM OF ACOUSTIC TUMOUR.

BY

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TUMOURS of the acoustic nerve or nerves have been reported in medical literature occasionally for many years. They have usually been included under the head of "tumours of the cerebello-pontine angle." It is largely due to the work of Cushing that the acoustic tumours proper have been clearly differentiated from other tumours in this region, and he has been the first to point out their characteristic course and symptomatology. He estimates acoustic tumours to form 6 per cent. of all intracranial growths, and over 20 per cent. of those of the posterior fossa of the skull. Histologically, the isolated tumours of the acoustic nerve arise from the endoneurium; their structure, according to Cushing, consists of two kinds of tissue—a dense fibrous tissue and a looser areolar tissue, possessing some of the characters of a glioma. Bilateral tumours, however, are commonly part of a general tumour formation, which may affect many nerves—in fact, a general neurofibromatosis. Such cases have been not infrequently reported. It should be noted, however, that bilateral deafness has been found to occur in cases where only one acoustic nerve was involved. Henneberg and Kochl¹ were the first to point out that tumours of the acoustic nerve could form part of generalized neurofibromatosis. Oppenheim² remarks that he has seen eight cases where a general neurofibromatosis was masked by the symptoms of intracranial tumour.

A familial or inherited tendency has been noted in only a small proportion of the cases of neurofibromatosis. Thus Alexis Thomson³ was able to collect but ten examples from the literature. As regards acoustic tumours proper, the available literature does not record any familial or hereditary tendency. Hence the history of the family recorded in this article is of unusual interest. None of the cases have been verified as yet by operation or autopsy, but the clinical histories and the condition of those patients who were under careful observation are so striking and characteristic, and the existence in one of them of multiple tumour formation renders the diagnosis of acoustic tumours practically certain. The chronological order of symptoms, as described by Cushing—namely, auditory and labyrinthine disturbances, headache, cerebellar ataxia, disturbance of the function of other cranial nerves and the increase in intracranial tension, with optic neuritis and failure of vision—are well exemplified in the history of the family, which is as follows:

First Generation Available.

Mary Ann B., aged 80, had three brothers and sisters, all healthy. One is still alive; the others died at a ripe age.

John B., aged 80, is deaf and too senile to answer questions intelligibly. He was one of a family of ten, of whom only one sister survives. Several died young, but no details have been obtained except concerning Susan, who died, aged 30, of an illness lasting a few years, accompanied by headache and deafness, and Sarah, who died, over 60, of "something the matter with her head."

Second Generation.

Mary and John B. had ten children, of whom one died in infancy; four others are alive and well. But one, Richard B., has been a patient at the Hospital for Epilepsy and Paralysis, Maida Vale (see below). He is now in a Poor Law infirmary.

bedridden, totally blind and deaf. Another, John B., died at the age of 30 after an illness of over two years' duration. He complained first of deafness and noises in the head and was noticed to stagger in walking. Some five months after the onset of his illness he was sent to the Exeter Hospital, where he remained for a month or two. On his return his sight was failing, and he eventually became quite deaf and blind, although for some time he could walk with support. No history of vomiting or headache was obtained, but his friends and relations had noticed a swelling in the neck about the size of a hen's egg for many years. He died about two years after leaving the hospital.

Elisabeth B. died at the age of 33 after an illness of two years' duration. The chief symptoms noticed by the relations were unsteadiness of gait, deafness, and progressive blindness. She was for a time an inmate of the Exeter Hospital and was considered to be suffering from a cerebral tumour. Operation was suggested but refused.

Susan B. died aged 29. Her illness began in somewhat the same way, but she died suddenly from a "stroke."

George B. died aged 52 after an illness of some years' duration, which was accompanied by deafness, blindness, and an unsteady gait.

Third Generation.

Richard 2nd, son of John 2nd, has lately been a patient at the Hospital for Epilepsy and Paralysis, Maida Vale (see below). He has multiple neurofibromata.

William, his brother, states that at one time he had some lumps about his body, but that they faded away. He looks upon himself as foredoomed to die in the same way as others of his family. At present he has no symptoms.

The following is a more detailed account of two members of the family who were recently under the care of one of us at the Hospital for Epilepsy and Paralysis, Maida Vale.

CASE I.

Richard 1st, aged 41, was admitted on July 1st, 1919, under the care of A. F., complaining of deafness, headache, and inability to walk properly. He was then so deaf that it was impossible to obtain from him any accurate history, but one of us (E. W.) had previously seen him, and was able to supply the following history. He first came under observation with suspected pulmonary tuberculosis in October, 1913, but made a good recovery, and by October, 1917, the disease had evidently become arrested. In October, 1918, he complained of headache and weakness of the legs. From January to February, 1919, he was in a sanatorium, but was sent home as his symptoms were entirely nervous in origin. In April, 1919, he was found to be suffering from headache, chiefly occipital in site, and to be getting deaf. His gait was then slightly unsteady, with a tendency to fall to the right. By May, 1919, he had become very deaf, the headache was worse, and he had begun to vomit occasionally. Vision had become defective. The gait was markedly ataxic. The pupils were unequal, the left being larger than the right, and there was well marked double optic neuritis. Nystagmus was present. On admission to hospital in July, 1919, the condition was as follows:

He suffered from a severe and continuous headache, chiefly localized to the occipital region on the left side. There was complete bilateral deafness. The pupils were equal in size and reacted normally. Examination of the fundi showed well-marked double optic neuritis, with much swelling of both discs. Nystagmus was present in all directions, but was more marked when the eyes were directed to the left. With the exception of slightly diminished sensation in the area of the first division of the fifth nerve on the left side, the functions of the other cranial nerves appeared normal. There was no paresis of either the arms or the legs. No static tremors were present. Some slight ataxia was observed in the upper limbs on voluntary movement. The gait was decidedly ataxic, with a tendency to fall to the left. The tendon reflexes were active, the abdominal reflexes present, and the plantar responses were of the flexor type. The Wassermann reaction in the blood was negative.

A diagnosis of bilateral tumours of the cerebello-pontine angle, probably of the acoustic nerves, was made. Operation was advised but refused, and the patient left the hospital. At the present time he is an inmate of a Poor Law infirmary, where he is quite bedridden, totally blind and deaf.

CASE II.

Richard B. (son of John 2nd), aged 20, was admitted to the Hospital for Epilepsy and Paralysis, Maida Vale, on January 2nd, 1920, under the care of one of us (A. F.). As he was almost completely deaf in both ears and could neither read nor write, it was impossible to obtain an accurate history of the condition. But it appeared that deafness had been noticed to begin about nine months before and to progress rapidly. He also complained of constant roaring noises in the head. There was no head-

ache and no vomiting, and no complaint of difficulty in walking nor of giddiness.

On examination the pupils were found to be unequal in size, the right being larger than the left; the right pupil reacted briskly to light and accommodation; the left pupil was contracted, slightly irregular in shape, and reacted only very sluggishly to light. There was no nystagmus and no ocular paralysis. The fundi were normal. With the exception of the deafness the functions of the other cranial nerves were not disturbed in any way. There was practically complete bilateral nerve deafness. He was examined by Mr. Muecke, who considered that the nerve conduction from the labyrinth was almost completely blocked on both sides, more so on the left.

There were no other signs of disease in the central nervous system. But there were present three subcutaneous tumours whose characters suggested that they were fibromata or neurofibromata. These were exceedingly interesting and important in view of the condition of the auditory apparatus and the family history. In the right side of the neck, underneath the sterno-mastoid muscle, was a large, rather spindle-shaped tumour, about the size of an elongated lemon, extending from the angle of the jaw nearly to the sterno-clavicular joint. It was hard, freely mobile, and quite painless. There was no evidence of surrounding inflammation and no enlargement of lymphatic glands. A second but smaller tumour was found in the subcutaneous tissues of the chest wall, just over the right costal margin in the anterior axillary line. It was about the size of a walnut, hard, irregular in shape, freely movable, and painless. A third tumour, about the size of a small date, with similar characteristics, was situated in the upper part of the left gluteal region. The patient stated that he had had these tumours as long as he could remember. The smaller one, in the chest wall, was removed under local anaesthesia for histological examination. Dr. Canti reported that it showed the structure of a soft fibroma. It is more than likely, however, that it arose from a nerve sheath, since it is not easy to demonstrate the existence of nerve tissue in these neurofibromata which have been present for a long time. Operation on the skull was not advised.

REFERENCES.

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²Oppenheim, *Textbook of Nervous Diseases*, vol. ii. ³Alexis Thomson, *On Neuroma and Neurofibromatosis*, Edinburgh, 1900. ⁴Cushing, *Tumours of the Nervus Acusticus*.

THE OPERATIVE TREATMENT OF ULCERATIVE COLITIS.

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THE discussion on ulcerative colitis which took place at the Royal Society of Medicine in 1909 first revealed the importance of the disease both as regards its prevalence and commonly fatal consequences. Since then our knowledge of it has advanced considerably, but chiefly in respect of the epidemic forms rather than the sporadic chronic cases which are more common in this country.

Very little advance seems to have been made in the diagnosis and treatment of the truly chronic ulcerative forms. There are many reasons for this, one being that the chronic cases have proved almost a complete failure from the point of view of bacteriological investigation. In a large number of cases the stools have been investigated by most competent bacteriologists, but so many different micro-organisms are found, and the difficulties of deciding which is responsible for the disease are so great, that no useful information is, as a rule, obtained. My own experience has been that, with one or two conspicuous exceptions, bacteriology has been of little assistance.

Medical treatment also has not been very successful except in the mild cases, though here I must admit I may be prejudiced by the fact that as a surgeon I am more likely to see the cases in which medical treatment has failed.

Some of the worst cases of ulcerative colitis are those in which the chronic condition is secondary to an acute epidemic form such as amoebic or bacillary dysentery. There is a certain similarity between these cases and the

ordinary form of chronic phthisis. In both we find an acute specific infection causing lesions which become secondarily infected with other septic organisms, and it is this latter secondary infection which causes much of the trouble.

These cases of chronic ulcerative colitis are particularly important at the present time, because very large numbers of men have been infected with acute dysentery while fighting at different parts of the front during the last five years, and are now suffering from the chronic and often much more serious condition.

Not only have a large number of these cases returned to this country during the last eighteen months, but quite a considerable number of cases have occurred among men who have never left England, and among women. These latter cases do not start with acute amoebic dysentery, but are chronic from the beginning. I have little doubt, however, that they have in many cases become infected from the class of cases first mentioned. This is rather borne out by the fact that several cases of ulcerative colitis which came under my observation last year came from one seaport town where many troops from abroad were landed.

A typical case would be that of a man who contracted acute dysentery in Africa or Arabia during the war and had been more or less successfully treated at the time, but the diarrhoea has recurred subsequently until it has become almost continuous, and he has gradually begun to go downhill with constant liquid blood-stained stools, progressive loss of weight, failure of appetite, etc.

An examination with the sigmoidoscope (the value of which in these cases as a means of diagnosis does not even yet appear to be fully appreciated) shows deep and extensive ulceration of the mucous membrane of the rectum and colon. In a bad case islands of mucous membrane can be seen standing up from the ulcerated surface, presenting an appearance closely resembling a polypoid condition.

Examination of the stools show numerous bacteria, mostly of the *coli* or streptococci groups, but no sign of protozoa.

The clinical history of cases of ulcerative colitis varies considerably. In those cases in which the disease is not secondary to acute dysentery the condition often starts insidiously. The patient appears to recover satisfactorily from the initial attack, but recurrences become more and more frequent and less amenable to treatment, until the condition becomes so serious that no treatment seems to have any effect. It is generally at this stage that the surgeon is called in.

There may sometimes be confusion between chronic ulcerative colitis and other forms of chronic recurring diarrhoea, but this can at once be cleared up by an examination with the sigmoidoscope. In true ulcerative colitis the temperature is almost always raised 1° to 2° F. at night. Blood is more or less constantly present in the stools, and wasting is a marked feature of the case.

The best treatment for ulcerative colitis is appendicostomy, and I have seen very many really desperate cases recover as a result of this operation, which I and those associated with me were certain must otherwise have died. Unfortunately the surgeon is too often only called in when the patient is already very seriously ill and the ulceration has become very extensive. The operation should be performed at a much earlier stage of the disease, and recovery would be then much more rapid and more certain.

In true ulcerative colitis non-operative treatment appears to be of very little use. Even when recovery does occur it is a slow and tedious process. One of the reasons for this is that the ulcerated surface is very large and cannot be directly dealt with. Douches and enemas cannot be tolerated in sufficient quantities to ensure that they reach the whole of the diseased area.

Appendicostomy acts in two ways. The first and most important action is to allow of the large ulcerated area being kept clean and free from pus and accumulated discharge. The frequent washing of the colon by removing the products of inflammation allows the ulcers a chance to heal, and to a large extent prevents the absorption of septic by-products into the blood.

The second important action is to compensate for the great loss of body fluid, which is always present owing to the constant diarrhoea. Many of these patients are wasted till they are little more than skin and bone, and it is often quite remarkable to see the improvement that follows