The first point which arises is, that no conclusion should be drawn as to the sensitiveness of an organ which has been stimulated through a structure itself sensitive. Nearly all conclusions have been drawn from observations made by pressing on the organs through the sensitive abdominal wall. Many observers state that they have demonstrated the sensitiveness of an organ by first observing its position by various methods, such as the *x* rays, and later by pressing over it and eliciting pain. The error here lies in the failure of the observer to take the precaution of excluding the sensitive external body wall. If, for instance, when such sensitiveness has been detected, an attempt is made to delimit the area that is sensitive, it will be found that the size and shape of the area painful on pressure bears no relation to the size and shape of the organ supposed to be tender. I discussed this matter with a very skilled physician. He demurred to my statement that there was no evidence of the sensitiveness of organs. He declared that he had a patient with a large liver which

FIG. 5.—The shaded area represents the liver, the dotted area the extension of hyperalgesia beyond the liver margin, showing the pain elicited by pressure over the liver was due to an increased sensitiveness of the tissues of the external body wall.

he could demonstrate to me was extremely tender. I asked to see this patient and was shown him. The enlarged liver was easily palpated, the lower margin being sharp and well defined. On gently pressing over the liver the patient winced from the pain. I asked the physician to map out the region that was so manifestly tender, and to his surprise he found the tenderness extended 3 in. lower than the margin of the liver. (Fig. 5.)

It is well known that pain does occur in disease of the viscera, and in animals distress has been provoked by certain experiments. But here the experimenter never realized that the evidences of suffering, even if these were due to pain, gave no information as to the locality of the pain—a fact absolutely essential to the understanding of the

mechanism by which visceral pain is produced. The same neglect of this essential matter is found in the observations of surgeons and others who have studied pain in the exposed organs of the human subject.

When, in testing the sensibility of organs, pain is elicited, a clear conception of the nature of the stimulus giving rise to it should always be obtained. The need for this and for the recognition of the locality in which a pain is felt is shown by the following experience. In resecting the bowel in a patient who was not under the influence of an anaesthetic, I tested the sensitivity of various organs and their coverings. On pinching the bowel the patient, after a slight delay, complained of pain. I asked him where he felt the pain, and he pointed to the region of the umbilicus. The portion of bowel stimulated lay some distance off on the left side, and it was manifest that the place to which the pain was referred had no direct relation to the part stimulated. Had I been content with simply noting that pinching of the bowel produced pain, I would have overlooked this very important matter of localization.

But there was more in the observation. There was a slight delay in the pain which came in wave-like intensity, and I observed that the pinching had set up a strong peristalsis of the bowel, and that the pain only occurred when this peristalsis was set up. So here I had evidence that the mechanical stimulus I applied only produced pain when a vital process—that is, peristalsis—was set in motion in the bowel.

There are other matters which have been overlooked in this investigation, but what I have said will suffice to show the need for a thorough undertaking of methods before we can observe accurately. Although I do not claim that I have solved the problem of visceral pain, yet the pursuit of this inquiry for many years has given me a certain amount of skill, which has enabled me to discover many facts from which I have been able to draw certain reasonable deductions. These have proved of considerable value in practice.

(To be continued.)

SOME POINTS IN THE PROGNOSIS AND TREATMENT OF CHRONIC NEPHRITIS.*

BY

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THE management of cases of chronic Bright's disease in private practice is, under the methods generally employed, most tedious and uninteresting. It is the purpose of the present lecture to point out some of the newer methods of examination which not only lend a fresh interest to the cases, but which enable us to adopt a more scientific line of treatment and permit of a more accurate prognosis.

Prognosis.

In order that one may make a well considered prognosis it is necessary first to adopt some practical form of classification, and the classification which I suggest to you is that seen in the accompanying table:

Classification of Chronic Nephritis.

TYPE.	Urine.				Oedema.	Uraemia.	Eye Changes.	Blood Pressure.	Prognosis.	Renal Efficiency.
	Sp. gr.	Quantity.	Albumin.	Casts.						
Degenerative (cloudy swelling)	+	-	-	-	-	-	-	-	Good	
Tubular nephritis (large white)	+	-	+	++	++	-	-	-	Fair	Fair
Glomerular ...	-	-	+	+	-	+	+	+	Bad	Bad
Secondary sclerotic (chronic interstitial)	-	+	-	-	-	++	+	+	Bad	Bad
Arterio-sclerotic ...	+	+	-	-	-	-	-	++	Good	Good

There are, of course, cases of chronic nephritis which cannot be fitted into any one of these categories—cases of mixed nephritis—but for practical working purposes you will find that the classification is one of great value, and that the time spent in obtaining the information under the various headings has not been wasted.

This classification splits up the old group of "chronic diffuse nephritis" into two: (a) tubular, (b) glomerular; and of these the latter is by far the more serious. It also draws a sharp distinction between the secondary sclerotic (contracted granular) kidney and the arterio-sclerotic.

The most important distinction between these varieties of chronic nephritis is the degree of "renal efficiency," by which I mean the measure of the power of the kidney to excrete water, salts, nitrogenous and other waste products.

For gauging renal efficiency most of the tests are such as can be employed in general practice, the only apparatus required being a large vessel graduated in cubic centimetres or ounces, a tall narrow cylindrical vessel, and a small specific gravity bulb. The materials for carrying out the other tests are always to be obtained at the clinical laboratory to which the specimens of urine are to be subsequently sent.

Tests of Renal Efficiency.

1. *Water Excretion Test.*—Empty the bladder. Two and a half hours after breakfast give 500 c.cm. (17½ oz.) of water, and collect all the urine that can be passed in the next two hours. A healthy person passes about 400 c.cm.

2. *Specific Gravity Test.*—The urine is to be passed every two hours from 8 a.m. to 8 p.m. Each quantity is measured, and

Chart 1.

Chart 2.

CHART 1.—Two-hourly specific gravity curve of urine. Severe case of glomerular nephritis.

CHART 2.—Two-hourly specific gravity curve of urine. Case of glomerular nephritis, moderate severity.

the specific gravity taken. The urine passed between 8 p.m. and 8 a.m. is also measured, and the specific gravity taken. In health the quantity of day urine should be greater than the quantity of night urine; the specific gravity of the various day specimens should show wide variations, and at least once in the

* Abstract of a post-graduate lecture delivered at the Bristol General Hospital, July 17th, 1919.

day should reach 1020. Fixation of specific gravity, and especially a persistent low specific gravity, shows renal inefficiency. (See Charts 1, 2, 3.)

CHART 3.—Specific gravity curve of urine passed two-hourly. Case of glomerular nephritis, convalescent.

3. *Test of the Rate of Secretion.*—Inject 6 mg. of phenolsulphonaphthalein into the buttock muscles, and give the patient a drink of water. At the end of one hour and ten minutes get the patient to pass urine, and again at the end of a second hour. Send the two specimens of urine to the clinical laboratory, where the percentage of dye is estimated by the colorimeter. A healthy kidney excretes about 60 per cent. of the dye in the two hours and ten minutes. Small capsules of phenolsulphonaphthalein containing 6 mg. in solution can be obtained at any of the large chemists. The ordinary hypodermic syringe can be used.

4. *Sodium Chloride Excretion Test.*—Send to the clinical laboratory a sample of the mixed twenty-four hour urine for the estimation of sodium chloride. If the amount of sodium chloride excreted falls to one or two grams per diem (average in health 15 to 20 grams) the prognosis is grave.

5. *Estimation of Non-protein Nitrogen in the Blood.*—Draw off 10 c.cm. of blood from a vein in the arm and send this to the clinical laboratory for the estimation of the non-protein nitrogen (urea, uric acid, and creatinin). If this amounts to more than 0.4 gram per litre there is a dangerous accumulation in the blood, possibly due to alteration of kidney efficiency. The blood (together with 1 c.cm. of a solution of potassium oxalate to prevent clotting) is to be drawn off in a sterilized syringe provided for the purpose by the laboratory.

No one of these tests is by itself of great value, but the combined tests, together with the data mentioned in the table (albumin, casts, blood pressure, etc.) will enable us to form a fairly accurate estimate of not only the efficiency of the kidney in any given case of nephritis, but also of the general condition and progress of the patient.

The tests should be repeated at least every six or twelve months and any variation noted. During the whole period of examination the patient is kept on the standard renal diet, to which I shall refer later.

Tests of renal insufficiency are superior to the old tests for albumin, casts, etc., inasmuch as they enable us to detect disease of the kidney in a much earlier stage. In the future we may look forward to detecting the pre-albuminuric stage of nephritis. At present we seldom detect the chronic case until two-thirds of the kidney substance is destroyed. Such tests are also of value in distinguishing between the coma of nephritis (uraemia) and the coma of arterio-sclerosis (cerebral haemorrhage or thrombosis).

Space does not permit me to touch on other important points of prognosis, but I would point out that the outlook in cases with simple chloride retention is good, even if uraemic symptoms be present, whilst that of cases with nitrogen retention with uraemia is much more grave. In secondary sclerotic nephritis, if the patient escapes the dangers of uraemia, he is, if a man below 50 years of age, most likely to succumb to cardiac failure, whilst at any later period cerebral haemorrhage is most to be feared.

Treatment.

The greatest advance in recent years has been the recognition of the fact that many cases of chronic Bright's disease are originated and kept up by cryptic infections such as pyorrhoea, chronic nasal suppuration, diseased tonsils, and chronic bowel infections.

We are, of course, familiar with the septic origin of acute nephritis. We have had one such case recently in this hospital.

A soldier had suffered for four months from a shattered elbow with profuse suppuration. During this time he developed nephritis. On February 1st, 1919, Mr. Hey Groves amputated the arm through the lower third of the humerus; severe secondary haemorrhage a week later necessitated blood transfusion, but by February 14th the albumin which had been two parts per thousand was reduced to a mere trace, and by February 24th the urine was normal.

Similarly in chronic nephritis the removal of a septic focus in the teeth, nose, or ear, may lead to immediate

amelioration of symptoms, and ultimately to complete recovery. In this way chronic nephritis is comparable to rheumatoid arthritis and it is worthy of note that a very large percentage of the cases of rheumatoid arthritis ultimately die of chronic nephritis.

It is not my intention to touch upon the subject of drug treatment in chronic nephritis, this being of far less importance than the arrangement of the dietary and habits of living. If you follow out the tests I have indicated, then the main principles of dietetic treatment are plainly before you.

1. Where renal efficiency is low and nitrogen is retained you must limit the intake of nitrogenous food, and allow plenty of fluid.

2. When there is general oedema and the chloride elimination is low, you must cut off the supply of salt and diminish the allowance of water.

Dealing first with the cases of nitrogen retention, we find these are chiefly patients who, though suffering from chronic interstitial nephritis, are yet in moderate health and capable of conducting their business. To such we allow 2,500 to 3,000 c.cm. (4 or 5 pints) of fluid a day and limit the nitrogenous intake to about 50 grams a day. These requirements are met by a diet such as the following:

Breakfast.—Porridge prepared from 2 oz. of dry oatmeal, bacon, 3 oz. of toast, tea.

Lunch.—Milk pudding or vegetables, bread-and-butter 3 oz., jam, fruit, and sugar.

Tea.—Tea, 2 oz. bread-and-butter.

Dinner.—Vegetable soup made with milk, 3 oz. of fish, flesh, or fowl, 3 oz. potato, cooked or fresh fruit, sugar.

Sugar is given plentifully to raise the calorie value of the diet and to spare the nitrogen katabolism. Peas, beans, asparagus, onions, rhubarb, strawberries, condiments, meat extracts, alcohol, and tobacco are excluded, either as being too rich in nitrogen or as being renal irritants or stimulants.

This, which I call a standard nephritic diet, is suitable for use when the urinary tests are being made. From time to time the dietary may be varied; thus the patient may be ordered to take only a Continental breakfast of rolls and coffee, or may be directed to go for two days in the week without meat. No distinction is made between fish, fowl, and red meats.

In cases of chronic parenchymatous nephritis, with retention of chlorides and a surplus of water in the tissues, the indications are to cut off altogether the intake of salt and to reduce the consumption of water to about 1,000 c.cm. (2 pints) daily. It has been claimed that the oedema of chronic parenchymatous nephritis may be reduced entirely by adherence to a salt-free diet, with limitation of fluid intake. This is sometimes known as the *Karrell treatment*. Karrell's dietary was as follows:

One and a half pints of skim milk daily (7 oz. every four hours), 6 oz. of coffee. Unless there be uraemia or exhaustion, keep this up for one week, then add stewed fruit, bananas, rice, arrowroot, cereals and cream, toast, vegetables, and sugar. Later give eggs and meat. Use no salt for cooking or in eating. Limit liquids to one and a half pints a day.

The reduction of oedema by the adoption of a salt-free diet has not been very successful. When an oedematous case of large white kidney is first put to bed an increased flow of urine is the immediate result of the rest given to the heart, but in nine cases out of ten the oedema persists, however strictly the diet be enforced.

The reduction of the oedema by the use of diuretics is equally disappointing. Indeed, in cases of pure nephritic origin the use of diuretics is contraindicated as tending to lower renal function. Alkaline salines are least open to this objection and may be tried. On the other hand, such oedema as may be due to circulatory failure is readily removed by diuretics of the digitalis and caffeine groups.

Successful cure of the oedema of chronic parenchymatous nephritis is much more likely to follow the procedure suggested by Epstein. He pointed out that the steady loss of large quantities of albumin in this variety of nephritis caused a decrease in the osmotic pressure of the blood, which fact favours the absorption and retention of fluid by the tissues. Hence the great oedema. The blood in such cases shows a marked decrease in protein and an increase in fatty bodies (lipoids). The indications for treatment, therefore, are first to increase the protein content of the blood and thus restore its osmotic power, and, secondly, to remove the excessive lipoids. To effect these ends he administers a diet rich in protein and poor in fat: Fish, lean meat, lean ham, white of egg, oysters,

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-