some importance from a medico-legal point of view. It also suggests that the immediate extraction of a foreign body from the heart is not always justifiable unless symptoms are urgent.

SUMMARY OF CLINICAL FINDINGS.

The electro-cardiograms show that the spread of the excitation wave through the heart muscle is not significantly altered in its course or time relations in these cases. It is well known that after infarction of the heart muscle certain changes may occur in the electro-cardiogram particularly affecting the form of the T waves. These alterations are more frequently observed when the lower part and apex of the left ventricle are involved in the infarcted area. The T deflection is said¹⁴ to show a certain sequence of changes in shape and direction, gradually returning to normal as healing in the muscle takes place. It is possible that shortly after the injury sustained by the heart in the above cases, some deformity of the T wave might have been observed. After a lapse of years no abnormalities are to be detected in the T waves of these patients, even in the presence of inert metal buried not far from the apex. Apparently but few cases with retained missiles have presented themselves for electro-cardiographic examination. Lea¹⁵ has recorded one instance in a young soldier with a fragment of shell embedded near the apex of the heart, in whom complete heart-block was present. In the single electro-cardiogram published the T waves were upright and of normal form. In the case of Jones¹⁶ the T waves were inverted in lead 2 thirteen weeks after the injury. The left ventricular preponderance observed in Case ii of this series is not considered of much significance. The heart was not found enlarged or displaced by clinical or x-ray examination. Pericardial adhesions over the posterior surface of the heart might possibly account for this change.

The vital capacity test indicates that these men suffered from no diminution in ability to take a full deep breath. The test has been used to some extent as a measure of physical fitness, and has proved of value in following the course of heart disease. The fact that the result obtained in Case i was a little lower than might be expected is probably attributable to his voluntary abstinence from most forms of physical exercise. The man was flabby and in poor physical training. In Case ii the high reading recorded indicates that, so far as this test is concerned, there was no impairment of his respiratory powers.

In Case iii it is assumed that, after obtaining a heart wound, this man was sufficiently fit to rejoin a front line regiment. Any cardiac disability under such circumstances is extremely unlikely. The man survived a heart wound, and must have led a vigorous life thereafter, ultimately to succumb to the effects of a compound fracture of the femur.

CONCLUSIONS.

1. Foreign bodies such as bullets, shrapnel, or fragments of shells may remain buried in the myocardium for years without the production of symptoms.
2. Two cases are described in which missiles were found embedded in the muscle of the left ventricle of the heart by x-ray examination thirteen years after wounding.
3. Both men are in good health and fit for active work at the present time, the foreign bodies being *in situ*.
4. The electro-cardiograms presented no significant alterations in the form of the T waves or other deflections.
5. In the third case, at *post-mortem* examination, a fragment of shell was found buried in the left ventricle. It was not related to the death of the patient.

My thanks are due to Professor D. Murray Lyon, who kindly placed Cases ii and iii at my disposal. The x-ray photographs were taken in the radiological department of the Royal Infirmary, and I am indebted to Dr. Woodburn Morison for the reduced prints. Case i was kindly sent to me for examination by Dr. S. Lipetz, to whom I am indebted.

REFERENCES.

- 1 Beck, C. S. : Wounds of the Heart—the Technique of Suture. *Arch. of Surg.*, xiii, 1926, 205.
- 2 Davenport, G. L. : Suture of a Wound of the Heart. *Journ. Amer. Med. Assoc.*, lxxvii, 1924, 1840.
- 3 Ballance, Sir C. A. : *The Surgery of the Heart* (Bradshaw Lecture). London, 1920.
- 4 Cutler, E. C., Levine, S. A., and Beck, C. S. : Surgical Treatment of Mitral Stenosis. *Arch. of Surg.*, ix, 1924, 689.
- 5 Weber, J. C. : Quoted by Fischer, G. (*infra*).
- 6 Grainger, Mr. : An Account of a Ball found in the Heart of a Buck. *Edin. Med. Journ.*, xii, 1816, 498.
- 7 Croly, H. G. : Case of a Musket Bullet found in the Pericardium. *Dublin Med. Press*, li, 1864, 630.
- 8 Delorme, E. : *Traité de Chirurgie de Guerre*, vol. 2, p. 731. Paris, 1893.
- 9 Vaquez, H., and Bordet, E. : *The Heart and Aorta* (English edition), p. 236. New Haven, 1920.
- 10 Vance, B. M. : Three Unusual Cases of Bullet Wounds of the Heart showing Attempts at Healing. *Journ. Amer. Med. Assoc.*, clxix, 1925, 872.
- 11 Fischer, G. : Die Wunden des Herzens und des Hertzbeutels. *Arch. f. Klin. Chir.*, ix, 1863, 571.
- 12 *Official History of the Great War—Medical Services*, vol. i, p. 442. London, H.M. Stationery Office, 1922.
- 13 Bland-Sutton, Sir J. : Missiles as Emboli. *Lancet*, i, 1919, 773.
- 14 Parkinson, J., and Bedford, D. E. : Successive Changes in the Electro-cardiogram after Cardiac Infarction. *Heart*, xiv, 1928, 195.
- 15 Lea, C. E. : Shell Wound of the Heart Causing Complete Heart-block. *Lancet*, i, 1917, 493.
- 16 Jones, L. : Gunshot Wound of the Pericardium and Heart. *Brit. Journ. Surg.*, iv, 1916, 103.

THE TREATMENT OF NEPHRITIS.*

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THOUGH nothing as dramatic as insulin, or as specific as liver extract in pernicious anaemia, has yet been applied to the treatment of nephritis, great advances have been made within recent years; and there comes a time when, with the gradual accumulation of new facts and theories, it is useful to review our knowledge of a subject, and bring our plan of campaign as far as possible up to date.

The more important functions of the kidney are: (1) The regulation of salt and water metabolism and the osmotic pressure of the blood. (2) The excretion of certain end-products of metabolism, especially from protein, and of other substances not required by the body. (3) The maintenance (with the help of other apparatus) of the acid-base equilibrium of the body. The first of these functions is represented clinically in the amount and specific gravity of the urine, and in the occurrence of oedema. With regard to the second function we have in the estimation of the non-protein nitrogen of the blood a delicate and important test, and one which is often most helpful in treatment and prognosis. It must be remembered that the normal kidney can, and will, excrete protein when required to do so—as, for instance, injected proteins, or haemoglobin when liberated from its normal position in the red cell.

The following classification of nephritis, based on that of Christian,¹ will be adopted in this paper.

1. Acute nephritis (oedematous, haemorrhagic, recurrent).
2. Subacute nephritis with oedema.
3. Chronic nephritis (a) with oedema; (b) without oedema; (c) vascular hypertension progressing into nephritis.

ACUTE NEPHRITIS.

Nearly every case of acute nephritis, whether oedematous or haemorrhagic, is a sequel of an acute infection, most commonly a throat infection, which it usually follows after an interval of ten to fourteen days. Scarletinal nephritis, which in all cases is a sequel of a streptococcal tonsillitis, is no exception.

There are two factors at work in the production of acute nephritis: (1) the specific factor which causes the renal damage; (2) the factor of functional overstrain which determines the amount of the damage. Animal experiment² is quite clear on this point, for whatever the poison used—some of the recent work has been done with scarlet fever streptococcus endotoxin³—the amount of damage done by a given dose depends on the functional strain thrown on the kidney; in other words, on the amount of protein and salt in the diet. This applies not only to rabbits, whose normal protein intake is low, but also to animals normally carnivorous. A further point which stands out from the experimental work^{4,5} is that fresh vegetables in the diet are not merely neutral in effect, but appear to have a positive influence in limiting the degree of renal damage.

The treatment of acute nephritis must therefore consist in the prevention of functional overstrain during the stage

* Abridged from an address given before the Sheffield Medico-Chirurgical Society, January 24th, 1929.

of acute renal damage, and the elimination of any existing source of further infection when the acute stage is over. The handling of the acute stage is therefore largely dietetic. The fluid intake is restricted wherever there is oedema, for if the kidney is retaining water it is obviously useless to give more water and so increase the oedema. Flushing out the kidneys is not only unnecessary and undesirable, but also impossible. The same principle applies to the treatment of the transitory anuria, which sometimes lasts for a day or two in the initial phases. If, as Volhard⁶ suggests, this is due to a slowing down of the glomerular circulation brought about by congestion and oedema of the kidney, the condition is far more likely to be relieved by restriction of fluid, which will reduce the oedema, than by the administration of fluid, which will increase it. Cases of anuria in acute nephritis which I have treated by strict limitation of fluid intake have responded to their thirst by the recommencement of urinary secretion. For the rest, the diet should consist almost entirely of carbohydrate, with a minimum of salt and with a daily portion of fresh vegetable food. A limited allowance of milk is probably desirable, its small protein and salt content being compensated for by its value as a food in other ways. Milk as the sole or main article of diet in acute nephritis is, however, very far from ideal. These principles are represented in the diet sheet below:

Breakfast: Milk, cereals, tomatoes, bread or toast, marmalade.

Dinner: Potatoes, green vegetables, Yorkshire pudding, milk pudding.

Tea: Bread or toast with jam, plain fruit cake.

Supper: Biscuits or bread, salad.

No salt or butter. Total daily allowance of milk $\frac{3}{4}$ pint; other fluids $\frac{3}{4}$ pint (water and fruit juice).

One cannot pass over the acute stage of acute nephritis without considering the treatment of convulsions. These are practically indistinguishable from eclamptic fits, and are presumably of the same origin. True uraemia is exceedingly rare in acute nephritis, though the only certain distinguishing test is the blood non-protein nitrogen. Following Volhard,⁷ who believes that these convulsions are due to cerebral oedema, I have practised lumbar puncture in their treatment with success. Convulsions may add to the immediate gravity of the situation, but are usually relieved at once by lumbar puncture and have no effect whatever on the eventual prognosis.

By the end of a few weeks of dietetic treatment most cases will be free from oedema and haematuria, and there may or may not be some albumin remaining in the urine. If the patient is discharged at this stage he will either gradually recover entirely or develop subacute nephritis, or he will return very shortly with recurrent acute nephritis—a serious disease which in a certain number of cases again becomes subacute or chronic in type. These recurrent cases will, in my experience, all be found to have the same focus of sepsis which caused the first attack. The treatment of acute nephritis is therefore quite incomplete unless the septic foci have been thoroughly eradicated. To confirm this view I have been through the records of eighty-four cases of acute nephritis and have been able to follow up and re-examine fifty-four of them. The results are briefly given in the accompanying table.

Acute Nephritis.

	No. of Cases.	No. Cured.	Percentage Cured.	Error.
Group I. No septic focus found ...	28	21	75	±20
Group II. Septic focus still present	12	3	25	±25
Group III. Septic focus found and removed	14	14	100	—

In the first group I found no obvious sepsis present on re-examination, but my search for it consisted merely of inspection of the tonsils and an inquiry into history of colds, sore throats, etc., and several cases of septic foci are probably included in this group. In the rest of this group the septic factor causing the original attack of nephritis was probably of a temporary or transient nature; 75 per cent. of these cases were well, and by this I mean that they had had no further attacks of nephritis, and had on

re-examination no albuminuria or casts, no oedema, and no rise of blood pressure. Of the remainder, five have had between one and seven further attacks of nephritis, others have albuminuria or oedema still, with headache, malaise, and similar symptoms. In all cases it is at least a year since discharge, and although this is a short period, nephritis which is going to recur does so, in my experience, within six months (usually much less) of the initial attack.

The next group of cases all had obvious sepsis on re-examination, usually in the tonsils. They show a recovery rate of only 25 per cent. Of the rest, two are dead (one from subacute nephritis, the other from uraemia), the others show oedema, albuminuria, or both, or have had recurrent attacks.

Most of the patients in the third group were treated by tonsillectomy, and though the group includes four patients who had previously had two, three, or four attacks of nephritis, all are well to-day. It must be emphasized that but for this treatment these cases would have belonged to Group II, with the worst recovery rate of any.

The final column of "error" is based on the statistical formula $P \pm 3 (0.6745) \sqrt{(Pq/n)}$; where P = proportion of successes, q = proportion of failures, and n = number of cases.

SUBACUTE NEPHRITIS WITH OEDEMA.

This oedematous type, known to older authors as the large white kidney, and now sometimes called nephrosis, is typically unaccompanied by cardio-vascular or retinal changes, or by nitrogen retention, and in many cases there are no casts in the urine. It arises insidiously and has only three essential features—namely, oedema, albuminuria, and oliguria. Sometimes, however, such conditions as raised blood pressure and nitrogen retention occur later in the disease, or are present from the first, perhaps because the nephrotic syndrome has been secondary to a true nephritis.

Before dealing further with the disease it is necessary to consider a few theories of oedema. The capillary wall may be looked upon as a semi-permeable membrane separating the blood from the lymph. Salts—that is, crystalloids—pass through the membrane, but not colloids such as the plasma proteins. An increase of intracapillary pressure (such as occurs in heart disease) will cause more fluid to leave the vessel, and hence will produce oedema; a reduction of plasma protein, by reducing the osmotic pressure within the vessel, will act in the same way. Such a reduction in plasma protein occurs in nephrosis owing to the prolonged action of a pronounced albuminuria. The effect of loss of fluid into the tissues will be to cause the kidney to excrete less water. Hence oedema and oliguria may both be dependent on albuminuria; and, remembering that the normal kidney can excrete protein, we see that the disease may be due to an error of metabolism whereby serum albumin can no longer perform its proper functions in the body, and is therefore excreted, just as sugar is excreted by the normal kidney in diabetes. This, briefly, is Epstein's theory of nephrosis,⁸ and he has, in fact, lately named the disease "diabetes albuminuricus." Other interesting facts pointing to the general metabolic nature of the disease are the increase of lipid substances in the plasma and elsewhere and the extraordinary tolerance of nephrotic patients to doses of thyroxin which would be sufficient to poison a severe case of myxoedema. The changes in the kidney are of a degenerative rather than an inflammatory nature (hence the term "nephrosis"), and may be secondary to the prolonged albuminuria. This has, in fact, recently been confirmed by the observations of Jungman,¹⁰ who removed a portion of kidney from an early case and found it to be normal.

The present treatment, on which some of the cases, even after months of invalidism, may recover, apparently completely, is as follows:

1. The diet should be rich in protein and very low in fat.
2. Thyroxin should be given in large doses by the mouth, and, if necessary, intravenously. Epstein has given as much as 10 mg. intravenously.
3. Diuretics, such as ammonium chloride, calcium (possibly in conjunction with parathormone), and novarsuril,¹¹ have been used with some success.
4. Blood transfusion is rational, and useful as a temporary measure.

5. The removal or treatment of any obvious focus of sepsis is as important as in acute nephritis. I have seen one case in which the surgical drainage of an empyema was followed in six weeks by disappearance of albuminuria and oedema.

CHRONIC NEPHRITIS.

Chronic nephritis is a disease unlikely ever to become curable, though prevention may some day be within our power. The type with oedema is rare, and is a mixture of the previous and following types.

In the non-oedematous case there is nitrogen retention and high blood pressure, but no difficulty in water excretion. The indications in treatment are, therefore, restriction of activities and a low protein diet; but there is no need to reduce the fluid intake, and it is wrong to attempt to reduce the blood pressure by drugs, even if permanent reduction were possible. The high blood pressure exists to keep an adequate circulation through the diseased cerebral or renal vessels. It is surprising how often the onset of uraemic symptoms may be traced to a failure of the heart to keep up the high pressure. As the pressure falls the renal function becomes inadequate, and the patient dies of uraemia before he has time to die of heart failure. In all such cases the indication is to treat the heart and raise the blood pressure again; I have recently used pituitrin for this purpose, I think with some success.

URAEMIA.

In relation to the treatment of uraemia the following points are worth noting. (1) Patients with true uraemia occasionally recover temporarily and lead a more or less comfortable life for a few years. (2) Lumbar puncture is useful in convulsions, even in true uraemia. (3) Intravenous injection of glucose solution is probably more effective treatment than the older method of injecting saline. (4) Since the respiratory type of uraemia (so-called "renal asthma") is due to a true acidosis brought about by failure of the kidney to maintain the acid-base equilibrium, treatment by two-hourly administration of alkaline sodium phosphate in 30-grain doses, as suggested by Meakins and Davies,¹² is a rational procedure.

REFERENCES.

- ¹ Christian, H. A.: *Journ. Amer. Med. Assoc.*, 1925, 85, p. 1701.
- ² Allen, F. M., Scharl, R., and Lundin, H.: *Ibid.*, p. 1658.
- ³ Duvall, C. W., and Hibbard, R. J.: *Ibid.*, 1926, 87, p. 898.
- ⁴ McLean, H., Smith, J. F., and Urquhart, A. L.: *Brit. Journ. Exper. Path.*, 1926, 7, p. 360.
- ⁵ Cowell, S. J.: *Ibid.*, 1928, 9, p. 164.
- ⁶ Volhard, F.: *Die doppelseitigen hämatogenen Nierenerkrankungen*, Berlin, 1918, p. 416.
- ⁷ Volhard, F.: *Loc. cit.*, p. 201.
- ⁸ Epstein, A. A.: *Amer. Journ. Med. Sci.*, 1922, 163, p. 167; and other papers.
- ⁹ Epstein, A. A.: *Arch. f. Verdauungskrankheiten*, 1928, 44, p. 31.
- ¹⁰ Jungman, F. N.: *Deut. med. Woch.*, 1928, p. 41.
- ¹¹ Keith, N. M., Barrier, C. W., and Whelan, M.: *Journ. Amer. Med. Assoc.*, 1925, 85, p. 799.
- ¹² Meakins, J. C., and Davies, H. W.: *Respiratory Function in Disease*, London, 1925, p. 140.

Memoranda:

MEDICAL, SURGICAL, OBSTETRICAL.

HEROIN AFTER ABDOMINAL OPERATIONS.

THE interesting article by Mr. A. E. Mortimer Woolf, on March 16th (p. 499), tempts me to record an experience of my own.

A woman, aged 29, and in other respects perfectly healthy, was operated on by Mr. Geoffrey Bate for a twisted left ovarian cyst on March 22nd, 1928, at 9.30 a.m. Before the operation she received a hypodermic injection of 1/6 grain morphine and 1/180 grain atropine. No difficulties were encountered either with the operation or with the anaesthetic, which was administered by Mr. C. N. Chadborn.

At 3.30 p.m., as the patient complained of some pain, she was given hypodermically, 1/12 grain heroin hydrochloride. The matron of the nursing home, feeling tired herself, sat down in an armchair after giving the injection of heroin, intending to rest in the patient's room. Half an hour later, hearing the patient grunt, she rose from her chair, and found her cyanosed and comatose, with complete cessation of respiration. Artificial respiration was started at once, oxygen freely given, and 1/20 grain of strychnine was injected hypodermically. When seen by me fifteen minutes later the pupils were small, but reacted to light, the corneae were insensitive, the respirations nine per minute, and the pulse good. Half an hour later the patient had come round completely.

There cannot, I think, be any doubt but that had the patient been alone in the room at the time, as might well

have happened, she would have been found dead. The symptoms of poisoning were attributed at the time to an idiosyncrasy, and I do not think were in any way connected with the operation six hours earlier.

Personally I agree entirely with what Mr. Mortimer Woolf writes as to the dosage of heroin, 1/12 grain of which is nearly always an adequate dose, although the tendency in recent years appears to favour 1/6 grain.

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AN UNUSUAL CASE OF ECLAMPSIA.

THE case of eclampsia reported below may be of interest for the following reasons: (1) the strictness of the antenatal care throughout; (2) the unusual delay between the confinement and the onset of the fits; and (3) the large number of fits.

The patient, who was 26 years of age, came up for first examination in the twenty-fourth week of pregnancy. Her history revealed nothing of interest, though she had nursed two patients of mine with mild scarlet fever a year previously. She was a well-built country woman, and her urine was free from abnormalities. She was asked to report at monthly intervals.

At the next attendance (thirtieth week) there was a slight cloud of albumin in the urine. She was put on a modified protein-free diet and advised more rest, and told to report in a fortnight. This time the albumin had increased, and there was slight oedema of the feet. Blood pressure 150/110; fundi normal.

She was put on a strict milk diet and confined to bed. The following week the albumin decreased and the blood pressure was 140/90. The diet was maintained with the addition of carbohydrates, and she was allowed up for two or three hours in the afternoon. The next week the albumin increased again, and she was ordered back to bed, where she remained until her confinement, which took place a fortnight before full term. She had a remarkably easy labour, lasting only four hours, and was delivered at 7 a.m. on December 16th, 1928, of a healthy female child weighing 4 lb. 8 oz. The next three days were uneventful. She felt very well and the temperature and pulse were normal. She was still on a milk diet. Without any warning, at 5 a.m. on the morning of the fourth day after labour, she had her first fit. In all she had twenty-three severe fits.

She was removed five miles in an ambulance to the War Memorial Hospital, Melton Mowbray. Before removal she was given 2/3 grain of morphine sulphate hypodermically in two separate doses. On admission her stomach and colon were washed out with sodium bicarbonate; 2 oz. of castor oil were left in the stomach, and she had 2½ drachms of chloral hydrate, in 30-grain doses every two hours, per rectum. Venesection was performed and 15 oz. of blood withdrawn; 1/50 grain of nitroglycerin was given hypodermically. She was catheterized every eight hours. She had at six-hourly intervals 1½ pints, 1½ pints, and 1 pint of saline subcutaneously.

The urine at first was solid with albumin, but this has diminished every day. Her recovery has been uninterrupted. At no time has the fundus oculi been other than normal.

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A COLOSSAL OVARIAN CYST.

THE following extraordinary case seems of sufficient interest to warrant recording.

A married woman was sent into hospital by Dr. Milburn of Kingston with a diagnosis of ovarian cyst. The patient stated that she had had a swelling for five years, but since Christmas it had increased to such an extent that she could hardly get about. She suffered little or no pain, no trouble with micturition, but much constipation.

On examination, I found that she was 4 ft. 10 in. in height, and her abdomen measured 56½ in. at the level of the umbilicus. There was no swelling of the lower extremities, her heart and lungs were fairly normal, but she looked like bursting at any moment. On February 2nd, under novocain, I made a small incision to the right of the navel and soon came upon the thickened wall of the cyst. I inserted a De Pezzer's self-retaining catheter with a clamp on the end, and had the fluid drawn off gradually at the rate of one gallon every two hours. Had I removed the whole cyst at one operation I feel sure she would have succumbed. On February 3rd the pulse, which had been rapid and somewhat irregular, had recovered, so I decided to operate. By a mid-line incision I removed the cyst, which was adherent over the whole surface of the abdominal cavity, and very thick-walled and multilocular, and non-malignant.

The thick brown fluid was very carefully measured, and amounted to 55½ pints. The patient made an uninterrupted recovery and left the hospital in eighteen days.

It seems very strange in these days that anyone should develop such a huge swelling without seeking medical advice. The amount is surely a "record" for so small a woman.

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