have been given calomel in the form of teething powders. The connexion has not been finally proved, but in some cases the renal symptoms entirely cleared on withdrawal of calomel and returned when its administration was recommenced. Others have recorded similar cases occurring during troxidone therapy for petit mal.

Diagnosis

In the acute stage the diagnosis of Type 1 nephritis usually presents no difficulty, but in a few cases the symptoms are predominantly those of acute heart failure. Whether this is due to the acute hypertension which accompanies the illness or, more probably, to haemodilution as a result of retention of water and salt is not entirely established. Sometimes the signs and symptoms of the acute stage are minimal and may pass unnoticed. Sometimes convulsions are the first evidence of disease.

Acute focal nephritis is looked upon by some as a subvariety of acute nephritis. Typically it is seen at the height of an infection, and manifests itself solely by haematuria without oedema or hypertension. Its prognosis is good, but it may return if the infection recurs. It is much less common now that most pyogenic infections are promptly treated by sulphonamides or penicillin.

The oedematous stage of Type 2 nephritis is indistinguishable on ordinary clinical examination from two other disorders which cause the same syndrome—namely, the diabetic glomerulosclerosis which occurs as a (very deadly) complication in some diabetics of long standing, and amyloid disease affecting the kidney. In the first the history of preceding diabetes gives the clue; in the second the history of chronic purulent or tuberculous infection. The correct diagnosis of amyloid disease is very important nowadays, and may often be confirmed by the congo red test (the injected dye disappears very rapidly from the blood serum as it is taken up by the amyloid material). I have seen several cases of amyloid disease with gross albuminuria and renal oedema recover completely when an efficient attack on the underlying infection was made by surgery and antibiotics.

The finding of a symptomless albuminuria at a routine examination may raise a diagnostic problem which cannot here be dealt with fully. Tests for orthostatic albuminuria, tests of renal function, radiology, and cystoscopy may all have to be employed. Patients in the latent stage of Type 1 nephritis often feel perfectly well and may come into this group, the albuminuria being found by an examination for military service or for life assurance. There should be a history of an acute attack of nephritis in the past, and renal-function tests (concentration power and urea clearance) will show impaired efficiency. The blood pressure will be raised.

Chronic non-oedematous renal disease, leading to renal failure, presents two kinds of diagnostic difficulty. The first is the difficulty of realizing that the patient has renal failure. The reserves of the kidney are such that patients with slowly progressive renal disease do not complain of symptoms until renal failure is in a very advanced stage. If it is accompanied (as is often the case) by hypertension it is usually the hypertensive complications, such as headache, retinopathy, or cardiac failure, which bring the patient to the doctor. It may then be difficult to know whether the hypertension or the renal disorder is primary. I have dealt with the relation of hypertension to renal disease in a previous article of this series (*British Medical Journal*, 1950, **1**, 951). Patients with renal failure who have no severe hypertension most often complain of the symptoms of anaemia—languor, pallor, dyspnoea, and palpitation—for a normochromic anaemia, unresponsive either to iron or to liver, invariably develops as the blood urea rises. Others make no complaint until advanced uraemia brings with it anorexia, vomiting, loss of weight, and extreme weakness. Uraemia should be suspected if such a patient has a dirty pallor like fading sunburn, if he complains of thirst and nocturia, and if his skin is dry. In my opinion uraemia has no characteristic smell, but a dry dirty mouth with bleeding gums (which are common in the terminal stage) provide what has been mistaken for it.

Practically all cases of renal failure (uraemia) have albuminuria, and all have a raised blood urea, so it is not difficult to prove the diagnosis once suspected. When renal failure has been diagnosed the problem is to determine its cause, and this is important, for a few cases are amenable to treatment. Dr. D. A. K. Black has dealt with the subject of uraemia in his article in this series (*British Medical Journal*, 1950, **1**, 893), but some points in diagnosis must be restated.

Uraemia may occur as the end stage of any progressive renal disease, including the various types of nephritis which are the subject of these articles. The commonest causes are nephritis, chronic pyelonephritis, and the malignant phase of essential hypertension. Of these, chronic pyelonephritis requires emphasis particularly as an important cause of renal failure. A history of acute "cystitis" or "pyelitis" in the past normally provides the clue. In the chronic stage the urine may no longer show evidence of active infection. When uraemia terminates Type 1 or Type 2 nephritis there should be a past history of an oedematous phase, which may have occurred many years before.

Extrarenal uraemia may occur in salt deficiency and in alkalosis and is especially important in diagnosis, as it is curable.

It is a fair rule that in the young the commonest causes of renal failure are nephritis and chronic pyelonephritis. In the middle-aged malignant hypertension is added to these two. In the elderly the diagnosis of uraemia is usually wrong, except in association with prostatic disease. Most elderly patients suspected of uraemia have cerebral arteriosclerosis with thrombosis. A few, however, are genuine, usually from chronic pyelonephritis or multiple myelomatosis.

Hypercalcaemia must not be forgotten in the differential diagnosis of renal failure. It may occur in hyperparathyroidism, or as the result of taking calciferol in high dosage, and is curable if not too advanced. There are, of course, many other causes of renal failure, including congenital and so-called surgical renal disorders.

[*Part II, which will appear in our next issue, will deal with the prognosis and treatment of nephritis.*]

Correction.—In the Refresher Course article on "Rheumatic Fever" (July 19, p. 142) vitamin K is recommended for the treatment of haemorrhagic complications of salicylate therapy. The preparation suggested is Inj. menaphthon., 20 mg., but it should be given intramuscularly, *not* intravenously as stated in the article. Inj. menaphthon. is an oily solution and therefore unsuitable for intravenous injection.

SHIP SURGEON

[FROM A SPECIAL CORRESPONDENT]

Going to sea, like playing golf, is a pastime most doctors find time for only at the two extremities of their professional lifetime. There are a handful of practitioners who have made the Merchant Service their career, but these are hard-worked men on big P. & O.s and Cunarders with a couple of thousand people under their care, two or three ship's hospitals, an operating theatre, and even an *x*-ray apparatus. My life illustrates the more usual existence of a ship surgeon: I go to sea for a holiday, which I am taking at present as the only doctor on a small passenger steamer outward bound for Australia.

The medical centre of my ship is next to my cabin, at the after end of the promenade deck. There is a small hospital with four bunks in it, a bathroom, and a combined consulting-room and dispensary next door. At nine in the morning I hold surgery for the crew, with the assistance of my hospital attendant. He is a capital fellow, whose accomplishments include the ability to tie a perfect figure-of-eight bandage, play the saxophone, and perform card tricks, all of which he demonstrates to me frequently. He has an efficient way of organizing the untidy line of men who collect