

parents, the weight record at clinic was persistently unsatisfactory, and the briefest observation of the mother handling her baby showed her uncertain of herself and needing sympathetic guidance. In fact the only advice given was always to use boiled water for washing the baby's face, and that "babies always cry a lot." No instruction or supervision was offered, though the importance of regular attendance at clinic was stressed repeatedly. The mother was rated because she would miss one clinic while staying with a general practitioner's family. There, two days' common sense and precept restored peace and plenty.

If local authorities are determined to usurp the position of the family doctor in maternity and infant welfare, and make a fetish of clinic attendance, it would be well that the job were competently done from a family viewpoint, with adequate domiciliary supervision and consistent advice. It also seems unnecessary to pay doctors who leave their patients to such mass-production services at the same rate as those who, as is usual in rural areas, accept and carry out personally their responsibilities to the family.

It so happens that this patient is a French doctor's daughter. I wonder what her father now thinks of British maternity and infant care?—I am, etc.,

Norwich.

JOHN A. EDDINGTON.

### Mercurial Diuretics and Nephrosis

SIR,—Clinical and necropsy findings similar to those described by Dr. Margaret Riddle and her colleagues and by Dr. J. Burston and his colleagues in your issue of May 31 (pp. 1274 and 1277) were noted in two adults who died in the Doncaster Royal Infirmary. The outstanding histological changes were damage of the proximal and distal convoluted tubules progressing to extreme flattening of the epithelium and dilatation of the lumen, which was filled with eosinophil debris. Minimal regenerative activity was noticed only in one of the cases.

This case, a man of 65, had recurrent swelling of the legs for five years as the result of myocardial fibrosis. He was treated with a course of mersalyl in 1952 and chlormerodrin in 1955 and 1956 respectively. When admitted on June 6, 1956, he had generalized oedema, fundi normal, blood pressure 120/80; serum albumin 1.1, globulin 3.8, g./100 ml.; electrophoresis showed a marked increase in the alpha and a slight increase in the beta globulin fraction and decrease of albumin and gamma globulin; serum cholesterol was 650 mg./100 ml. and blood urea 45 mg./100 ml. There was no response to mersalyl. He died on July 31 in uraemic coma. The necropsy findings were oedema of lower extremities, ascites, and a left pleural effusion, myocardial fibrosis resulting from coronary sclerosis. The kidneys weighed 480 g. and contained mercury, 2.8 mg. per 100 g. of weight; the renal venous blood contained 1.0 mg./100 g. Sarcoid granulomata were present in spleen and liver.

The second case was a male aged 48, treated with mersalyl several weeks prior to admission to hospital for cor pulmonale resulting from pulmonary emphysema. He died three days after admission.

An infant aged thirteen weeks which died from thrombosis of the dural sinuses following "enteritis" showed renal lesions identical with those seen following the use of organic mercury compounds. In addition, deposits of calcium phosphate were present in the necrotic cellular debris. In view of these rather suggestive findings the kidneys were submitted for analysis to Dr. A. Curry, who had carried out the previous analysis, and a level of 1.58 mg. mercury per 100 g. of kidney was obtained. Application of teething powders or calomel was denied by the parents.—I am, etc.,

Doncaster.

H. LEDERER.

SIR,—It was with great interest that I read the papers in the *Journal* of May 31 by Dr. Margaret Riddle and her colleagues (p. 1274) and Dr. J. Burston and his colleagues (p. 1277) on the nephrotic syndrome following mercurial diuretic therapy. I wish to report a further such case, which I feel may be of additional interest in that it represents what is presumably a successful response to treatment of the fully developed syndrome with B.A.L. (dimercaprol).

A man, aged 66, was admitted to Queen Mary's Hospital, Roehampton, on July 19, 1956, complaining of progressive swelling of the legs of two months' duration, despite regular injections of meralluride sodium which he had been receiving, together with digitalis folia, gr. 1 (65 mg.) twice daily, ever since he had been an in-patient in June–September, 1955, with cardiac failure secondary to hypertension and myocardial ischaemia, with auricular fibrillation.

On the present admission he was not in cardiac failure, but showed gross generalized oedema, most marked in the lower limbs, and a left-sided pleural effusion. His blood pressure was now only 140/95, but there was well-marked cardiac enlargement and controlled auricular fibrillation was present: an E.C.G. showed, in addition, ventricular extra systoles and a left bundle-branch block. Gross albuminuria was present (8.6 g. per litre) and biochemical investigations confirmed the diagnosis of nephrotic syndrome. Total serum proteins were 3.6 g./100 ml., with albumin 1.5 g. and globulin 2.1 g. Electrophoretically, in addition to a low albumin, there was a marked increase in the alpha II and some rise in the alpha I globulins. The serum cholesterol was 470 mg./100 ml., and blood urea 57 mg./100 ml. Serum sodium, potassium, and chlorides were all within normal limits. Mercurial diuretics were stopped and he was treated with a high protein, low salt diet with little effect, until a course of dimercaprol was given (total dose 1.2 g. spread over six days). A most gratifying diuresis commenced three days after the initiation of this therapy and continued for almost three weeks after its completion, the urinary output being constantly between 90 and 110 fl. oz. (2.7 and 3.3 litres) daily. The oedema gradually subsided, while the blood chemistry concomitantly returned to normal limits with the ultimate disappearance of albuminuria. He was eventually discharged from hospital, the single course of dimercaprol only having been given.

When seen as an out-patient on December 17, 1956, he was oedema-free, with no albuminuria and with completely normal blood chemistry, there being no evidence whatever now of a nephrotic syndrome. He had been maintained on digitalis, but had had no further mercurial diuretics. The biochemical response to treatment at this stage may be summarized as follows:

Date	Urine Protein (g./litre)	Serum Albumin (g./100 ml.)	Serum Cholesterol (mg./100 ml.)	Blood Urea (mg./100 ml.)
20/7/56	8.6	1.5	470	57
2-7/8/56	Course of dimercaprol			
20/8/56	1.5	2.2	370	38
14/9/56	1.0	3.1	320	44
10/10/56	Trace	3.1	230	30
12/12/56	Nil	4.8	170	39

He was readmitted on May 27, 1957, with acute on chronic bronchitis and cardiac failure, as a result of which there was now a trace of albumin in the urine, but the blood chemistry was still within normal limits. His condition gradually deteriorated and he began to have Stokes-Adams attacks, E.C.G. now showing auricular fibrillation and a varying left and right bundle branch block. He died in such an attack on July 19, 1957. At necropsy the heart weighed 864 g. and there was considerable enlargement of the left ventricle and dilatation of the right ventricle; serious coronary sclerosis was present, with occlusion to a marked degree in the right coronary artery and a thrombosis about 3 cm. down its course. There were congestive changes in all the viscera. Kidneys showed no naked eye evidence of disease, and the histological report (Dr. J. Kohn) was as follows: "Kidney sections show traces of tubular damage with regeneration. There is, however, no evidence of active tubular damage present. Glomeruli are intact. Scattered foci of small cell infiltration present. The kidney is grossly hyperaemic, probably due to the terminal cardiac failure."

One would say that the post-mortem findings, in relation to the kidneys, provide confirmatory evidence of a reversal of the nephrotic syndrome in this case.

My thanks are due to Dr. G. E. Hosking for permission to report this case.

—I am, etc.,

London, S.W. 15.

G. CLENNAR.