Though the renin content of a kidney is stated to be reduced by denervation and steroids (as in kidney allotransplantation presumably) there appears (in a large proportion of rejection episodes) little interference with the mechanism which allows hypertensive states to be reached.

So far as I can determine by experiment, the role of renin in renal failure remains an interesting speculation lacking direct evidence in its support. There is, however, some direct evidence against its primary involvement.—I am, etc.,

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Diarrhoea and Light Therapy in Neonates

SIR,—Several different centres have shown that the maximum level of serum bilirubin in premature infants is significantly lower in those exposed to light therapy.¹ So far as we are aware there has been only one reference² to the occurrence of diarrhoea associated with this treatment.

Recently we have started using an air shield phototherapy unit manufactured to the specifications of Dr. J. F. Lucey.³ In several infants about three hours after starting treatment frequent loose stools were passed, and this continued until the therapy was stopped altogether or the light was turned off for one hour in every four.

We consider that this complication should be more widely known, otherwise an outbreak of acute gastroenteritis may be feared. We are intrigued by this phenomenon and wonder whether any of your readers have noted it and have worked out the aetiology.—We are, etc.,

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Azotaemia Aggravated by Tetracycline

SIR,—I agree with Dr. O. M. Edwards and others (3 January, p. 26) that tetracycline may cause clinical deterioration in patients with any degree of renal insufficiency more often than the reports suggest.

There are many reasons for this lack of reports. Firstly, lack of suspicion, particularly where there is no known renal disease or the non-specific symptoms of anorexia,

nausea, vomiting, etc., can be accounted for by the illness for which the drug was given. Secondly, the previously popular five-day course may have been too short to produce the full effect—if this is so, cases may be expected to increase with the larger courses, and perhaps with newer formulations such as Chymocyclar. Thirdly, there is often considerably difficulty in proving the relation of tetracycline to the rise in urea because immediately pre-tetracycline urea levels are not available. Thus of sixteen cases where it has been highly likely that deterioration was caused by tetracycline in only two were the relevant urea levels known.

The urea curves for these two patients were strikingly similar to those of Westminster Case II (see Figure) and the two

FIG.—74-year-old man admitted with lobar pneumonia. Deteriorated during convalescence.

cases reported by Wray and colleagues,¹ and illustrated some important practical points. Firstly, there is a delay of several days after starting the drug before the increasingly rapid rise of urea begins, so that too early monitoring of the urea may miss the rise and produce a false sense of security. Secondly, there is a further delay after withdrawal of the drug before the equally rapid fall of urea starts. During these days the urea may continue to rise, making it difficult to resist requests for immediate dialysis. It is usually, however, reasonable to wait and see, provided no dangerous complication such as severe hyperkalaemia is present. Thirdly, if the expected fall does not occur this may be due to several reasons, apart from renal disease. The extent of the fluid intake/output imbalance may not have been appreciated if assessed over too short a period and inquiry not made for any early diuresis, etc. Thus a patient may present with oliguria due either to acute renal failure or to dehydration alone. Similarly, gross calorie deficiency is common, particularly after a period of nausea and in postoperative patients who may also be hypercatabolic, and this will keep the urea high. Finally, many of these patients may have been taking high protein, electrolyte-containing food such as Complan in large amounts in spite of the maker's warning, either as outpatients or in hospital, particularly when tube-fed. Complan causes an osmotic diuresis, and, in the absence of adequate fluid intake, dehydration, hypernatraemia, and a raised urea.

Cases obviously at risk are those with renal disease and with diseases likely to be associated with renal insufficiency, such as diabetes mellitus, systemic lupus erythematosus, myelomatosis, etc., and also the el-

derly. It has been found useful to give such patients a note explaining why tetracycline is contraindicated and to ask their family doctors to avoid prescribing it if possible.

It is impracticable to monitor all patients on tetracycline in general practice, but unexpected deterioration should raise the suspicion of underlying renal disease and calls for urea and electrolyte estimations. Abnormal results should lead to immediate withdrawal of the drug and full investigation.

The case shown in the Figure was admitted to Stoke Mandeville Hospital in 1965 under the care of Dr. W. B. Thomson, who diagnosed tetracycline-induced azotaemia.—I am, etc.,

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Dermatitis Herpetiformis and Gluten-free Diet

SIR,—Drs. R. Marks and M. W. Whittle rightly say (27 December, p. 772) that we have failed to find improvement in the rash of dermatitis herpetiformis in response to treatment with a gluten-free diet,^{1,2} and indeed our more recent observations provide further evidence for this view.^{3,4} This failure of the rash to respond to gluten withdrawal was seen in patients who over the same period of time showed considerable improvement in their coeliac syndrome. Our observations are not so very different from those of Drs. Marks and Whittle, though the conclusions both they and, on another occasion, Fry *et al.*⁵ draw differ from ours.

The authors point out the difficulties which arise in assessing the result of treatment in a condition which has a fluctuating course and a reported mean duration of four years,⁶ and we believe the apparent "improvement" in the rash in the majority of cases they describe can equally well be attributed to the natural history of the disease. In addition, the fact that the dose of dapsone (mean 144 mg./day and range 50-300 mg./day) taken by their patients before starting the diet is considerably greater than that in our own patients, some of whom are taking as little as 100 mg./week, makes us wonder whether they omitted to establish that the dose of dapsone taken by the patient before as well as after starting the gluten-free diet was in fact the minimal dose required to control the rash. If the minimal effective dose of dapsone was not established before starting the diet this would explain the apparent decrease in dapsone requirements while the patient was on the diet. In any case it may be that the amount of dapsone required to control the rash is not the right way of assessing the effect of the diet on the skin, and that the question we should be asking is, "Is the rash any better when the dapsone is stopped while the patient is taking the diet than while he is eating normal food?" In the patients in whom we have stopped the dapsone in the course of the gluten-free diet we have found the rash to return with the same severity as before treatment was started.³

The debate now centres on the patients who were able to stop dapsone completely