responses, some of which might promote either heamorrhage or its deleterious effects. Clearly, these data should be studied before a new therapeutic regimen is tested in man. Recent work suggests that after haemorrhage Nature herself cannot constrict the intestinal bed by sympathetic means and has resorted to the use of vasopressin. [11] [12] I am, etc.,

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Beriberi in Bethnal Green

SIR,—Dr. M. W. P. Carney's account of two cases of beriberi (10 April, p. 109) prompts me to report another case recently seen.

A woodmill worker, aged 64, presented to his general practitioner with a three weeks' history of shortness of breath and purulent cough. He was noted to be cyanosed and to have heart failure and was admitted on 1 March 1971 to Bethnal Green Hospital. On admission his jugular venous pressure was elevated to the angle of the jaw, he had slight ankle oedema, and a good volume pulse. The chest showed evidence of extensive infection and the liver was enlarged four finger breadths. A diagnosis of acute on chronic bronchitis with cor pulmonale was made and he was treated with tetracycline and cyclopenthinzide (Navidrex K).

His infection rapidly improved, but the oedema became progressively more severe and he gained ½ stone (3·2kg) in weight. His diuretics were changed to frusemide, 80 mg daily, and digoxin was added. There was no obvious improvement and spironolactone was added. There was still no improvement and therefore the possibility of beriberi was considered. He was given thiamine, 25 mg intramuscularly, followed by Parentrovite, 1 + 2 intravenously. This produced a startling diuresis of 6 litres over the next two days and in the course of a week he lost a stone (6·4 kg) on vitamin supplements and digoxin alone.

In retrospect we learnt that this man was a heavy drinker and that he was single and lived with his brother. There was no evidence of peripheral neuropathy and pyruvate levels were not performed. However, the dramatic response to thiamine in severe refractory oedema, the known poor diet, and the finding of a mild iron deficiency anaemia point very strongly to the diagnosis. This again underlines the importance of being aware that this disease is likely to occur in at-risk groups.—I am, etc.,

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Shut Away

SIR,—You are to be congratulated on the timely leading article "Shut Away" (17 April, p. 119), which presented a comprehensive and balanced sympathetic understanding of the problems of hospitals for the mentally handicapped.

The general response to the unhappy state of affairs which is being repeatedly exposed in these hospitals is predictable. Firstly there is the rush to find a scapegoat, and doctors, nurses, and administrators are sitting targets. Secondly, there is the search for a magic formula which will solve all the problems.

The clamour for the appointment of a "health commissioner," another official to chase officialdom, seems strange in a country which has usually shown a suspicious disrespect for red tape. A health commissioner, however, highly paid, is not by dint of his office necessarily any more of a paragon of impartiality, knowledge, integrity, and sympathy than any other government officer or department. A health commissioner cannot himself press people to become nurses of the mentally handicapped. He cannot compel citizens to be voluntary workers in hospitals and he cannot provide money which is not there. If hospitals for the mentally handicapped are overcrowded can a health commissioner ask relatives to take patients home, or order them to visit their less fortunate kin in hospital if they do not wish to do so? Will a health commissioner direct local authorities to take patients into the community which has no facilities for them?

The danger in the appointment of a health commissioner is that it will be another mechanism by which the community can escape from its responsibilities to the mentally handicapped. It is an easy manoeuvre to solve the public's conscience in the face of a problem about which so many people still prefer not to know.—I am, etc.,

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Bacteriuria Again

SIR,—Your leading article (13 February, p. 361) suggests that gentamicin can cause vestibular damage "even when serum levels are within what is considered to be the safe range."

It is our duty to base dosage recommendations on clinical experience. Such experience has shown that labyrinthine damage occurs only if the blood level exceeds 10 μg/ml.[1] In the few cases of labyrinthine damage which have been reported to us the blood level was in excess of this limit because of significant impairment of renal function or unnecessarily high or prolonged dosage in patients with normal renal function. It is perhaps noteworthy that a level of 10 μg/ml has frequently been exceeded without toxic effects.

In return, it is the duty of clinicians to report clinical details if they have found that lower concentrations have produced vestibular damage in order that recommended limits are indeed safe. Reports so far received certainly do not warrant the comment that "gentamicin is notoriously toxic," and the evidence justifies our current recommendation which may even prove to be conservative.—I am, etc.,

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1 Garrod, L. P., and O'Grady, F., *Antibiotic and Chemotherapy*, Edinburgh, Livingstone, 1971.

SIR,—In your leading article "Bacteriuria Again" (13 February, p. 361) the statement that generation can cause vestibular damage "even when serum levels are within what is considered to be the safe range" requires further examination.

It has been advised by the manufacturers that a serum gentamicin level of 10 μg/ml should not be exceeded. Serum levels of less than 10 μg/ml have occasionally been reported in patients with ototoxicity during treatment with gentamicin, but these may not necessarily have been the highest serum concentrations achieved. It is likely that peak serum levels higher than 10 μg/ml may well have occurred following injection in these patients. Patients with ototoxicity with normal renal function have in retrospect had grossly excessive dosage, or have been assumed wrongly to have normal renal function. More than 50 of the few reported patients with ototoxicity due to gentamicin therapy have had impaired renal function.[1] Unfortunately, insufficient data are provided to allow estimation of the probable serum levels obtained with the dose regimen employed in these patients, but accumulation to high serum and tissue levels probably occurred. Since using the previously reported dose regimen designed to avoid this occurence[2] I have not encountered any instance of ototoxicity.

A large number of patients have received gentamicin therapy, usually for clinically significant urinary tract infections with multiresistant organisms including *Pseudomonas aeruginosa* and often in the presence of impaired renal function. It seems probable that ototoxicity due to gentamicin in man is dose related, and at the present time there is, to my knowledge, no evidence to suggest that valid peak serum gentamicin levels of less than 10 μg/ml have been associated with permanent vestibular dysfunction.—I am, etc.,

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Unnecessary X-rays

SIR,—Mr. David F. Thomas (10 April, p. 105) seeks support in condemning the submission of patients to indiscriminate and unnecessary *x*-ray. There can be little doubt that too many *x*-rays are ordered in casualty