Prevention of Tetanus

SIR,—You report (7 November, p. 376) five fatal cases of tetanus which occurred this year.

The first patient reported was given antibiotics and the wound stitched, but no antitoxin was given. The third patient, injured in a car accident, was given antibiotics but no tetanus antitoxin. Finally, the fifth case, a two-year-old boy who had not been immunized against tetanus, was given tetanus toxoid, but no tetanus antitoxin. So he received no protection against tetanus developing from his lacerated scalp. These people might not have died if they had received tetanus antitoxin.

I think the danger of anaphylactic shock developing as a result of A.T.S. has been exaggerated. The present fashion of using only antibiotics and tetanus toxoid in tetanus-prone wounds, even when the patient is not known to have been previously immunized actively, is not logical, and is unsafe in my opinion.—I am, etc.,

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Urinary Tract Infection

SIR,—Dr. A. J. Wing (26 September, p. 753 and 3 October, p. 35) has given an excellent account of the natural history of urinary tract infection and of the potentialities of antibacterial therapy. However, in relation to the common and troublesome recurring ascending infections in women, he does less than justice to the value of simple prophylactic measures, mentioning only the importance of emptying the bladder after sexual intercourse. Surely it is even more important to prevent access of faecal bacteria to the urethra by a careful perineal hygiene, especially after defaecation. The staff of this centre have found such a regimen so effective in practice that a printed sheet of instructions on perineal hygiene is handed to each patient and a copy sent to her doctor for information. Reprints of this regimen are available on request.

In several patients with persistent recurrences the daily administration of yoghurt has been successful, presumably owing to suppression of virulent faecal organisms by the lactobacillus.—I am, etc.,

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Consultancy in Radiology

SIR,—It is true that many more radiologists are in training than in the past (Dr. K. C. Simpkins, 31 October, p. 308), but this in itself does not solve the problem. It will solve it only if emigration is drastically reduced—this, I feel, is the crux of the problem. The fact that there are about 60 vacant posts and that 30 radiologists emigrated in 1968 should cause considerable disquiet. Certainly, if this rate of 30 or so per annum is maintained, the future is anything but happy.

Dr. Simpkins describes radiology as reasonably well paid. I would strongly disagree with this opinion. With the exception of those with a lucrative private practice and or with a merit award, the financial reward is not commensurate with the long and often arduous training. As radiology has less private practice than many other specialties and less chance of a merit award (this may not apply to teaching hospitals) the position is proportionally less attractive.

It should be noted that from a teaching hospital things are seen from a somewhat different viewpoint to that of a regional consultant. In a teaching hospital there are registrars, senior registrars, and trainees who can give help in many ways. Compare this with the radiologist working, sometimes alone, without help; the Department refusing to increase the establishment; and facing an ever increasing work-load as he becomes older and often unable even to obtain a locum. The teaching hospitals do invaluable and essential work, but it should be remembered that between 80 and 90% of work in the country is performed by regional consultants.

I do hope Dr. Simpkins is right and will have a happy future, but, in view of the facts, I have serious reservations.—I am, etc.,

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Corticosteroids and Glucose Tolerance

SIR,—A disassociation between the therapeutic and glucocorticoid effects of corticosteroid drugs would clearly be of clinical value and pharmacological interest. Early reports[1,2] on Sintisone (prednisolone stearoylglycolate) suggested that this steroid offered a better ratio of therapeutic to toxic effects than prednisolone, but the actions of the two drugs on carbohydrate metabolism have not been adequately compared.

We have studied prednisolone, Sintisone, and placebo in an acute test derived from the cortisone glucose tolerance test of Fajans and Conn.[3]

The subjects were mothers who had given birth to "heavy" babies, "heavy" being defined empirically as exceeding 9 lb. 12 oz. (4·6 kg.). Such subjects were selected as a group in whom the chance of discovering diabetes mellitus is increased and who might be expected to be especially sensitive to any stress to carbohydrate metabolism. Eleven subjects gave their informed consent, and underwent three 50-g. oral glucose tolerance tests. Before each test the subject received prednisolone 30 mg., Sintisone 60 mg. (an equimolecular dose), or placebo, the sequence of the respective tests being systematically varied. As the time course of Sintisone activity is slower than that of prednisolone the above dose was given in three equal parts, ten hours, four hours, and immediately before ingestion of the glucose; all tests were carried out in the morning. The results are summarized in the Tables.

All subjects showed normal glucose tolerance after the placebo tablets. Most values of blood glucose were significantly higher following either of the steroids compared with the placebo, but there were no statistically significant differences between the two steroids. The levels of plasma insulin at one and two hours were also higher than control, but similar for the two steroids.

Several authors[4,5] have reported that the therapeutic potency of Sintisone is 1½-3 times by weight that of prednisolone, so in molecular terms Sintisone (molecular weight 685) is three to six times as therapeutically potent as prednisolone. In this study we have found that doses of equal molecular content exert similar effects on glucose tolerance and insulin response. As the effect of corticosteroids on glucose tolerance is known to be dose-related,[3] a given dose of Sintisone will be less likely than a prednisolone dose of equal therapeutic effect to impair glucose tolerance in the vulnerable patient.

Sample	Control	Prednisolone	Prednisolone Stearoylglycolate
Fasting	87±4	97±2	* 98± 3
30 mins	131±7	*163±7	153± 8
60 mins	130±6	*157±8	*168±11
90 mins	106±5	*131±7	*150±15
120 mins	96±4	112±7	*124± 8
150 mins	78±4	*102±4	*105± 5

Mean (±S.E.) Blood Glucose, in mg./100 ml., fasting, and following 50 g. Glucose orally.
*Different from Control $P<0.05$.

Sample	Control	Prednisolone	Prednisolone Stearoylglycolate
Fasting	4·5±0·3	5·0±0·3	* 5·8±0·4
60 mins	10·8±1·5	*21·2±3·6	*19·0±2·3
120 mins	5·9±0·5	* 8·4±1·2	4·5±1·3

Mean (±S.E.) Plasma Immunoreactive Insulin, in μu./ml., fasting, and following 50 g. Glucose orally.
*Different from Control $P<0.05$.

I would like to thank Mr. Keith Mashiter for carrying out the estimations of plasma insulin, Professor W. J. H. Butterfield for his advice, and Carlo Erba Ltd. for their support.

—I am, etc.,

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Hyperglycaemic Hyperosmolar Coma

SIR,—The report of a case by Dr. A. I. Vinik and others (17 October, p. 155) of hyperglycaemic hyperosmolar coma occurring in the absence of detectable immunoreactive plasma insulin is of great interest. However, one cannot unequivocally accept their interpretation that this militates against the prevalent theory of pathogenesis of the syndrome—namely, that ketoacidosis is prevented by the action of some endogenous insulin.

Previous reports[1] indicate that plasma insulin is usually present in hyperglycaemic hyperosmolar stupor. In addition, this type of coma may occur in the absence of diabetes mellitus[2-4], or when maturity-onset diabetes is present, conditions in which