

expectant mother from the benefit of such a drive?

Dr. P. E. Dipple (p. 360) is at pains to defend the Clinistix test; he may have overlooked my reference to it as a "useful screening test" (p. 206). Nevertheless, being an enzyme test, it is subject to the influence of anti-enzymes such as uric acid. I have found urines with 40 mg./100 ml. glucose or more negative to Clinistix, and in this way some cases of diabetes might pass undetected; conversely, when anti-enzymes in urine are unusually low, as little as 10 mg./100 ml. glucose or less can give a positive Clinistix, resulting in an unnecessary glucose tolerance test. I think Dr. Dipple has missed the main point of my paper, which has been admirably summed up in a leading article on the subject in the same issue of the *B.M.J.* (p. 189) and I recommend him to study it.

In my investigations I have endeavoured to trace the influence of method on the incidence of glycosuria observed, and beginning with a series of 1,547 cases found only 9% by using the conventional methods of random sampling and Benedict and Clinistix testing. In a second series of 1,000 cases, using a quantitative method but still adopting random sampling, the incidence rose to 25%. In the final stage of my investigation I discarded random sampling, and by closely relating the timing of tests to carbohydrate ingestion I obtained an incidence of 90%—*the true incidence*.

As the writer of the leading article states, "Since such a large proportion of pregnant women can be shown to have glycosuria under standardized conditions it is obviously illogical to subject them to time-consuming glucose tolerance tests because of the chance finding of glucose in a random specimen of urine."—I am, etc.,

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J. FINE.

SIR,—I read with interest Dr. J. Fine's study of glycosuria of pregnancy (28 January, p. 205), but would accept his conclusions with greater equanimity if certain aspects of his statistical analysis could be explained.

The original sample of 1,547 antenatal cases is defined, as are the 242 glycosurics who had glucose tolerance tests performed with two new diabetics discovered. A second series of 1,000 patients—source unknown—are added to give a total population of 2,547, but only 50% of the glycosurics in the second series had glucose tolerance tests performed. Because he does not state how he selected this 50% it is not valid to assume that the remaining 50% of glycosurics show identical behaviour; or as Dr. Fine assumes in Table III that this 50% contains no diabetics. Similarity must be proved, not assumed. An exclusion of this size automatically tends to bias.

The method of selection of 30 patients for round-the-clock urine sugars is not stated. Neither is there any description of the age distribution and sex of the controls in any of the special subgroups. The group of 50 for 50 g. glucose are said to be unselected. This also increases bias, and a random sample would have been much better. "Unselected" in statistical terms means selected by observer bias such as a quiet clinic day, early morning arrival, co-operative patient.

In his calculation of the incidence of unknown diabetics discovered Dr. Fine assumes that there are no diabetics in the women who did not show glycosuria in routine testing. Excluding the contentious second series of 1,000 and the known diabetics he produces figures thus:

2 new diabetics from 242 glycosurics	= 0.8 %
∴ 2 new diabetics from 1,547 pregnant women	= 0.13 %

Dr. Fine admitted that it was chance that determined whether or not a woman was investigated—he did not assess the blood-sugar levels in his patients without glycosuria. There is an equal chance that the women who were not investigated had a similar prevalence of diabetes. So the prevalence of 0.8% for the sample of 242 should be applied to the whole series—the 242 shared the characteristics of the whole series. This prevalence of 0.8% is similar to the incidence found in several total population surveys to discover new diabetics.

Either diabetes is uncommon in pregnant women (0.12%) but urine testing will discover it on routine non-glucose-loaded specimens, or else it is much commoner (0.8%) and nearly one woman in a hundred may run severe risk for herself and her baby because she is not diagnosed. Instant biochemistry is not yet available for mothers not attending hospital antenatal clinics.—I am, etc.,

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SIR,—I am surprised to find Dr. J. Fine at a loss to explain glycosuria of pregnancy (28 January, p. 205).

A normal adult with a glomerular filtration rate of 120 ml. per minute develops glycosuria when the blood sugar rises above 180 mg./100 ml.—that is, when the amount of sugar delivered to the proximal tubules exceeds $120 \times 180 = 21,600$ mg. per minute. During pregnancy the glomerular filtration rate rises to 180 ml. per minute.^{1,2} Assuming proximal tubular function to remain unaltered, glycosuria is now likely to occur when the blood sugar value rises above $21,600 \div 180 = 120$ mg./100 ml.

This certainly is an over-simplification, but in general fits the facts. For instance, 15% of Dr. Fine's patients had glucose tolerance curves with a maximum below 120 mg./100 ml. (Folin and Wu), and approximately the same percentage did not have significant glycosuria.—I am, etc.,

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REFERENCES

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Mental Subnormality as a Specialty

SIR,—I read with interest Dr. D. A. Spencer's letter on "Mental Subnormality as a Specialty" (14 January, p. 110). I agree with him that the subject is badly "sold" as a desirable specialty, and that more often than not doctors enter the field by accident. This accident, however, usually has a happy outcome in that the victim enjoys his convalescence so much that he decides to remain a permanent invalid.

The average hospital for the mentally subnormal—or mentally retarded, as they are generally designated in North America—has

a wealth of material for both research and clinical practice, both of which are extremely rewarding. I agree with Dr. Spencer that affiliation to university and other postgraduate teaching centres is essential. Here in British Columbia we have vigorously pursued this aim. We have had varying success, but are encouraged by our recent attempts to link ourselves to university facilities. Starting last month, we now have a paediatric resident (registrar) on a two-month rotating basis from the university hospital. The residents in child psychiatry visit us for various clinics, whilst postgraduate students in special education also spend some time at Woodlands. Several of our staff hold university appointments. Social-work students have field-work placements with us, week-long courses are held for public health nurses (health visitors), and in the summer physiotherapists, occupational therapists, and psychologists in training all spend a few weeks at the hospital school.

Mental subnormality is a rewarding and interesting specialty which has yet to be sufficiently "advertised." As Penrose has pointed out, we can learn much about the genetics of human development, about the variables of mental illness, and indeed about education from the study of the mentally retarded. All students of the medical and social sciences should be exposed to the study of mental subnormality, which must no longer be considered a poor relation.—I am, etc.,

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Renal Failure and Low-molecular-weight Dextran

SIR,—We agree with Mr. N. A. Matheson (12 November, p. 1198) that it is difficult to determine the cause of renal failure and that the mechanism is often obscure. However, it is usual in people who go into acute renal failure to have a possible precipitating factor in the history, and if renal biopsy is performed structural changes of diagnostic nature are often found.

The group of three patients that we reported (24 September, p. 737) were culled from eight patients who developed renal failure after infusion of dextran 40. The other five were not reported because there were other possible precipitating or complicating factors (hypotension, 2; inadequate knowledge of fluid intake, 2; initial elevation of blood urea, 1). In two of the reported cases it is unusual for reversible anuria to occur suddenly. In both these cases and in two unreported cases oliguria was noted within six days of commencing dextran 40 at a time when the underlying disease process was not likely to cause renal damage.

These cases do not prove that dextran 40 causes anuria, neither does the failure to demonstrate damage in animals or volunteers prove that it is safe. Dextran 40 is not a panacea for all circulatory and coagulation problems. The increasing frequency with which renal failure is being observed and reported demands that care be taken in its use.—We are, etc.,

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