Changing Sex Ratios in Diabetes

SIR,—Dr. W. A. Nicholson (20 November, p. 465) reports an increase in the proportion of male diabetics aged 50 years and over in the second of two consecutive decades ending in 1967. We have completed a similar survey of patients attending the diabetic clinic at the Mater Misericordiae Hospital, Dublin.

Patients were classified in groups according to age at onset of diabetes—namely, at 15 years of age or less; between 15 and 45 years; and at 45 years or over. The Table

Age at Diagnosis of Diabetes	1951-60		1961-70	
	Men	Women	Men	Women
45 years or over	117 (32.8%)	239 (67.2%)	377 (41.9%)	522 (58.1%)

illustrates the sex distribution among diabetics diagnosed at 45 years of age or older in the two decades 1951 to 1960 and 1961 to 1970. The proportion of men diagnosed at 45 years or over rose from 32.8% in the first decade to 41.9% in the second—an increase of 9.1%. The figures (allowing for differences in method) are similar to Dr. Nicholson's figures of 25.9% for men at 50 years and over in the first decade and 38.2% in the second—an increase of 12.3%. The number of men diagnosed at

50 years or over in his clinic increased from 60 in the first decade to 165 in the second, an increase of 175%, while in our clinic male cases diagnosed at 45 years or over increased from 117 in the first decade to 377 in the second—an increase of 219.6%. In both surveys the increase in the number of women was much less. At the Hartlepool Clinic female diabetics increased from 172 to 262, an increase of 52.3%, and in Dublin from 239 to 522—an increase of 118.4%.

The marked increase in the proportion of

men in the older age group in both surveys, together with the less sharp increase in the number of women, supports the view¹ that a change in the sex ratio has occurred among maturity-onset diabetics.—We are, etc.,

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¹ Malins, J. M., Fitzgerald, M. G., and Wall, M., *Diabetologia*, 1965, 1, 121.

Specimens from Female Genital Tract

SIR,—We note with interest the microbiological findings in the female genito-urinary tract described by Dr. W. H. Hughes and Mr. J. M. Davies (13 November 1971, p. 424). We have been carrying out a similar but more restricted investigation in Malawi on groups of African women who are: (1) pregnant or immediately postpartum; (2) healthy and of menstrual age; (3) attending a sterility clinic. Some of our results are summarized and compared in the Table with selected results from the Hughes and Davies's London survey.

able to assess the incidence of *N. gonorrhoeae* found in fresh swabs only from a Gram film: probably therefore we have underestimated it. Nevertheless, our clinical impression of the incidence of gonorrhoea is such that even if it were practicable to mount a routine screening programme here it would not be worthwhile. The effort and money put into such a programme would be prohibitively costly for any country. A much smaller sum spent in tracing contacts of established cases more vigorously than at present would have a much greater return.—We are, etc.,

Organism	London Survey	Malawi Survey
<i>Trichomonas vaginalis</i>	35 (3.5%)	53 (28%)
<i>Candida albicans</i>	152 (15.2%)	52 (27%)
<i>Neisseria gonorrhoeae</i>	3 (0.3%)	0

Total patients examined: London 1,000; Malawi 191.

Whatever the causes, and there are probably many, the incidence of *Trichomonas vaginalis* and *Candida albicans* is much higher in the Malawian women. We were

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Neurological Disease and Folate Deficiency

SIR,—I should like to record a case of folate deficiency anaemia causing myelopathy.

A woman, aged 63, was admitted with a history of depression with anxiety of about four years' duration. She was known to be anaemic, and her anaemia had been treated with iron by her own doctor but with only limited success. Recently she had been getting weak in her legs, stayed in bed a great deal, and was complaining of numbness in her feet and legs. Her appetite was poor but her weight loss was negligible.

On examination she was sensible but depressed, anxious, and pale. She had some weakness in the hands and forearms but quite marked weakness in the legs. Tactile and pain sensation were intact but vibration and position sensations were lost in the lower limbs. Romberg's sign was positive. All the tendon jerks were brisk and the plantars were upgoing on both sides. Fundi, cranial nerves, and systemic examination revealed no other abnormalities. A clinical diagnosis of subacute combined degeneration of the

spinal cord due to pernicious anaemia was made.

Investigations showed: Haemoglobin 8.2 g/100 ml; R.B.C. 2,300,000/mm³; W.B.C. 5,600/mm³; P.C.V. 28%; M.C.H.C. 31%; M.C.V. 117 μ m³; serum iron 156 μ g/100 ml; saturation of iron 91%; total iron binding capacity 171 μ g/100 ml; platelets 264,000/mm³; serum B₁₂ 300 pg/ml; and serum folate 0.6 ng/ml.

The bone marrow was mildly megaloblastic with hyperactive erythropoiesis and increased iron storage. Myelopoiesis was active with giant metamyelocytes and hypersegmented polymorphs.

Treatment was started with hydroxocobalamin, but there was no improvement haematologically or neurologically within 10 days. Reticulocyte response was 2%. After receiving the results of B₁₂ and folate, treatment was started with folic acid 10 mg t.d.s. and hydroxocobalamin was stopped. Her haemoglobin went up to 10 g/100 ml in 15 days, reticulocyte response was 12%, and physically her improvement was remarkable. Her legs became stronger, the numbness disappeared, no sensory sign was detected but tendon jerks remained brisk and the plantars were upgoing.

It is generally accepted that folate deficiency does not cause neurological disease.^{1,2} But Grant, Hoffbrand, and Wells³ showed that there was a definite association between folate deficiency and neurological disorders. Anand described a case of folate deficiency causing peripheral neuropathy.⁴ Hansen *et al.*⁵ reported a 50-year-old epileptic man who had been treated with anticonvulsants and developed atrophy of muscles and loss of cutaneous sensibility. He died four months later from pneumonia and at necropsy lesions were found in cerebellum, spinal cord, and peripheral nerves. Thus there is some definite evidence to suggest that folate deficiency causes not only depression, anxiety, and irritability, but also neurological manifestations.

I would like to thank Dr. B. K. Samtani for his permission and suggestion to report this case.

—I am, etc.,

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¹ Cecil-Loeb, *Textbook of Medicine*, ed. P. B. Beeson, and W. McDermott, 13th edn., p. 1472. Philadelphia, Saunders, 1971.

² De Gruy, G. C., *Clinical Haematology in Medical Practice*, 3rd edn., p. 138. Edinburgh, Blackwell, 1970.

³ Grant, H. C., Hoffbrand, A. V., and Wells, D. G., *Lancet*, 1965, 2, 763.

⁴ Anand, M. P., *Scottish Medical Journal*, 1964, 9, 388.

⁵ Hansen, H. A., Nordqvist, P., and Sourander, P., *Acta Medica Scandinavica*, 1964, 176, 243.

Collecting Vesicle Fluid

SIR,—Some two years ago expediency led us to employ a disposable Mantoux syringe, fitted with an appropriately fine needle, to collect vesicle fluid from a case of atypical varicella in which virological studies were felt necessary to exclude any possibility of variola. We were impressed both by the painless ease with which the vesicles could be punctured and by the good harvest of fluid. Prompted by this early success we have continued to use Mantoux syringes for this purpose and feel it is probably a