

of connective tissue (dotted line Fig. 1A). An annular pancreas results from a defect in rotation of the ventral primordium towards the dorsal pancreatic bud [3]. The fact that all the abnormally-developed tissue showed a distinct pancreatic polypeptide immunofluorescence proves the hypothesis that the ventral primordium is the origin of the pancreatic tissue rich in pancreatic polypeptide. However, the reason for the differing pancreatic polypeptide and glucagon contents of the two primordia remains to be established.

Yours sincerely,

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Diabetologia (1982) 23: 142

Exercise-Induced Proteinuria in Diabetic Children

Dear Sir,

The November 1981 issue of *Diabetologia* contained an article on exercise-induced proteinuria in children with Type 1 (insulin-dependent) diabetes [1]. In view of the results of studies on exercise proteinuria obtained in our laboratory, it would seem likely that the use of a radioimmunoassay as a means of estimating glomerular permeability in early diabetes may be providing correct, but less than optimal, information.

Polyacrylamide gel electrophoresis studies on the nature of the urinary proteins excreted after light exercise have shown that adolescent girls with normal renal function may excrete proteins of molecular weight as large as 170,000-215,000 daltons [2]. We have found similar size proteins in the pre-exercise urines of gymnasts, physical education students and rugby football players (unpublished observations). These findings have led us to conclude that, during everyday activities, large plasma proteins may pass through the glomerular filter. Such temporary increases in glomerular permeability are enhanced during exercise and the high molecular weight proteinuria will be associated with increased urinary protein concentration.

We have found that the urinary protein profiles of patients with the nephrotic syndrome are not very different from the post-exercise urinary protein profiles of healthy young athletes. Urine from diabetic patients, studied by the same technique, showed similar protein profiles to some post-exercise samples, but in those cases where the ESR was raised the urine contained larger plasma proteins. From this point of view, the difference between exercise proteinuria and pathological proteinuria appears to be the persistence and chronicity of the latter.

Recently I have proposed that increased blood viscosity is an important factor in the mechanism of proteinuria and capillary leakage [3]. As it has been well demonstrated that diabetic patients with poor metabolic control have increased blood viscosity, it is probable that, in circumstances where blood viscosity was even slightly increased, the intra-renal vascular changes associated with exercise would result in proteinuria. However if the excretion of albumin alone was being monitored then the significance of the proteinuria might be missed. It

seems likely that those diabetic patients with raised basal urinary albumin levels who showed marked increase in albuminuria after exercise [4] also had raised blood viscosities. As normalisation of blood viscosity occurs when good metabolic control is restored in diabetes, the removal of the cause of chronic proteinuria will not prevent temporary high molecular weight proteinuria occurring after athletic activity.

In order to utilise recently published information in the fields of exercise proteinuria and haemorheology, I would suggest that, where possible, studies of 'diabetic' proteinuria should include investigations into low shear rate blood viscosity.

Yours sincerely

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