Glycosylated Haemoglobin and Red Blood Cells in Diabetes

Glycosylated haemoglobin has an increased affinity for oxygen [1], and in diabetics the number of red blood cells has been found to correlate with glycohaemoglobin (GHb) level [2]. If confirmed, this could indicate compensatory polycythaemia resulting from chronic hypoxaemia. We have reviewed in this respect our data relating to 79 adult diabetic males and 48 postmenopausal women, all of them living in the coastal area, non-smokers and suffering from no known heart, lung or blood diseases. No differences were found between patients treated by diet alone, oral drugs or insulin. Blood count was done in the Coulter counter, and GHb was determined by the Biorex 70 column method [3]. Results are presented in the table and show statistically significant but very weak correlation for GHb positively with of red blood cells ($p = 0.04$), and negatively with mean corpuscular volume ($p = 0.034$) in males only.

Table. Red blood cells (RBC), haematocrit, mean corpuscular volume (MCV), and glycohaemoglobin (GHb) in diabetics ($M \pm SD$)

	Males	Females
RBC, $10^6/\text{mm}^3$	5.00 ± 0.488	4.58 ± 0.462
Haematocrit, %	44.4 ± 3.85	40.9 ± 3.77
MCV mcm^3	89.4 ± 4.95	89.4 ± 5.89

In 21 male patients the tests were repeated after a change in the diabetic control and stabilization of GHb at another level. No significant correlation was found between the changes in GHb on

the one hand and red blood cell number ($r = -0.274$; NS) or MCV ($r = -0.012$; NS) on the other.

Our data do not confirm in this indirect way the chronic hypoxic effect of GHb. The changes in the diabetic control as reflected by GHb were not accompanied by the changes in the opposite direction of MCV or the changes in the same direction of red blood cell number. The unlikelihood of a significant hypoxic effect of increased GHb in human diabetes has been pointed out already by Bunn et al. [2].

References

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Arye Lev-Ran, M. D., Ph. D.
Scripps Clinic and Research Foundation
La Jolla, CA 92161, USA

Prevalence of Permanent Congenital Diabetes Mellitus

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A recent publication by Crossley in *Diabetologia* furthers our understanding of the epidemiology of juvenile diabetes [1]. Although the increased incidence of juvenile diabetes early in adolescence is well documented in Crossley's and other such reports [2, 3], few if any comment on what appears to be a decreased prevalence seen during the first year of life – especially the number of neonates with a permanent congenital insulin-dependent state.

We have cared for a child who had documented hyperglycaemia within his first twenty-four hours, and who continues to require insulin at the age of three years.

Perinatal history revealed the infant was delivered without complication to a 24-year-old gravida 2, para 2 mother after a 38-week pregnancy marked by glycosuria during the week prior to delivery. Birth weight was 1.950 g, length was 43.5 cm, and Apgar

scores were 6 and 9. The placenta was unremarkable and weighed 510 g.

At 24 h of age, the infant was noted to have 2% glycosuria. Blood glucose was 570 mg/dl and serum electrolytes were normal. An insulin level was 6 $\mu\text{U}/\text{ml}$ and TORCH titres were negative.

Initial treatment consisted of intravenous Ringer lactate and $1/16$ unit of crystalline insulin administered subcutaneously approximately every 8 h. Daily insulin requirements gradually increased, and the infant was discharged at ten weeks of age on $3/8$ unit of NPH every 12 h and feedings at 4 h intervals. His weight upon discharge was 2.400 g, length 45.5 cm, and head circumference 36 cm.

The infant's mother was reported to have a normal glucose tolerance test one week after delivery. The remainder of the family history was negative for diabetes or other endocrinopathies.