Glycosylated haemoglobins are synthesised throughout the life span of the mature erythrocyte by attachment of glucose to N-terminal valine of the betaglobin peptide chains by a post-translational nonenzymatic reaction. This condensation process is practically irreversible under physiological conditions and depends on the circulating concentration of blood glucose; thus, the concentration of the glycosylated haemoglobin fraction reflects the plasma glucose concentration integrated over an extended period of time determined by the life span of the erythrocyte. Glycosylation is an indiscriminate process and can affect all proteins containing free amino groups.

Blood samples were obtained from fasting diabetic patients managed as outpatients and in the emergency room and non-diabetic outpatients at the Kaiser Foundation Hospital in Honolulu. Assays were performed on fresh blood in batches consisting of equal numbers of diabetic and non-diabetic subjects.

Whole blood was used for the measurement of glycosylated haemoglobin, glucose-6-phosphate, pyruvate kinase, and phosphohexose isomerase activities. Glycosylated haemoglobin was measured by cation exchange chromatography.

As shown in the Table, the glycosylated haemoglobin concentrations were significantly raised in the diabetic group over the non-diabetic control. The three enzymes demonstrate insignificant variation in activity between diabetic and normal groups as indicated by the Student's t test. Plasma glucose concentrations among fasting diabetic outpatients ranged from 82 to 305 mg/100 ml (4.55-17 mmol/l). The red cell enzyme activities in two patients, who were initially seen in the emergency room, with highly raised plasma glucose concentrations (650 and 804 mg/100 ml (36 and 44 mmol/l)) and glycosylated haemoglobin concentrations (17.9 and 24.9 g/dl), were in the normal range—clearly demonstrating lack of effect of extreme hyperglycaemia on the activities of these enzymes. Although exposure to excess glucose has deleterious effects on some tissues, glucose-6-phosphate dehydrogenase, phosphohexose isomerase, and pyruvate kinase do not appear to be functionally altered in hyperglycaemia. However, the possibility remains that these proteins may be glycosylated at residues which do not affect the active site. McMillan et al. postulate that observed reduction in erythrocyte deformability is caused by increased cytoplasmic viscosity due to glycosylation of haemoglobin. If this is the case, it is tempting to suggest that glycosylation of other soluble proteins in the red cell may contribute to this effect.

**References**


**Enzyme activities and glycosylated haemoglobin concentrations the erythrocytes of normal and diabetic subjects**

<table>
<thead>
<tr>
<th>Enzyme Activity</th>
<th>Normal (No. Mean ± SD)</th>
<th>Diabetic (No. Mean ± SD)</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glycosylated haemoglobin (g/dl)</td>
<td>41 7.97 ± 0.89</td>
<td>42 10.96 ± 3.32</td>
<td>p &lt; 0.001</td>
</tr>
<tr>
<td>Glucose-6-phosphate dehydrogenase (IU/g Hb)</td>
<td>41 7.0 ± 1.0</td>
<td>42 6.8 ± 1.1</td>
<td>NS</td>
</tr>
<tr>
<td>Phosphohexose isomerase (IU/g Hb)</td>
<td>27 33.2 ± 5.5</td>
<td>30 34.1 ± 7.7</td>
<td>NS</td>
</tr>
<tr>
<td>Pyruvate kinase (IU/ml RBC)</td>
<td>27 2.60 ± 0.43</td>
<td>25 2.44 ± 0.52</td>
<td>NS</td>
</tr>
</tbody>
</table>

NS = not significant.
Letters to the Editor


Polly-pica treated by xerography

In these days of photocopying it is difficult to know why so many reprints are requested. Below is one reason: it concerns our article which appeared at the front of this journal in 1977.1 Extracts of a letter received from the HLS Lab (Preston) are reproduced below.

"In this laboratory the J Clin Pathol is sent to the binders they are kept well hidden. Recently, contrary to normal practice, two years' issues prior to binding were sent to an MLSO preparing for her examinations. She returned them after two days, white-faced and solemn with the following story:

Her pet parrot (Fig.), a bird that seldom flies or even walks, had been left alone in the room with the journals. On her return a half-eaten apple, a vandalised plant, and the front of J Clin Pathol 1977;30:1-12 were spread over the carpet.

May we please therefore request a reprint of your article.

As well chewed over.

well chewed over.

AB Price*

*Northwick Park Hospital,
Harrow
†St Thomas' Hospital,
London SE1

Aetiological agents and laboratory diagnosis of bacteraemic shock

In your report of a symposium on septic shock,1 Dr Shanson claimed that "during the last 30 years in Britain the incidence of bacteraemias due to Gram-negative organisms has greatly increased." He gave the numbers of isolates from blood cultures during 1977 in a district hospital in London; Escherichia coli was the most frequent cause of bacteraemia.

In Ayrshire we have found that during the past two years E coli has decreased in importance as a cause of bacteraemia, probably due to increasing use of short courses of perioperative antibiotics for gastrointestinal and pelvic surgery. Thus from 1974-8, E coli was the most frequent pathogen isolated each year from blood cultures in Ayrshire. In 1979 the most frequent pathogen was Staphylococcus aureus, which was isolated from 27% (26%) of 102 patients with bacteraemia, E coli from 22%, and other coliform species from 14%.2 In 1978 the most frequent pathogens were E coli from 28%, coliform species from 20%, and Staph aureus from 15%. Isolates of Bacteroides species decreased from 9% in 1978 to 5% in 1979. The decrease in Enterobacteriaceae and Bacteroides species was associated clinically with fewer cases of bacteraemic shock.

References


References
