SUMMARY

Insulin dependent diabetes mellitus (IDDM) is considered to be the commonest endocrinal disorder in childhood.

In our study we investigated the serum growth hormone releasing hormone (GHRH) and growth hormone levels after a mixed meal. The degree of metabolic control was assessed by measuring the glycosylated haemoglobin A_{1c}. Growth parameters including weight, height and weight by stature were measured as a centile according to the age.

In our work 56 children were included:

- 20 healthy controls (10 males and 10 females).
- 36 IDDM children (12 males and 24 females).

Both groups were subjected to:

- Full history and thorough clinical examination
- Urine analysis: sugar - acetone - albumin.
- Complete blood picture and sedimentation rate.
- Kidney function tests: blood urea & serum creatinine.
- Fundus examination.
- X-ray chest.
- Serum GHRH and GH levesl were measured using radioimmunoassay.

Our results showed:

* A non significant difference between both patients and controls regarding GHRH ($P > 0.05$).

* A significant increase in s. G.H. in diabetic patients as compared to the control.

* A highly significant increase in glycosylated haemoglobin (HbA1C) in diabetic patient as compared to the control ($P < 0.001$).

* A non significant difference between diabetic patient group and control group regarding growth parameters.

Recently an argument has been held around the role of GHRH outside the hypothalamic pituitary axis. GHRH secreting pancreatic tumours causing acromegally has been identified. Many researchers has postulated the release of GHRH from the upper GIT and pancreas especially after a mixed meal. So, we wanted to find out whether there is any difference between diabetic patient group and control normal group, but we could not find a significant difference. so, it seems unlikely that circulating GHRH plays a significant, if any, role in the regulation of the metabolic control in diabetic children.

The significant increase in serum G.H. concentrations in diabetic patients when compared with the control group, with the following explanation:

* Insulin like growth factor I (IGF-I) decrease causes an increase in G.H. level as a compensatory mechanism by the pituitary.

* A failure of the pituitary to be suppressed in response to hyperglycaemia implies a possible second abnormality. It is probably more likely that glucose acts indirectly by stimulating hypothalamic somatostatin secretion. Another explanation is that although the blood glucose levels are high, the intracellular glucose concentrations are decreased i.e. "Starvation in the midst of plenty". These intracellular hypothalamic nuclei (which secrete GHRH and somatostatin) will stimulate the pituitary G.H. secreting cells.

Regarding growth parameters, there was a non significant difference between both groups. IDDM children who are now on two daily doses of insulin can maintain their growth well without a significant influence on the achieved growth. There was a non significant correlation with glycosylated haemoglobin A_{1c} i.e. degree of metabolic control.