

SUMMARY EVENING DISCUSSION

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The entire panel participated in the responses to most questions; the summary represents a consensus.

Prof. Saling opened the discussion with two important questions (1) when and whom to screen for hypoglycemia and (2) what is explanation of LGA neonate in the absence of glucose abnormalities or hyperinsulinism.

All agreed that all high risk infants be screened frequently, beginning at or shortly after birth. To the high risk group Prof. Saling added the infant with 2 hours or more tachycardia prior to delivery.

To explain increased birth weight, the following were proposed (1) HbA_{1c} to detect unrecognized gestational diabetics (2) note carefully any disproportionate organ growth e.g. small brain and kidney in the presence of excess fat, heavy placenta, liver and heart suggests hyperinsulinism. (3) Maternal diet and its composition e.g. 12,600 births compared between Palermo and Gotteborg, Prof. Karlberg found in Italy, gestation was 5 days shorter, infants 10 cm. shorter and 100 gms heavier than in Sweden. Is this related to high carbohydrate (Pasta) diet? (4) Sould pregnant women nibble rather than eat 1 or 2 or 3 meals/day?

Another major area discussed - at what level of blood glucose does brain damage occur? Is this related to rate of fall of glucose or to age of infant (hours vs days after birth)? There are no definitive answers without precise measures of neonatal brain function. Suggestions for study included the infants activity, the presence of a normal evoked potential on EEG and long term follow-up. All agreed that the new techniques to measure brain glucose metabolism as well as blood flow on an ongoing basis as is available with Positron Emitting Tomography (P.E.T.) will be necessary to answer this question.

All agreed that early feeding (breast, formula) and support of the blood glucose is important if we are to prevent whatever damage hypoglycemia may induce.

A discussion of the safety and use of intralipid indicated that while most teaching centers in Sweden use intralipid, all appear satisfied that it stimulates gluconeogenesis and provides needed calories and energy for the SGA infants. Potential dangers include increased oxygen consumption and its effect on cardiac output. In therapy, intralipid (2 gm/day) is used as an adjunct to ongoing intravenous glucose therapy.

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