## Management of Hypoglycaemia (general)

Severe hypoglycaemia can result in convulsions and coma and should be treated promptly, It may occur in the following circumstances.

1. In a diabetic child on insulin therapy
2. Spontaneously, usually as a result of substrate deficiency during an inter-current illness, or more rarely due to an inborn error of metabolism.

Any child admitted with convulsions or altered consciousness should routinely have a near-patient blood glucose test done to exclude hypoglycaemia.

### Investigation

In spontaneous hypoglycaemia (i.e. un-associated with insulin treatment for diabetes) blood should be drawn for confirmation of hypoglycaemia and to identify the cause -

**First line tests**:

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| **Plasma glucose**: | If the glucose is <2.6mmol/L, further investigation is required (see second line tests). |
| **Plasma lactate**: | Increased in metabolic liver disease, secondary to prolonged convulsions or glycogen storage disorders.  |
| **U&E**:    | Hyponatraemia and hyperkalaemia are observed in either primary adrenal insufficiency of adrenal steroid biosynthetic disorders.Urea may be increased due to pre-renal uraemia caused by dehydration due to prolonged vomiting.  |
| **LFTs**:   | Abnormal liver enzymes e.g. hyperbilirubinaemia may suggest specific disorders.  |
| **Bicarbonate**:    | Reduced bicarbonate is suggestive of a metabolic acidosis.  |
| **Cortisol**:       | A low cortisol may be suggestive of adrenal insufficiency – *interpret neonatal results with caution; there is no established reference range for the Abbott Architect.* |
| **Plasma Ammonia**: | Increased in liver dysfunction, and hyperinsulinism with hyperammonaemia. |

**Second line tests (if the glucose is confirmed to be <2.6mmol/L)**

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| **NEFA & βOHB**:    | *Will be analysed ASAP after confirmation of a low glucose*.  Lipolysis is usually suppressed in hyperinsulinism, whereas in fatty acid oxidation defects ketogenesis is defective.A ratio of NEFA:βOHB is usually <2.  Levels above this are suggestive of a fat oxidation defect.  |
| **Insulin & C-peptide:** | Will be inappropriately increased in hyperinsulinism. The C-peptide identifies the insulin source as exogenous or endogenous. |
| **ACTH**:   | Only analysed if cortisol is below 100mmol/L. An elevated ACTH with low cortisol confirms adrenal insufficiency. |
| **Growth hormone**: | May indicate pan-hypopituitarism or isolated GH deficiency. |
| **Urine organic acids**:    | May suggest a specific organic acid or fatty acid defect. |
| **Serum amino acids**:      | May suggest an amino acid defect. |
| **Acylcarnitines**: | *Only analysed if contacted by the Clinician.  Guthrie cards will be stored until all other results are available (~ 6 months)*.  Abnormal acylcarnitines may suggest a specific fatty acid oxidation defect. |

All the necessary samples are contained within the **'Hypoglycaemia Box'** located in the Children's Assessment Unit and on the paediatric wards. The box also includes a container for collection of the next voided urine sample which should be sent for analysis of organic acids and ketones **(Please ensure that urine is sent to Biochemistry and not to microbiology).** A pre-printed request form kept with the Hypoglycaemia box should be sent with the samples – Please ensure that you answer the questions on the back of the ‘HOG’ request form as these are an essential aid to interpretation of the results.

### Management

If oral glucose (including glucogel) cannot be used, intravenous glucose should be given, but **hypertonic (50%) glucose solutions can be dangerous and should be avoided**.

The following regime is suggested:

1. Give 10% Dextrose intravenously, 2 ml/Kg (200 mg/Kg) over 3 minutes.
2. Continue with the glucose infusion intravenously at a rate of 0.1 ml/Kg/minute.
3. Measure glucose concentration by Dextrostix after 4-5 minutes and adjust glucose infusion to maintain the blood glucose at 5-8 mmol/L and no higher.
* If hypopituitarism is suspected also give Hydrocortisone intravenously.( < 1 year 25mg, 1-5 years 50 mg, >5 years 100mg)
1. If there is no improvement in the state of consciousness after normal glucose concentration is restored an alternative explanation should be sought.
2. Do not give Glucagon unless venous access is not possible or is lost.

Authors: Dr A Webber, Dr J Barton, July 2002

Reviewed Dr Barton 2005,

Reviewed Dr Pryce Dr Hawkes 2014

# 7 Prolonged Fasting in the Investigation of Hypoglycaemia or Suspected Metabolic Disease

(Morris et al., Arch Dis Child 75:115-119 (1996)

Monitoring the metabolic and endocrine changes following the withdrawal of dietary energy can identify or exclude a number of serious defects in children who present with suspected or documented hypoglycaemia. The procedure, however, is time-consuming and may provide an inadequate 'stress' resulting in failure to reproduce hypoglycaemia. A diagnosis is therefore best achieved by measuring hormone and metabolite concentrations in blood and organic acids in urine **during the presenting episode** as described in the previous section.

A prolonged fast is an elective procedure and should only be undertaken when a child is well and has previously been consuming their normal diet. The maximum duration of a diagnostic fast is determined by the age of the child:-

 < 6 months 8 hours

 6-8 months 12 hours

 8-12 months 16 hours

 1-2 years 18 hours

 2-7 years 20 hours

 > 7 years 24 hours

In the young infant the fast should normally begin at 06.00 am but in the older child can usually commence overnight unless the history suggests hypoglycaemia may occur after a shorter period of fasting. The investigation should be scheduled to allow samples to arrive in the laboratory within normal working hours. The lab should have been notified in advance and all samples should be clearly labelled to include the time of the sample in relation to the fast duration.

A secure IV cannula should be in place to allow blood samples to be drawn at intervals and hypoglycaemia to be treated should it occur. Water is allowed during the investigation but no calories should be consumed. The fast must be terminated prior to the time planned if hypoglycaemia occurs or the child becomes unwell (e.g. encephalopathic), or if IV access is lost.

Blood glucose should be monitored at hourly intervals from 06.00 am (using calibrated glucose meter) and at least 3 blood samples obtained during the course of the fast for measurement of metabolite and hormone levels (precise timing should be agreed with the consultant). **It is essential that blood is obtained for analysis before hypoglycaemia is corrected** or the fast terminated for any reason. The last urine sample passed during the fast or after correction of hypoglycaemia should also be collected for organic acid estimation.

Intermediate fasting samples request - Glucose, Lactate, Pyruvate, 3- OH butyrate, free fatty acids.

Final fasting sample - Glucose, Lactate, Pyruvate, 3- OH butyrate, Free fatty acids, Insulin, Cortisol, Growth Hormone, Filter paper blood spots for Acyl Carnitines.

**The child must have a meal and normal post-prandial blood glucose before going home.**

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