

and are tested using glucose dehydrogenase pyrroloquinolinequinone (GDH-PQQ)....

Glucose oxidase based systems can as well be influenced.

StatStrip glucose meter (Nova) compensate for interference effects.

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DO WE MANAGE TO FEED THE PAEDIATRIC CARDIAC PATIENT ENTERAL AFTER OPEN HEART SURGERY?

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Aim: PICU in Lund are using a feeding protocol with guidelines for enteral feeding. The Schofield equation for weight to predict the basal metabolic rate is used for calorie prediction. The aim of this study was to examine how the paediatric cardiac patient after open heart surgery is fed enteral at PICU.

Method: Retrospective study including 64 patients between 1 day and 14 years (mean 14 months). Patient's undergone openheartsurgery requiring CPB with a minimum stay at PICU for three days.

Results: Standard is bolus feeding every three hours. Mean ventilator days 3, 3 days. 97 % of the patients had started enteral feeding within 24 h. Predicted calories were reached in 4, 3 days. Differences were seen between groups of age. Age 3-12 months had 70 % reached their goal on postoperative day 3. Children older than 6 years did not reach the goal before leaving PICU. No significance between time on CPB, cross-clamp time and when the calories were reached. Significance were seen between days on ventilator, patients left with open sternum and reached calories. 67% of the patients had bowel movements or stool day 2. TPN were started on 3% of the children. Reasons for not reaching the goal faster were time of fasting. The most common reason for fasting was extubation.

Conclusion: The results shows that enteral feeding is started fast and the goals are possible to reach in the first five days. Problems are seen in the older children.

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VITAMIN D DEFICIENCY AND HYPOCALCAEMIC CARDIOMYOPATHY

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Aims: The aims of this study were to document the incidence of hypocalcaemic cardiomyopathy and to highlight its association with vitamin D deficiency.

Methods: A retrospective chart review was performed of all infants presenting to our Cardiology service with hypocalcaemic cardiomyopathy. Laboratory profiles were documented for each infant, specifically serum calcium, alkaline phosphatase, parathyroid hormone (PTH) and 25OH-Vitamin D. Maternal blood results and the method of infant feeding were also documented.

Results: Two male infants presented between January 2009 and March 2010, aged 24 days and 3 months respectively. Both were exclusively breastfed, and were of African origin. Intubation, ventilation and inotropic support was required for each baby. Echocardiography confirmed dilated cardiomyopathy. Blood results were consistent with vitamin D deficiency; calcium 1.23mmol/l and 1.28mmol/l, alkaline phosphatase 988U/l and 1487U/L, PTH 150.6ng/l and 392ng/l and 25OH-vitamin D 12nmol/l and 10.4nmol/l. The mothers were both confirmed as being vitamin D deficient.

Conclusions: Hypocalcaemia is a recognised cause of dilated cardiomyopathy. Vitamin D-deficient rickets is re-emerging as a clinical entity in Ireland. The Food Safety Authority of Ireland recommends that all exclusively breastfed infants, particularly those who are dark-skinned, receive 200 IU of vitamin D from birth to 12 months of age. This is not universally implemented. To date there are no further recommendations to assess or improve vitamin D status in population subgroups, such as pregnant women. Health professionals and parents should be made aware that vitamin D deficiency is prevalent in Ireland, particularly among dark-skinned infants.