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Fetal Alcohol Syndrome and Lactic Acidosis

To the Editor:

We recently saw a newborn infant with the fetal alcohol syndrome and lactic acidosis. The mother was 32 years old, gravida 5, para 2, with one elective abortion. One previous infant had died in the perinatal period, of massive congenital anomalies; a second infant died with the sudden infant death syndrome. The mother had a long history of chronic alcohol abuse. After she learned of the current pregnancy, she became depressed and went on a drinking spree. She continued to drink to some extent during most of the pregnancy.

The baby was delivered at term, by cesarean section because of fetal distress. It was a 2.77-kg male with Apgar scores of 4 and 7 at 1 and 5 min. Resuscitative efforts were required at birth, and the child developed transient tricuspid insufficiency due to asphyxia.

Tachypnea continued after birth and the infant had hypoglycemia (blood glucose concentration of 260 mg/dL). Arterial blood gas studies showed metabolic acidosis: arterial pH of 7.32, pCO2 of 16 mmHg, and [HCO3-] 8.3 mmol/L. He was treated with bicarbonate and glucose, and over the several days continued to have tachyphnea and compensated acidosis. Six days after delivery, blood lactate concentration measured 7.6 mmol/L, pyruvate 10 μmol/L (760:1 ratio). The child was diagnosed as having fetal alcohol syndrome and an associated lactic acidosis. The next day the lactate concentration had declined to 4.3 mmol/L, and both child and mother were discharged. Two weeks postpartum, the infant was seen in the outpatient department for failure to thrive and was referred to a medical geneticist for further studies.

Lactic acidosis is a common finding in adult alcoholism but apparently has not been frequently seen in the fetal alcohol syndrome. The metabolism of ethanol to acetaldehyde and acetate by the microsomal ethanol-oxidizing system leads to a stoichiometric increase in the NADH/NAD+ ratio. An increase in reduced equivalents in the form of NADH will lead to at least two metabolic abnormalities: increased lactate, due to a shift in the redox ratio of lactate to pyruvate mediated through the lactate dehydrogenase reaction, and a decrease in gluconeogenesis, probably secondary to decreased availability of pyruvate. These effects will result in a stoichiometric shift towards the formation of lactate.

Lactate measurement may be important to newborns with the fetal alcohol syndrome and metabolic acidosis.

References


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Chloride Interference with Use of an Iodide-Selective Electrode for Urinary I-

To the Editor:

In a recent study of urinary iodide excretion by subjects in Auckland, New Zealand, we evaluated an iodide-selective electrode (iodide electrode 94-53A with Digital Analyzer 701A; Orion Research Inc., Cambridge, MA 02139). We compared results with those obtained by a chemical method (1), using both aqueous standards (KI in distilled, de-ionized water) and a standard curve prepared by use of urine previously de-iodized with ion-exchange resin (Iobeads; Technicon Corp., Tarrytown, NY 10591).

Excellent results were obtained when the electrode was applied to aqueous iodide standards and the standard curve was linear to 1 × 10^-6 mol of I- per liter. In contrast, the results were highly inconsistent when the electrode was used with dilutions of iodide in urine, where linearity was only achievable to 1 × 10^-3 mol/L.

Subsequently, theoretical calculations were based on a modified Nernst equation (2):

\[ E = \frac{RT}{F} \log (a_{I^-} + K_{Cl^-} a_{Cl^-}^{\text{pex}} a_{Cl^-}) \]

where \( R \) is the gas constant, \( T \) the absolute temperature, \( F \) the Faraday constant, \( a_{I^-} \) the activity of iodide in solution, and \( K_{Cl^-} \) the selectivity coefficient of the electrode for iodide with respect to chloride (for the electrode in question, the manufacturer's booklet gives a value of 1 × 10^-6).

These calculations revealed that at ratios of \( a_{Cl^-}: a_{I^-} \geq 10^6 \) there is a significant and increasing error caused by chloride.

At average urine concentrations of chloride and iodide (for example, chloride 0.2 mol/L, iodide 1.8 × 10^-6 mol/L) the calculated chloride-induced error is 5%. This error rapidly increases with an increasing \( a_{Cl^-}: a_{I^-} \) ratio.

Because the physiological concentration of chloride in urine may range from 50 to 600 mmol/L, we conclude that, on the basis of chloride error, the iodide-selective electrode is unsuitable for the accurate experimental determination of iodide in urine.

References


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Glucose Method Comparison Criticized

To the Editor:

Your recent publication of a three-page scientific note (Clin. Chem. 28: 2405, 1982) comparing four different methods for determining glucose may be misleading to some of your readers. Two of the four methods are currently obsolete in the United States. Of the 6100 laboratories reporting glucose values in the 1982 comprehensive surveys of the College of American Pathologists (1), fewer than 20 reported glucose values assayed by the neocuprine or alkaline ferricyanide methods—a slight decrease from the 24 or fewer laboratories reporting each of these two methods in 1981. Therefore,