11.2: Acid-Base Physiology in Children

Most aspects of acid-base physiology in children are the same as for adults and will not be repeated here. Some differences in neonates and infants are briefly indicated below. The most common acid-base problems in neonates are respiratory disorders due to respiratory insufficiency.

Many inherited disorders affecting intermediary metabolism can result in an accumulation of organic acids and these nearly all present during childhood. These are briefly considered below.

11.2.1: General Factors affecting Acid-Base Balance in Infants

Low Bicarbonate depends on Gestational Age

As compared to normal adults, the plasma $[\text{HCO}_3^-]$ in neonates is lower due to the lower renal threshold and lower capacity to reabsorb bicarbonate. The more immature the neonate, the lower the level. Very low birth weight babies have bicarbonate levels of 12-16 mmoles/l but term babies have levels of 20-22 mmol/l.

Low Reserve to excrete an Acid Load

At birth in term infants, acid excretion is working near maximum capacity and there is little reserve to deal with acidosis. The lower bicarbonate levels in preterm babies means they have even less capacity than a term neonate to buffer an acid load. The ability to excrete an acid load improves over the first couple of months of life.
Other Factors

• Growth results in deposition of base in new bone as the calcium salts in bone are alkaline salts.

• On a weight basis, fixed acid production is higher than in adults (eg neonates and children < 12 months: fixed acid production is 2 to 3 mmol/kg/day).

11.2.2: Infantile Metabolic Acidosis

As mentioned previously, a large number of different inborn errors of metabolism cause a metabolic acidosis. This may be:

• organic acidosis (enzyme defect resulting in accumulation of acidic metabolic intermediates)

• lactic acidosis

• hyperchloreaemic acidosis

Feeding difficulties often in association with tachypnoea are common in neonatal metabolic acidosis.

Some examples of organic acidoses in children are:

• maple syrup urine disease

• methylmalonic acidaemia

• propionic acidaemia

• isovaleric acidaemia

• glutaric aciduria.

Some of these disorders also cause a ketoacidosis.

Typical Presentation

A typical presentation of many organic acidaemias is as recurrent episodes of metabolic acidosis with coma often preceded by vomiting, mental obtundation, hypotonia or seizures.

Episodes may be precipitated by increased protein breakdown associated with surgery.

These inherited conditions, though individually uncommon, should be considered in any child with an acidosis especially
if associated with coma. Neurological manifestations are common. Expert advice and investigation is required to sort out these disorders.

[The interested are referred to Ozand & Gascon (1991) for a review of organic acidaemias.]

Lactic acidosis can also result from enzyme defects and present during childhood. For example, pyruvate carboxylase deficiency, fructose-1,6-diphosphatase deficiency and pyruvate dehydrogenase deficiency. The lactic acidosis is not an isolated finding as these children have serious dysfunctions of organ systems esp affecting brain, liver and muscle.

Renal tubular acidosis may be hereditary and cause a hyperchloraemic acidosis in infants. Without treatment, growth retardation occurs in these children.

### 11.2.3: Other Acid-Base Disorders in Children

**Final points:**

- Insulin dependent diabetes mellitus usually presents during childhood or adolescence.
- Poisoning in children may cause an acid-base disorder and the disorder may be different from that typically seen in an adult (eg salicylate poisoning).