

Meconium Aspiration Syndrome

Mishra and Kumar(1), in their editorial on Meconium aspiration syndrome have been able to provide the readers with food for thought. Indeed, no other topic in the neonatal medicine has generated so much of enthusiasm and controversies as the aspiration of this seemingly innocuous substance, *i.e.*, the meconium. We too, have our contentions in this regard.

Firstly, the passage of meconium *in utero* is not uncommon even below 38 weeks of gestation. Recently, Narang *et al.*(2) showed an incidence of meconium staining in 1.9% preterm babies between the gestational ages of 33-36 weeks. In an ongoing study at our hospital, the incidence of meconium stained amniotic fluid was about 3.3% in preterms of 34-36 weeks.

We agree that the passage of meconium *in utero* is not always due to intrauterine asphyxia and it may be a purely physiological event due to increasing maturity of the fetus. However, a bacterial endotoxin has been recently implicated in "causing the preterm labor and passage of meconium *in utero*(3). It is to be emphasized that only those neonates who have respiratory distress with radiological evidence of aspiration pneumonitis in the presence of meconium stained liquor can be labelled as meconium aspiration syndrome (MAS). The authors have confused the readers by defining MAS as presence of meconium below the vocal cords. This condition is simply known as meconium aspiration and is a prelude to the development of MAS.

We do not agree with the authors' view that immediate airway management in form

of intubation and suction should be guided by the state of asphyxia in a neonate rather than the presence of meconium itself. Meconium aspiration, without intrauterine asphyxia may also result in significant morbidity because of the physical and chemically injurious properties of the meconium and risk of predisposition to infection. Mechanical obstruction by meconium may involve both the large and smaller airways which may be complete or incomplete giving rise to atelectasis, R-L shunt, hypoxia; ball valve obstruction, pneumomediastinum, pneumothorax and hypercapnia, respectively (4). Another dimension of the problem is that conventionally, asphyxia is defined either at one or five minutes only. If the management of meconium was to be guided by the state of asphyxia, then one would have to wait for at least one minute before attempting endotracheal suctioning, which will be too late. It may not always be possible to identify an infant with meconium stained liquor who should be subjected to endotracheal intubation and suction at birth. Therefore, the current protocol of oropharyngeal suctioning at the time of delivery of head followed by thorough tracheal suction to prevent meconium aspiration should be continued in all deliveries till confirmatory evidence to the contrary are available.

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Diabetes Mellitus in Infancy

Diabetes mellitus is not rare in childhood, but onset in infancy is not common and diagnosis may be missed. We encountered a boy 3 months and 7 days old, weighing 4.6 kg, who presented with fever and breathlessness for two days, without associated diarrhea or vomiting. There was no family history of diabetes mellitus. On examination he was stuporous with severe dehydration, tachycardia, tachypnea and acidotic breathing. He had abdominal distention with palpable liver (2 cm) and spleen (2 cm); respiratory examination revealed no foreign sounds. Level of consciousness deteriorated further within few hours and the child became comatose.

He had a total white cell count of 11,700/cu mm, blood glucose of 782 mg/dl and urine sugar 4+ with ketonuria. CSF examination revealed protein 70 mg/dl, glucose 374.0 mg/dl with 18 cells/HPF. The child was treated with intravenous fluids, plain insulin, and antibiotics. He developed tonic spasms after 3-4 hrs of starting insulin, so mannitol was added to combat cerebral edema. The child improved and was fully conscious within about 20 hrs of therapy. He had two episodes of hypoglycemia on 4th and 5th day of therapy with

blood glucose less than 50 mg/dl. Before discharge he was stabilized on mixture of plain and lente insulin.

Although rare under two years of age, childhood diabetes has been reported at the age of 9 days(1). Onset in infancy is always acute, presenting in coma or precoma. Early onset is reported to be associated with a high prevalence of diabetes in the family, predominance of males, an apparent increase in susceptibility to recurrent and severe episodes of hypoglycemia, particularly with infection and few episodes of ketoacidosis(2). The very high incidence of severe hypoglycemia is probably due to an inability to make small changes in insulin dose as needed and the child's inability to recognize and report early symptoms of hypoglycemia rather than abnormalities in glucagon counter regulation(2). A very tight control of blood glucose may, therefore, be undesirable during this age.

After initial control of symptoms with insulin, remission may occur for a few weeks to months before total insulin dependence is established. During this period, hypoglycemia can occur even on small doses. Treatment should be aimed at keeping the urine free of ketone bodies and mandatory blood sugars (self home glucose monitoring) frequently to avoid hypoglycemic reactions.