Is meconium aspiration an *in utero* event or does it occur with the first few postnatal breaths? Both mechanisms are possible(3) but their relative importance is yet to be determined. There are no satisfictory randomized controlled trials to provide answers to the multiple controversies regarding MSL and MAS. Based on the available literature we reiterate that intrapartum aspiration of meconium is important in the causation of MAS, though an unknown proportion of cases have *in utero* aspiration.

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Reply

"Meconium Aspiration" and not "Meconium Aspiration Syndrome (MAS)" is defined as the presence of meconium below vocal cords. This error which escaped our attention during correction is regretted. We entirely agree with Drs. Gautham and Narang that MAS is defined as the occurrence of respiratory distress soon after birth in the presence of meconium staining of liqour or staining of nails, umbilical cord or skin, along with radiological evidence of atelectasis or hyperinflation. It is specially relevant to clearly define these terms as only a small proportion of babies born through meconium stained liquor have evidence of meconium aspiration and only a small fraction of the latter develop respiratory distress along with radiological changes characteristic of MAS.

Regarding the pathophysiologic concepts of MAS, it has been correctly pointed out by Drs. Gautham and Narang that MAS is thought to result from a

combination of acute airway obstruction, chemical pneumonitis, alveolar edema, increased pulmonary vascular and airway resistance, changes in compliance and functional residual capacity and ventilation-perfusion abnormalities. However, the subject of controversy is whether deleterious effects of meconium are primarily responsible for MAS. Recent studies(l,2) have identified evidences of fetal distress and chorioamnionitis, along with thick meconium as important predictors of MAS. Hypoxia induced pulmonary changes hamper the normal mechanisms of clearing of the airway by the newborn. Presence of thick meconium may add to the problem. These pathophysiologic aspects have been reviewed and discussed at length by Katz *et al.(3)* recently. Animal experiments(4) also suggest that the extent of lung destruction in MAS is directly related to the length and degree of asphyxia and to aspiration of meconium. In this experiment, the trachea of guinea pig pups, who had either been exposed or unexposed to hypoxic insult, were injected with either amniotic fluid or meconium at birth. After 24 hours, extensive pulmonary necrosis was observed in previously asphyxiated guinea pig pups while the non-asphyxiated ones developed only mild parenchymal reaction. No difference was noted between amniotic fluid and meconium induced pathologic condition.

The paper by Gregory *et al.* has been wrongly quoted—The study referred to was by Under *et al* (5) who found no significant difference between the outcome of newborns born through meconium stained liquor and were either subjected to tracheal suction or not suctioned immediately after birth.

Till such a time that the existing 'grey areas' in the pathophysiologic concepts of MAS are cleared by future well designed studies, there will continue to be controversies in the immediate airway management of non-asphyxiated meconium stained newborns. Immediate oropharyngeal suction of the newborn is the accepted standard care for all deliveries irrespective of the fact whether liquor is meconium stained or not. The need for endotracheal intubation and suction in all meconium stained newborns remains controversial and is better dictated by the asphyxial state rather than the presence of meconium except when the meconium is thick.

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