minal portion of every plot (figs. 2 and 3) was linear for at least five half-lives. This suggests that the analytical technique used is specific for intact droperidol. In addition, the terminal half-lives in the same subject on different days and between subjects were very similar. Based on these data, one might expect a uniform response, from subject to subject, following drug administration.

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Neonatology

LECITHIN SYNTHESIS AND RDS The authors suggest that the respiratory distress syndrome (RDS) is a consequence of inadequate lecithin synthesis in the lining of pulmonary alveoli. This in turn leads to alveolar collapse on expiration. RDS occurs mainly in premature infants.

Two pathways for pulmonary lecithin production have been recognized. The first, present by the eighteenth to twentieth weeks of fetal life, is slow to mature and does not produce adequate lecithin until full term, at 35–37 weeks. This pathway is resistant to hypoxia, acidosis, and hypothermia. It incorporates cystidine diphosphatase choline and D-α,β,diglyceride to form a lecithin with α-palmitic/β-palmitic acids.

The second pathway to develop is active by the twenty-second to twenty-fourth weeks of fetal life and becomes the major source of pulmonary lecithin until 35–37 weeks. This pathway is sensitive to hypoxia, acidosis below *p*H 7.2, and hypothermia below 35 C. In this reaction, phosphatidyl ethanolamine is methylated to phosphatidyl dimethyl ethanolamine, which is further methylated to form a lecithin with α-palmitic/β-myristic acids. If this methylation pathway produces adequate lecithin, the premature infant will not develop RDS; if inadequate lecithin is produced, RDS appears. Analysis of β-carbon fatty acid from lecithin then shows little or no myristic acid. If improvement in RDS occurs, myristic acid will have increased, indicating increased synthesis of lecithin by the methylation pathway.

Pulmonary lecithin can be analyzed for amount and type by study of tracheal and pharyngeal aspirates for the β-carbon fatty acids. These aspirates, formed by lung effluents, contain lecithins identical to alveolar lecithins. The authors suggest that the course and prognosis of RDS in the premature infant can be followed by such analysis. (*Gluck, L., and others: Biochemical Development of Surface Activity in Mammalian Lung. IV. Pulmonary Lecithin Synthesis in the Human Fetus and Newborn and Etiology of the Respiratory Distress Syndrome, Pediatric Res. 6: 81–99, 1972.*)