

ceptors in the lung is greater than that of other organ cells. M. E. Avery (Montreal) studied the side-effects of corticosteroid administration upon the fetus. No abnormality was apparent during the postnatal growth of rabbits pretreated with glucocorticoids *in utero*. There was, however, a 12 per cent decrease in lung cell count in steroid-treated rabbits compared with their littermates, as determined by DNA and protein measurements. This decrease may indicate that accelerated differentiation during gestation is associated with a simultaneous decrease in multiplication. Clinically, administration of corticosteroids to human infants at the time of birth does not influence the incidence or time course of RDS.

G. C. Liggins (Auckland) has begun a clinical trial with glucocorticoids administered to parturients before premature delivery. In cer-

tain cases, delivery was delayed by the infusion of alcohol to allow the glucocorticoids time to affect the fetal lung. The results were impressive. When the infants were delivered two to seven days after the injection of glucocorticoids, there was a drastic decrease in the incidence and mortality of RDS among premature infants before 32 weeks of gestation. Extreme caution in undertaking such treatment, and a critical follow-up study of treated infants, were urged by the participants. As pointed out by Avery, injection of corticosteroids in the fetus may not be as innocuous as it appears.

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Literature Briefs

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Literature briefs were submitted by Drs. R. Clark, L. Cooperman, L. Cronau, B. Dalton, B. Das, A. Goldblatt, J. Harp, E. Lowenstein, L. Mark, H. Rackow, J. Reitan, and J. Ryan. Briefs appearing elsewhere in this issue are part of this column.

Circulation

CHEST X-RAY AND POISONING WITH INSECTICIDE The pulmonary radiologic findings in three cases of severe Parathion poisoning were shown to be remarkably similar. Fluffy perihilar infiltrates, present in the absence of vascular congestion and/or cardiomegaly, were noted to regress within hours of treatment with atropine and Protopam. All three patients were comatose or semicomatose, with copious secretions. The authors consider the radiographic findings diagnostic of pulmonary edema, although its nature (direct pulmonary capillary damage, left ventricular failure) is not defined. (*Bledsoe, F. H., and Seymour, E. Q.: Acute Pulmonary Edema Associated with Parathion Poisoning, Radiology 103: 53-56, 1972.*) EDITOR'S COMMENT: This article is of value because it describes one aspect of the clinical pattern seen to develop after organophosphate intoxication. Such descriptive exercises are of little further use

when important clinical data, including arterial blood gases, are not included. It is quite important whether the level of arterial oxygenation is or is not consistent with the extensive radiologic changes in the chest x-ray.

Metabolism

CYANATE EFFECTS ON HEMOGLOBIN Urea may be useful in treatment and prevention of painful vaso-occlusive crises that occur in patients with sickle-cell anemia. In the body, urea is in equilibrium with ammonia and cyanate. Cyanate reacts with an α -amino group of hemoglobin (carbamylation), thereby diminishing the sickling phenomenon *in vitro*. Addition of cyanate did not affect glycolysis, ATP, 2,3-DPG stability, autohemolysis, or osmotic fragility. Potassium loss was less than in controlled cells and pyruvate kinase activity decreased, but other glycolytic enzymes were normally active. Oxygen affinity was increased, but the Bohr effect was unaltered. The authors conclude that their studies provide further support for the potential clinical use of cyanate in treating and preventing the anemia and painful crises of sickle-cell disease. (*DeFuria, S. G., and others: The Effects of Cyanate in Vitro on Red Blood Cell Metabolism and Function in Sickle Cell Anemia, J. Clin. Invest. 51: 566, 1972.*)