

R. PELTONEN*, L. HIRVONEN* and T. PELTONEN. The Cardiorespiratory Research Unit and the Department of Obstetric and Gynecology, University of Turku, Turku, Finland. Cinerentgenological studies on the flow of the foetal lung liquid during the perinatal period.

Eight lambs delivered by caesarean section were studied before and during the first breath with Philips 5" image intensifier and an Arriflex cinematographic camera, rate 16-24 per minute. A saline filled condom was fitted on the snout of each lamb as it was brought out of the amniotic cavity, prior to exposure to air. Into the trachea was introduced a polyethylene tube in order to measure the pressure changes in the liquid-filled air passages before and during the aeration of the alveoli. Through the tube was injected 1 ml "Urografin 76 % Leiras". The movement of the contrast material indicated before the aeration a flow from trachea out into the pharynx and the foetus swallowed it. If the foetus was asphyctic, there was a profuse stream from the trachea. During the aeration of the alveoli the lung liquid remaining in the respiratory passages were absorbed into the pulmonary circulation.

The film demonstration.

J.F. DESJEUX*, Y.H. TAI* and PETER F. CURRAN* (Intr. by H. Lestradet). Dept. of Physiology Yale University, New Haven, Conn. 06510. EFFECT OF CHOLERA TOXIN ON Na TRANSPORT IN INTESTINE.

Massive secretion of fluid and electrolytes into the intestinal lumen is observed in cholera. This study was, therefore, undertaken to determine the role of the epithelial cells in the Na secretion observed in cholera. From measurements of flux as a function of electrical potential, the transcellular Na fluxes (Jc) were estimated, in vitro, on ileum treated with cholera toxin (CT) for 60 minutes and compared with Jc from unchallenged control (C) intestine from the same rabbit. In absence of electrochemical potential, in Ringer's solution, CT caused a net Na secretion ($-1.35 \pm 0.21 \mu\text{eq/hr cm}^2$), without statistical change in short circuit current. The net secretion appears to be due to a decrease of transcellular Na flux from mucosa to serosa (Jcms) ($-0.94 \pm 0.35 \mu\text{eq/hr cm}^2$) and an increase of cellular flux from serosa to mucosa (Jcsm) ($+0.75 \pm 0.64 \mu\text{eq/hr cm}^2$). The addition of 10mM glucose caused an increase ($p < 0.01$) of $J_{\text{Na}}^{\text{net}}$, Jsc and Jcms almost identical in C and CT intestine. However, Jcsm did not change significantly with glucose. These results indicate that CT caused an active cellular Na secretion which is most easily explained by postulating that CT stimulates an electrically neutral (Na+Anion) transport process from serosa to mucosa but does not change the "electrogenic" Na absorption.

V. M. Der Kaloustian, N. W. Wakid* and A. Murib*. Departments of Pediatrics and Clinical Pathology, American University of Beirut, Beirut-Lebanon.

The effect of intravenous administration of aminoacids in acrodermatitis enteropathica.

An 8 months old male child affected with acrodermatitis enteropathica was treated intravenously with the dialysate of an enzymatic hydrolysate of casein for three periods of 7-10 days each. This treatment consistently resulted in dramatic improvement of the child's general condition with complete healing of the skin lesions. The disease relapsed whenever the protein hydrolysate was discontinued. When the hydrolysate was replaced by mixtures of essential aminoacids no improvement was noted, even when all nine essential aminoacids (including histidine) were administered. Available evidence indicates that protein hydrolysates contain factor(s) that are effective in the treatment of acrodermatitis enteropathica. It is also suggested that this disease is not due to malabsorption of essential aminoacids through the intestine.

R.P.ZURBRUGG, A.BLUM*, P.DEYHLE*, T.SCHAFFNER*, S.BRUN DEL RE* and H.R.GRIEDER*. Children's Hospital Wildermeth Biene, Triemli Hospital Dep of Medicine and Dep of Medicine Cantonal Hospital University of Zürich, Institute for Pathological Anatomy University of Berne, CH. Denudation of basal membrane: an ultrastructural correlate of impaired membrane transport in protein losing gastroenteropathy (PLGE).

Gastropathia hypertrophica gigantea MENE-TRIER in PLGE has only been reported 14 x in children; its etiology is unknown. A 5 y old boy showed vomiting of unusually viscous gastric juice, severe anorexia during measles, generalized edema and hypoproteinemia of 3.0%. No evidence for chronic malnutrition, allergy, renal, cardiac or liver disease. 12.5% of the radioactivity(RA) of an iv dose of ^{51}Cr albumin appeared in a 72 h fecal collection (no $< 0.7\%$); 50.5% of RA appeared within 2 1/4 h in gastric juice with a protein output of ca 300 mg/h equivalent to an albumin loss of ca 8 g/d which exceeds the rate of albumin synthesis and thus explains hypoproteinemia. Pentagastrin iv adm. revealed a "sealing effect" for protein, Na, K, and Cl possibly of therapeutical importance. Electron microscopy revealed (1) numerous microvilli as a manifestation of increased resorptive processes and (2) a denudation of the basal membrane from their endothelial cells in the capillaries, thus manifold enlarging the inter-endothelial porus. This observation may well be of pathogenetic relevance.