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Alterations of the placental circulation and of the reactivity of the umbilical artery in experimental hypoxia and asphyxia

In the first series of experiments exteriorization of foetuses was carried out in near-term guinea pigs. The experimental groups of exteriorized foetuses were: 1.Hypoxic /N_2 in cap/, 2. asphyctic /temporary cord-clamping in utero/ and 3. controls. The spontaneous closure of the cord vessels, transplacental antipyrine transfer, acid-base balance and survival rate of the foetuses were followed. After induction of spontaneous breathing in the foetuses which remained connected with the placenta, the previousely closed vessels reopened and the placental circulation was restored under the effect of the N_2-breathing. In postasphyctic foetuses the placental circulation persisted in spite of the breating of atmospheric air, resulting in a favorable effect on the survival rate. In the second series of experiments the reactivity of isolated human umbilical artery and rabbit central ear artery was studied. The ultrafiltrates of the serum of hypoxic newborns exerted a smaller pressor effect on the human umbilical artery than on the rabbit ear vessel, while the corresponding effect of the normal serum ultrafiltrate was higher on the umbilical artery. In conclusion, a humoral action of the blood of hypoxic-asphyctic newborns is assummed, which tends to preserve the placental circulation.

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The effect of amiloride in H^+ secretion in the isolated amphibian skin.

Hydrogen ion secretion by epithelial structures can be mediated by a cation exchange mechanism as in the gills of some fish or by an 'active', pump-mediated specific process. Experiments were made to test the cation exchange mechanism in isolated amphibian skins by substitution of Na^+ by other ions in the bathing fluids, by inhibiting Na^+ influx with amiloride, or by stimulating it with Pitressin. Chambers of the Ussing type were used and the H^+ secretion rate was monitored by the pH stat method. In the frog skin, suppression of Na^+ influx by amiloride as well as its stimulation by Pitressin had no effect on H^+ secretion rates. Again, Na^+ substitution by Mg^{++} did not influence H^+ secretion. However, experiments done in toad skin showed that H^+ secretion increased after stimulation with Pitressin and decreased or sometimes was suppressed when amiloride was used or when Na^+ was substituted, suggesting that in this last preparation, some part of the total H^+ secretion is linked to Na^+ influx. In the isolated frog skin this mechanism seems not to be operative.

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Alteration of membrane proteins as a possible cause of stomatocytosis.

Stomatocytosis is a rare inborn hemolytic anemia, which is characterized by a typical shape of the erythrocytes, high intracellular $Na^{\pm}$ and low $K^{\pm}$ values and increased rates transport of Na^+ and K^+ through the membrane. This can be demonstrated by a high activity of the membrane-bound Na^+, K^+-ATPase. A membrane defect has been discussed as a cause of this disease for a long time. In a case of a 7-year-old boy the EDTA-extractable erythrocyte proteins were separated on a two-dimensional electrophoresis in polyacrylamide gel. The separation in SDS dependent on the molecular weight was combined with the isoelectric focussing in urea. In this system distinctive differences between normal erythrocytes and stomatocytes can be demonstrated. Hence we conclude that the most probable explanation of the structural and metabolic changes in stomatocytosis is that of specific alterations of the membrane proteins.

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Studies on active and passive cation fluxes in congenital hemolytic anemia with high sodium and low potassium erythrocytes.

A six year old girl suffering from severe nonspherocytic hemolytic anemia which was characterized by stomatocytosis, macrocytosis, and autohemolysis type I was found to have erythrocytes with reduced potassium (16 mval/l) and elevated sodium (95 mval/l) concentrations. Glykolysis, ATP formation and ATP utilization of the cells were markedly increased. Active sodium outflux and potassium influx were increased ten- and six fold, respectively. Inhibition of the cation pump by ouabain decreased the active cation fluxes for about 60 %. Passive sodium and potassium fluxes were likewise markedly increased. Normal red cells reconstituted with intracellular cation concentrations which were similar to the concentrations of the abnormal cells showed only a slight increase of active cation fluxes. The results suggest a defect of passive cation permeability of the abnormal cells.