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Adrenergic receptor functions in the urinary bladder and its outlet region.

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Autonomic nerve impulses are conveyed to the smooth musculature of the lower urinary tract by transmittor substances acting on muscle receptors. These impulses have partly or completely ceased in children with congenital neurogenic bladder disturbances. In order to evaluate the significance of a defect sympathetic innervation for collection of urine and micturition the adrenergic receptor functions have been investigated. Alpha-adrenergic receptor function was found in the outlet region of the bladder, i.e. bladder base, bladder neck and proximal urethra, of man and cat. Stimulation of this type of receptor leads to a contraction of the internal sphincter of the bladder. Beta-adrenergic receptor function could be identified in both the corpus-fundus region and in the outlet region. Low concentrations of beta-adrenergic agonists relaxed the internal sphincter as did adrenaline and noradrenaline in low concentrations. Higher concentrations of these cathecolamines contracted the same region. In children with myelomeningocele the adrenergic receptor functions could be activated by local application of drugs in the bladder.

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Alterations of the fetal ECG as an early indication of intra-uterine asphyxia.

During the study of the cerebral reactions of the fetal lamb to asphyxia the fetal ECG was found to react to asphyxia with elevation of the ST-segment and high and peaked T-waves. These alterations of the ECG occurred earlier than the typical changes in heart rate - bradycardia, dropped beats, A-V block. The ECG changes were well correlated to the cerebral reactions in terms of the somatosensory evoked EEG responses. To evaluate the relations between the ECG changes and hemodynamic variables ten experiments were conducted on mature lamb fetuses. In acutely exteriorized preparations ECG, arterial blood pressure, left ventricular pressure, myocardial contractility (dP/dt), heart rate and systemic blood flow (thermodilution) were measured and the fetus was subjected to varying degrees of hypoxia and hypercarbia. A close relation between the ECG changes and the total systemic blood flow and the myocardial contractility was obtained. Arterial blood pressure and heart rate deteriorated at a later stage and at more severe degrees of hypoxia than the ECG. It is concluded that the ECG pattern conveys information on hypoxia before typical effects of hypoxia on the heart rate appear in the fetal lamb.

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The biliary bilirubin excretion pattern and beta-glucuronidase activity in duodenal bile of newborn infants with hyperbilirubinemia treated with phototherapy.

Experiments with Gunn rats have given evidence that phototherapy brings about an increased biliary excretion of bilirubin in the unconjugated form. The concentration of total and unconjugated bilirubin was measured in duodenal bile in 15 newborn infants with hyperbilirubinemia. 10 infants received phototherapy for 24 hours between two bile samples, while 5 infants had no treatment and served as controls. Phototherapy significantly altered the biliary bilirubin excretion pattern, more bilirubin being excreted as unconjugated bilirubin.

In order to test the hypothesis, that the observed change in the conjugation of bilirubin could be caused by an activation of liver- or duodenal beta-glucuronidase by phototherapy, the activity of this enzyme was measured in duodenal bile in 10 newborn infants, of which 5 were treated with phototherapy and 5 had no treatment. No significant alteration in the activity of beta-glucuronidase was observed during phototherapy. The change in the biliary bilirubin excretion pattern may be explained as secondary to a metabolic effect of the photooxidation products or to the formation of bilirubin adducts

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Evaluation of bilirubin cytotoxicity and phototherapy in baby Gunn rats by histochemical enzyme marking of Purkinje cells.

During the first few days of life homozygous Gunn rats are especially threatened by bilirubin toxicity. However, in this early period neurological signs and behavior do not indicate kernicterus clearly. Likewise, histological preparations stained by conventional procedures have failed to show the full extent of the nerve cell damage before 10 days after birth. Previous studies of our group have shown that histochemical demonstration of reduced activity of oxydative enzymes can be used as a sensitive detector for bilirubin encephalopathy. This technique has now successfully been applied to 4 - 8 day Gunn rats. By rating the number of Purkinje cells according to their enzyme activity, we have been able to document the increased neurotoxicity induced by sulfonamides (300-600 mg per kg sulfadimethoxine) or other drugs competing with bilirubin for the binding sites of plasma albumin, and the protective effect of phototherapy (Westinghouse F 20 T 12 BB special blue lamps, 400-500 nm: 3400 µ Watt per cm^2 at animal level, 4 days). The survival rate decreased or increased from 88 % (controls) to 0 or 0 to ~ 50 %, respectively, in the drug injected or drug injected plus illuminated animals.