108 IGE-MEDIATED LATE PHASE REACTIONS - THE PATHOPHYSIOLOGIC BACKGROUND OF CHRONIC BRONCHIAL ASTHMA?

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Late phase reactions (LPR) have been considered generally as type III allergy according to the classification of Coombs and Gell. IgE-antibodies, however, are able to initiate not only immediate, but also late cutaneous reactions (LCR). Using the skin blister technique we could show LCR to be mediated by inflammatory mediators generated during the preceeding immediate reaction. Various mediators as histamine, active kallikrein, platelet activating properties, leukotrienes and thromboxanes were demonstrable in skin blister fluids their concentration being in part related to the time course of the skin reactions. LCRs were diminished by eikosatetraynoic acid (not by indomethacin), by dazoxiben as well as by ethanolic onion extract. Betamimetics as salbutamol and anticholinergics as ipratropiumbromide could reverse late bronchial reactions (LBR), ethanolic onion extract could prevent LBR. Other authors found prostaglandin D2 and chemotactic substances during LPR as well as protective effects of corticosteroids, disodium cromoglicicum, tranexamic acid and combined histamine H1/H2 receptor antagonists.

Two types of LPR have to be distinguished: type I and type III. The further is initiated by a IgE-dependent mast cell degranulation and maintained by a complex inter action of mediators and cells. It represents the pathophysiologic correlate of long lasting bronchial asthma.

109 DEFICIENCY OF ALPHA-1-ANTICHYMOTRYPSIN IN ASTHMATIC CHILDREN.

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Deficiency of protease inhibitors (PI) may cause chronic lung disease (e.g. alpha-1-antitrypsin deficiency). Inhaled proteases have been shown to induce bronchoconstriction. We therefore determined serum concentrations of Cl-inactivator (Cl Ina), alpha-2-macroglobulin (a2 M), inter-alpha-trypsin inhibitor (IaI), alpha-1-antitrypsin (a1 AT) and alpha-1-antichymotrypsin (a1 Achy) in 138 children with bronchial asthma and 50 healthy controls, using radial immunodiffusion technique. The asthmatics were grouped according to the predominant precipitating factor (allergen, infection) and subdivided according to the degree of clinical severity. In each group children without signs of obstruction had lower levels of a1 Achy than controls ($p < 0.01$). This difference was masked during allergen induced symptoms. With infection, elevated levels for a1 Achy and a1 AT ($p < 0.01$) and decreased levels for IaI ($p < 0.05$) were obtained, whereas Cl Ina and a2 M remained unchanged. Recently a1 Achy has been demonstrated to be of major importance in the immediate and specific defence of lung tissue against chymotrypsin like proteases, which are released by polymorphonuclear leucocytes and mast cells. Deficiency of a1 Achy may lead to inadequate control of bronchial inflammation and this may contribute to bronchial hyperreactivity.

110 MAJOR DETERMINANTS OF THE FIRST BREATH

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Little information is available relating to the first breath. Previous reports have provided evidence that at birth the "typical" infant will generate a negative pressure of approximately 30 cmH_2O, and inspire 40 ml. of air; 17 ml. of this will form a functional residual capacity (F.R.C.) after a sustained positive pressure has been created in expiration. Using a mask/pneumotachograph/differential pressure transducer and a dual micropressure transducer, we have been able to obtain simultaneous data on volume achieved, intrathoracic pressure and intra-abdominal pressure changes during the first breath. From data in 16 babies in which the position of pressure recordings could be confirmed, we have found: 1. Volume changes in line with previous reports. 2. Larger pressure changes ($p < .05$) than in previous studies. 3. A positive and significant correlation between both inspiratory volume and the duration of inspiratory pressure with the volume of the F.R.C.

111 THE EFFECTS OF SURFACE TENSION ON LUNG FUNCTION DURING EARLY DEVELOPMENT.

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Pressure-volume and lung compliance/kg determinations were made in a total of 45 preterm lambs during gas ventilation (GR I: n = 31; 122-140 days gestation; 3.3-3.9 kg birthweight and liquid fluorocarbon (RIMAR 101) ventilation (GR II: n = 14; 106-138 days gestation; 1.1-3.4 kg birthweight. After effective ventilation (PaO_2 > 60mmHg; $PaCO_2$ = 30-45mmHg) and acid-base status (pH = 7.25 - 7.45) were achieved, pulmonary function was evaluated. Regression analysis showed significantly different relationships between lung compliance and developmental age for GR I ($r = 0.95$) and GR II ($r = 0.99$). Compliance for GR II lambs was consistently greater than for GR I lambs, this difference did not become significant until after 125 days gestation. These results indicate that minimizing interfacial surface tension has little effect on lung compliance in animals at earlier gestational ages, therefore suggesting that morphologic and tissue properties predominate surface properties in limiting lung distensibility. By defining the stage of development in which pulmonary function is most effectively improved by reduced surface tension, these findings suggest age-related criteria regarding the benefit of exogenous surfactant replacement therapy. (Supported by NIH Grant HL/HD 30525).

112 ASSESSMENT OF RESPIRATORY CONTROL IN INFANTS

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The hypothesis that the primary defect in infants dying of the Sudden Infant Death Syndrome (SIDS) lies in the control of ventilation has provoked much interest. We have used the measurement of occlusion pressure as an index of the neuromechanical output of the respiratory system to investigate the response of infants to hyperoxic hypercapnia All measurements were made on unsedated infants during quiet sleep as defined by EEG and EOG recordings. Infants breathed through a facemask and pneumotachograph with a shutter attachment. Progressive hypercapnia was achieved by using a rebreathing bag with an initial pCO_2 at 38 mmHg. The shutter was activated every 20-30 seconds and airway pressure was recorded from the facemask, until either the infant aroused or the pCO_2 reached 65 mmHg. Pressure was measured 100 msec following the onset of inspiratory effort ($P_{0.1}$). The slope of $P_{0.1}$ against pCO_2 was plotted for each subject.

Seventeen infants below the age of 20 weeks (4 'near-misses' for SIDS, 5 siblings of SIDS, 8 normal infants) were studied. The slope of $P_{0.1}$ against pCO_2 in the normal infants ranged from 0.08 to 0.47 $cmH_2O.mmHg^{-1}$ and the $P_{0.1}$ at 55 mmHg ranged from 4.05 to 15.5 cmH_2O. The values from the other groups of infants, who might be thought to be at increased risk for SIDS, all lay within these normal ranges.

In conclusion, no significant differences were found between the three study groups under these defined circumstances.

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Withdrawn