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CEREBRAL AUTOREGULATION IN PRETERM INFANTS. Margot van de Bor, Frans J. Walther, and Maureen E. Sims. University of Southern California, School of Medicine. Dept. of Pediatrics, Los Angeles, California, USA.

Cerebral autoregulation has been demonstrated in newborn animals. We studied the relationship between mean arterial pressure (MAP) and cerebral blood flow velocity (CBFV) in 90 stable preterm infants ≤ 32 weeks gestation. Mean (SD) gestational age was 28.9 (1.9) wks, birth weight 1,128 (230) grams, 5 min Apgar score 7.4 (0.9), and initial pH 7.36 (.08). All infants had been normoxic and normocarbic from birth onwards. Intracranial hemorrhage and patent ductus arteriosus were excluded. CBFV was determined at MAPs ranging from 27-50 mm Hg. Using 2-D/ Doppler ultrasound, we obtained CBFV waveforms from the pericallosal (PCA), internal carotid (ICA), and basilar (BA) arteries between 12-72 hours of age. Peak systolic, end diastolic, and mean (MFV) flow velocities were derived from 3 consecutive cardiac cycles.

A linear relationship between MAP and MFV was found in all 3 arteries: $r=.76$ in the PCA, $r=.73$ in the ICA, and $r=.70$ in the BA ($p<.001$). Multiple regression analysis showed that the MFV was solely determined by MAP and not by $PaCO_2$, PaO_2 , heart rate or mean airway pressure.

We conclude that between 27-50 mm Hg MAP a linear relationship between MAP and MFV exists in 3 major intracranial arteries. These data raise questions about the ability of the very preterm infant to regulate cerebral blood flow.

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DYSFIBRINOGENEMIA IN BURNED CHILDREN. Domenico Del Principe, Adriana Menichelli, Massimo Giordani, Carlo M. Colucci, Claudio Colistra, Stefano Di Giulio. Dept. of Public Health, University "Tor Vergata", Rome, Italy.

Severely burned children often show a dysfunction of microcirculation, due to overproduction of O_2 radicals and to the activation of blood coagulation. We studied the effect of H_2O_2 on fibrinogen collected from burned children. Controls were healthy adult donors. Fibrinogen was purified through precipitation with PEG and saltin-out procedure. The thrombin-induced transformation of fibrinogen to fibrin, as assayed by recording the polymerization curves, was faster in the patient native fibrinogen than in controls ($n=18$, $p<.001$). The oxidation of control fibrinogen with H_2O_2 caused a decrease of the structurally reactive protein (measured by a nephelometric method) and increased the velocity of fibrinogen-fibrin transformation. It was also associated to a decrease of tryptophane residues, as assayed by the spectral changes at 282 nm. At the same experimental conditions, similar changes were not observed in patient fibrinogen. Patient fibrinogen showed a decreased number of amino acid residues reactive to the "in vitro" oxidation. Our data demonstrate the existence of some structural differences between patient and normal fibrinogen, that may be relevant in the pathophysiologic events following burns.

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OXYGEN RADICALS PLAY A ROLE IN THE PATHOGENESIS OF ANAPHYLACTOID PURPURA (AP). Adriana Menichelli, Giovanna Mancuso, Claudio Colistra, Walter De Matteis, Domenico A. Rosati, Daniela Orsini, Domenico del Principe. Dept. of Public Health, University "Tor Vergata", Rome, Italy.

Ceruloplasmin (CPM) was measured by an immunologic method in sera of children with AP during the acute phase of the disease, and the values were compared to that estimated by measuring serum ferroxidase activity. Controls were healthy adults. In agreement with previous observations, more copper-containing protein was present in the serum of AP patients than in control serum (43.4 ± 12 vs 31.7 ± 7.3 $n=12$, $p=0.016$ by Student's t test). In control sera the same amount of CPM, measured either by immunologic or enzymatic method, was detected. CPM values of AP patients, assayed as ferroxidase activity, were, instead, significantly lower than it would be expected on the basis of CPM protein found ($p<.001$ by Student's paired t test). However values were well correlated with the amount of CPM immunologically determined ($r=0.971$, $p<.001$) suggesting for the presence of an abnormal protein. Our results indicate a critical role for oxidizing species in the pathogenesis of AP, since the discrepancy between the protein amount and its enzyme activity is a marker of "in vivo" presence of oxygen radicals.

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Hemorheology and hemostasis in nephrotic syndrome

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Increased aggregation and decreased deformation of red blood cells (RBC) may cause thromboembolic events.

Patients and methods: RBC aggregation (Myrenne

Aggregometer), RBC deformability (rheoscope), RBC surface charge (partition), plasma viscosity and several plasma proteins (radial immunodiffusion) were studied in 28 children with nephrotic syndrome (age 3-22 years) and in a control group.

Results: The children with nephrotic syndrome were divided into two groups according to the extent of proteinuria (*, $P<.05$):

Proteinuria (mg/m ² /d)	>100	<100	Controls
RBC aggregation	12.3 $\pm$ 2.9 *	7.3 $\pm$ 1.6 *	4.8 $\pm$ 0.9
Total plasma protein (g/l)	50.3 $\pm$ 11.4 *	68.0 $\pm$ 5.8 *	74.8 $\pm$ 5.6
Fibrinogen (g/l)	7.7 $\pm$ 1.8 *	4.2 $\pm$ 0.3 *	3.8 $\pm$ 0.4
α_2 -Macroglobulin (g/l)	7.0 $\pm$ 1.9 *	3.4 $\pm$ 1.0 *	2.1 $\pm$ 0.4
Albumin (g/l)	24.9 $\pm$ 11.0 *	40.5 $\pm$ 5.9	42.4 $\pm$ 3.1

No significant difference was observed in plasma viscosity, RBC surface charge, IgM and RBC deformability.

Conclusions: Nephrotic syndrome with marked proteinuria is associated with increased RBC aggregation due to increased concentrations of plasma macroglobulins. RBC charge does not contribute to increased RBC aggregation in these children. Increased RBC aggregation together with abnormal clotting factors may increase the risk of thromboembolic events in these children.