176 EARLY INVASIVE BACTERIAL INFECTION AFTER ASPIRATION AND ACUTE RESPIRATORY FAILURE. Robert K. Kanter, Leonard B. Weiner, Gloria A. Albarelli, Joy M.Tompkins, (Spon. by Roger E. Spitzer). SUNY Health Science Center, Department of Pediatrics, Syracuse, N.Y.

This study was performed to determine the clinical importance, incidence, and early identifying features of infection occuring in children in the first 48 hr after severe acute lung injury from aspiration. Charts from the past 6 yrs. were reviewed for all children at high risk for aspiration of pharyngeal secretions, requiring mechanical ventilation. (Drowning (13), aspirated foreign body (5), known aspiration of gastric contents (2), hydrocarbon aspiration (1)). Criteria for inclusion included survival >24 hr, and a blood culture obtained in the first 48 hr. Invasive infection was identified by positive blood culture. Possible infection was defined as: T>38.5°C, blood white cell count >10,000 or <5,000, and a potential pathogen in cultures of tracheal secretions. Of 21 high risk patients, 7(33%) had infections in the first 48 hr after aspiration. Five had invasive infection (S. pneumoniae(4), P. cepacia (1)). Two possible infections occurred (S. pneumoniae, E. aerogenes). One child died in septic shock. Older age was associated with infection (mean 60 and 21 mo in infected and noninfected patients, respectively; p<.05). Other variables had no predictive value: immune deficiency, cardiac arrest, neutrophil count and trend, arterial to alveolar pO_2 ratio and trend, antibiotic usage, assymetry on chest X-ray, and temperature . These observations are consistent with experimental data indicating the potential for invasive infection when bacteria are innoculated into severely injured lung. Since infection is difficult to predict in this high risk group, consideration should be given to immediate initiation of antibiotic therapy, and aggressive bacteriologic surveillance.

●177 REPERFUSION INDUCED FREE RADICAL FORMATION FOLLOWING GLOBAL ISCHEMIA. Jeffrey R. Kirsch, Anne M. Phelan, David G. Lange, Richard J. Traystman. (Spon. by Mark C. Rogers). Johns Hopkins Medical Institutions. Department of Anesthesiology/Critical Care Medicine. Baltimore, MD.

Although O_2 derived free radical mechanisms have been implicated to cause cerebral damage associated with ischemia, free radicals have not been measured in this situation. In this study the production of free radicals was evaluated in rats exposed to global ischemia and reperfusion. Male Sprague Dawley rats pretreated with the spin trap phenyl-t-butyl nitrone (1.5 ml, 0.1 M; IP) were anesthetized with sodium pentobarbital (65 mg/kg). After tracheostomy, ventilation was controlled to maintain $PaCO_2$ 35-40 mmHg and supplemental O_2 was administered to achieve a PO_2 of 100-200 mmHg. Jugular vein and tail artery catheters were placed and the great vessels were exposed through a median sternotomy. After paralysis (succinylcholine, 4 mg/kg; iv) and anticoagulation (heparin 150 U/kg; iv) one group of animals was designated as sham operated controls and another made ischemic by cross clamping the ascending aorta and inferior vena cava. At the end of 8 min clamps were released and systemic acidosis was treated with sodium bicarbonate (3 mEq/kg). Immediately after ischemia or 15 min after reperfusion the animals were killed and brains removed for lipid extraction and detection of free radicals by electron spin resonance. Our spectra are consistent with large accumulation of free radicals in animals exposed to ischemia and not in sham operated controls (500% increase) or non-reperfused animals. We conclude that free radicals are generated by the brain in response to 8 min of global ischemia and reperfusion. Supported by 20020 & HL 32134.

●178 BROAD SPECTRUM ANTI-PLATELET AGGREGATION THERAPY IMPROVES POSTISCHEMIC CEREBRAL BLOOD FLOW (CBF) AND CORTICAL SOMATOSENSORY EVOKED RESPONSE (CSER) RECOVERY, BUT FAILS TO BLOCK PLATELET ACCUMULATION IN THE DAMAGED HEMISPHERE P Kochanek, A Dutka, J Hallenbeck, Naval Medical Res Inst, Bethesda, MD Children's Hospital Washington, DC (Spon, B Fuhrman)

Previously we showed that platelets accumulate in areas of low CBF 4h after brain ischemia. Similarly, CSER and CBF recovery after brain ischemia was enhanced in dogs treated with either prostacyclin (PGI2) + indomethacin or the platelet-activating factor (PAF) receptor antagonist kadsurenone. Since these treatments inhibit platelet aggregation, we hypothesized that their beneficial effects were mediated via inhibition of platelet accumulation. To test this hypothesis, 22 dogs with 111In-labeled platelets were subjected to 1h of ischemia of the right hemisphere by air embolism. CSER in the right hemisphere was monitored during reperfusion, while CBF (autoradiography) and platelet accumulation (R-L ratio of 111In activity) were measured after 4h of reperfusion. 12 dogs served as controls, 6 were treated with PGI2 + indomethacin and 4 with kadsurenone.

Both treatments enhanced CSER recovery after 1h of reperfusion and eliminated neuron-disabling CBF at 4h (defined as <15ml/100g/min in gray matter and <6ml/100g in white). Despite this, platelet accumulation was not blocked by either treatment.

Group	CSER Amplitude Recovery	Platelet Accumulation R/L Ratio	Neuron Disabling CBF
Control	15.5 ± 2.8	1.5 ± 0.1	50%
PGI2 + Indo	27.1 ± 4.7	1.7 ± 0.3	0
Kadsurenone	36.0 ± 2.9	1.5 ± 0.2	0

Anti-platelet aggregation therapy does not block postischemic platelet accumulation in the brain. Other effects of these treatments including improved CBF may contribute to enhanced recovery.

179 EFFECT OF PHOSPHATE (P) CONCENTRATION ON IONIZED CALCIUM (iCa) CONCENTRATION IN VITRO Mary Lehmann, Francis Mimouni, Reginald C Tsang. Univ of Cincinnati Department of Pediatrics, Cincinnati, OH.

From previous studies measuring ultrafilterable Ca, it was felt that increasing P concentration would lead to a decrease in iCa, due to P binding to iCa. Since no specific studies measured directly iCa as a function of P, we examined the effect of P on iCa concentration in vitro. We hypothesized that within the range of clinically encountered pH an increase in serum P would lead to a decrease in iCa. A single donor serum was aliquoted into 25 samples stored in CO_2 sealed tubes, and divided into 5 subsets of 5 tubes. pH was altered in 4 of the 5 subsets by adding various concentration of HCL or of NaOH. The pH's studied were: 7.09, 7.28, 7.35, 7.51, and 7.63. P concentration was altered in each subset by adding various concentrations of a P buffer with a pH of 7.4. The P concentrations studied were 2.5, 4.6, 6.8, 8.3, 11.1 mg/dl. iCa was measured using a Radiometer iCa 1 ion-selective electrode (standard error of measurement: 0.04 mg/dl). There was an inverse relationship between iCa and P which was similar at all pH's studied (r ranging from -0.84 to -0.94, p<0.001). The slopes did not differ significantly from each other, indicating no deviation from parallelism of these 5 regression lines. iCa concentration correlated inversely with pH (p<0.001). There was no significant interaction between P concentration and pH. Therefore iCa concentration in vitro inversely correlates with pH and P concentration. We suggest that in addition to factors well known to influence iCa concentration (such as protein, bicarbonate and pH), serum P concentration also plays a significant role.

180 STEROIDS IMPROVE SURVIVAL IN EXPERIMENTAL NEONATAL SEPTIC SHOCK. Thom E Lobe, Dennis Gore, Cameron Mantor, M. Pamela Griffin, James C. Hilton, Peter Mancillas, Daniel L. Traber, David N. Herndon. The Shriners Burns Institute, Galveston, TX 77550

Neonatal septic shock from peritonitis is often fatal. To evaluate the efficacy of steroids in its resuscitation, anesthetized, neonatal pigs were subjected to fecal-E.coli peritonitis-induced septic shock by intraperitoneal inoculation. All animals received gentamicin and 5% albumin in lactated Ringer's solution at 10 cc/kg/hr. Metabolic acidosis was corrected as required, and ventilatory support was administered. Pigs were randomly divided into three groups. Group I, control animals, did not receive steroids. Groups II and III received methylprednisolone (30 mg/kg,IV) every 6 hours beginning when cardiac output dropped or one hour later, respectively. Microspheres were used to assess regional perfusion. Hemodynamics and regional blood flow were not significantly different by ANOVA. Pigs "survived" if hemodynamics were stable at 24 hrs, compared to baseline. Survival data are shown below:

Group	(n)	Mean Survival (SEM) In Hours	% Survivors
I	8	6.8 (1.0)	0
II	8	18.9 (2.4)*	38%
III	8	16.6 (2.2)*	25%

* p<0.002 compared with Group I (Student's "t" test)

Steroids exert a beneficial influence on survival in this experimental neonatal shock model.

181 A REEVALUATION OF SERUM IONIZED CALCIUM CONCENTRATIONS IN THE NEONATE. Jeffrey L. Loughead, Francis Mimouni, Reginald C. Tsang. University of Cincinnati, Dept. of Peds., Cincinnati, OH.

Neonatology textbooks define neonatal hypocalcemia (NHC) as a serum total calcium (Ca) concentration of <7.0 to <8.0 mg/dl, or a serum ionized Ca (iCa) concentration of <2.5 to 4.0 mg/dl. Studies leading to these figures used pooled data over the first few days of life, when dramatic changes in serum Ca occur, and when iCa was measured with ion-specific electrodes with much less accuracy & reliability than those used today. Studies in adults showed that newer electrodes yield higher results than older electrodes. We hypothesized that the normal range for iCa concentration in neonate would be higher than the published figures even at the nadir at 24 hours of age. The present study examined serum Ca (atomic absorption) & iCa (radiometer iCa electrode) concentrations in 30 full term infants born following uncomplicated pregnancies, labors & deliveries in order to determine the lowest normal newborn range. The serum Ca concentration averaged 9.0 mg/dl (2.3 mmol/l) with a 95% confidence limit of 7.8 to 10.2 mg/dl (2.0 to 2.6 mmol/l). The serum iCa concentration averaged 4.9 mg/dl (1.2 mmol/l), with a 95% confidence limit of 4.4 to 5.4 mg/dl (1.1 to 1.4 mmol/l). The range of serum total Ca concentrations fell within previously published ranges but the serum iCa concentration was markedly greater than previously believed. There was a significant correlation between Ca & iCa in serum (r = 0.71, p<0.001) but the predictive value of iCa concentration from total Ca concentration was clinically unacceptable. Because of the many factors which influence serum total Ca measurements & not the ionized fraction, and since iCa is the biologically active fraction of serum Ca, we propose the use of a serum iCa concentration of <4.4 mg/dl (1.1 mmol/l) to define the population at risk for NHC.