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THE QT INTERVAL IN ABORTED SIDS INFANTS. Gabriel G. Haddad, Mary A.F. Epstein, Ralph A. Epstein, Norman M. Mazza and Robert B. Mellins. College of Physicians & Surgeons and Sch. of Engineering, Columbia University, New York.

Prolongation of the QT interval has been implicated in the pathogenesis of the Sudden Infant Death Syndrome (SIDS). To test this hypothesis, 31 studies in 12 normal infants and 17 studies in 7 aborted SIDS infants were performed during sleep in the first four months of life. The QT index (QTc) was measured with an accuracy of 2 msec or better. In contrast to recent suggestions, the mean QTc was significantly smaller in the aborted SIDS than in the normal infants in both REM and quiet sleep ($p < 0.05$, unpaired t-test). Means $\pm$ SD are presented:

	NORMAL	ABORTED SIDS
REM	0.436 $\pm$ 0.015	0.416 $\pm$ 0.017
QUIET	0.443 $\pm$ 0.017	0.422 $\pm$ 0.022

In both the normal and aborted SIDS infants, the mean QTc was significantly smaller in REM than in quiet sleep ($p < 0.01$, paired t-test). We suggest that 1) the smaller QTc in the aborted SIDS infants results from a uniform increase in the activity of the sympathetic outflow to the ventricles or an increase in circulating levels of catecholamines and 2) the smaller QTc in REM in both groups makes it unlikely that there is an imbalance in the sympathetic innervation of the ventricles in aborted SIDS. Although these studies do not support the hypothesis relating SIDS to prolongation of the QT interval, they may provide a basis for distinguishing normal from aborted SIDS infants.

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MECHANISM OF FETAL VASOPRESSIN HYPERSECRETION DURING THE PERINATAL PERIOD. A.J. Hadeed, R.D. Leake, R.E. Weitzman, D.A. Fisher, Department of Pediatrics, UCLA School of Medicine, Harbor General Hospital, Torrance, California.

Cord blood levels of arginine vasopressin(AVP)are known to be high after vaginal delivery(VD)in contrast to ceasarian section (C/S)delivery without labor. However, the mechanism, significance, and postnatal duration of the AVP hypersecretion are not known. We studied serial values for AVP concentration by radioimmunoassay in 61 full term and 13 premature (32-36 weeks gestation) infants during the first 24 hours of life. All normal infants had Apgar values >7. Mean AVP values vs time did not differ for premature and full term infants. Paired (mean $\pm$ SEM, uU/ml) umbilical artery(UA)and vein(UV)levels following VD and C/S during labor were 90 $\pm$ 15 and 15 $\pm$ 5.6 respectively($p < .001$). Cord UA values for 2 anencephalic infants, 2 infants with breech presentation, and 9 infants delivered by C/S without labor, were significantly lower than values after normal VD and C/S with labor. Plasma AVP after VD or C/S with labor fell rapidly after delivery; peripheral venous levels in VD infants were 22% of UV values by 30 minutes ($p < .05$). By 2 hours AVP values for all infants approached adult basal levels.

Conclusion: a)Cord blood AVP is of fetal origin since UA>UV, and anencephalic AVP levels are low, b)Premature infants have an intact AVP secretory mechanism, c)Anoxia is not the stimulus for the fetal AVP surge in labor, d)Cerebral compression from VD may be the mechanism of AVP hypersecretion.

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OPTIMAL TIME OF STARTING CONTINUOUS POSITIVE AIRWAY PRESSURE (CPAP) IN THE TREATMENT OF RESPIRATORY DISTRESS SYNDROME (RDS). Thomas Hegyi & I. Mark Hiatt (Sponsored by W.C. Heird) Department of Pediatrics, Monmouth Medical Center, Long Branch, New Jersey

No significant differences in response to treatment were found between two groups of equally ill premature infants treated with early or late nasal CPAP. RDS was judged to be of equal severity in both groups utilizing $AaDO_2$ & PO_2 measurements during the first two hours of life. Ten infants, with a mean birth weight of 1800gms were started on early CPAP (FIO_2=0.3, PO_2>50 torr) at a mean age of 5.4 hours and six infants, mean birth weight 2320gms were treated with late CPAP (FIO_2=0.5, PO_2>50) at a mean age of 22.5 hours ($P<.01$). Therapy was discontinued when FIO_2 requirement fell below 0.3 and a stable PO_2 was maintained for 4 hours. Although no significant differences were demonstrated in duration on CPAP, rate of improvement, or morbidity, the early group attained the criteria for discontinuing therapy at 47 hours of age, whereas the late group reached this level at 83 hours of age ($P>.05$).

The response of the arterial oxygen (PO_2) and carbon dioxide (PCO_2) tensions to the application of 6cm CPAP was also similar. The PO_2 rose 14 in the early and 20 torr in the late group while the PCO_2 remained the same. In conclusion, we did not find significant differences between the early and late treated groups with respect to response to the CPAP. However, since both groups were determined to have disease of equal severity in the first hours of life, there is a suggestion that early intervention with CPAP may modify the progression of RDS.

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THE RESPONSE OF PO_2 AND PCO_2 TO THE WITHDRAWAL OF CONTINUOUS POSITIVE AIRWAY PRESSURE (CPAP) IN INFANTS RECOVERING FROM RESPIRATORY DISTRESS SYNDROME. (RDS) Thomas Hegyi & I. Mark Hiatt (sponsored by W.C. Heird) Department of Pediatrics, Monmouth Medical Center, Long Branch, New Jersey

Unexpected stability in arterial oxygen (PO_2) and carbon dioxide tensions (PCO_2) has been noted in infants recovering from RDS upon the discontinuation of CPAP at 6cm H_2O. Twenty-seven infants (weight 880-2650gms, gestational age 29-36 weeks) were treated with nasal prong CPAP at a mean age of 9.4 hours and for a mean duration of 53.7 hours. CPAP was discontinued when FIO_2 requirement fell below .3 and stable PO_2 was maintained for 4 hours. Abrupt cessation of CPAP at 6cm H_2O resulted in a mean change in PO_2 of -2torr and a mean change in PCO_2 of -1 torr. 43% of PO_2 values increased (mean, 10 torr) and 56% of PCO_2 values decreased (mean, 6 torr). This response did not vary with birth weight, gestational age, severity of disease, or duration on CPAP.

We postulate that the mechanism for the stability of arterial blood gas tensions unresponsive to the removal of CPAP is due to opposing physiologic changes, decreased alveolar ventilation and increased alveolar perfusion.

We conclude that PO_2 and PCO_2 are not affected by the withdrawal of nasal CPAP at 6cm H_2O and that time consuming weaning procedures are unnecessary.

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THE USE OF A NEW RESPIRATORY INDEX TO ASSESS THE SEVERITY OF RESPIRATORY DISTRESS SYNDROME IN THE PREMATURE INFANT. Thomas Hegyi & I. Mark Hiatt (sponsored by W.C. Heird) Department of Pediatrics, Monmouth Medical Center, Long Branch, New Jersey

There is a need for an accurate & reproducible classification of severity of illness early in the course of RDS. The use of a new respiratory index $RI=AaDO_2/PO_2$ (Goldfarb et al A.J. Surg 129:255) has been found to be an important adjunct in the evaluation of twenty-six ill premature infants. The patients were divided into three groups on the basis of their calculated respiratory indices: Group I (RI=0-3), Group II (RI=3-6) and Group III (RI>6).

	Group I (n=11)	Group II (n=9)	Group III (n=6)
RI (mean)	2.1	4.0	10.8
B.W. (mean)	1810	2200	1990
G.A. (mean)	33	34	35
Response to CPAP			
PO_2 (torr)	+23	+5	-10
PCO_2 (torr)	-6	+4	+ 5
Duration on CPAP (hrs)	41	72	94
N,FIO_2 0.6	0	3	6
N,FIO_2 0.4	5	7	6

The use of a numerical index facilitates the objective assessment of the initial severity of RDS. In addition, the RI may also be useful in tracking the course of disease and comparing the effectiveness of different treatment regimens.

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EFFECT OF PASTEURIZATION AND FREEZING ON THE BACTERIAL GROWTH INHIBITING ACTIVITY OF HUMAN BREAST MILK. Jacinto Hernandez, Pamela Lemons, James Lemons, and James Todd (Intr. L. Joseph Butterfield). Department of Pediatrics, The Children's Hospital of Denver and the University of Colorado School of Medicine, Denver, Colorado.

Recent recommendations have discouraged the use of frozen human breast milk for the feeding of preterm or sick newborns, based on animal model data which demonstrated a failure of frozen rat milk to inhibit bacterial growth in vitro or prevent bacterial induced necrotizing enterocolitis in vivo. Nine human breast milk samples were collected and aliquots were maintained at room temperature, frozen, and pasteurized. Samples were inoculated with 10-50 colony forming units per ml (CFU/ml) of E. coli (K1) or group B streptococcus (1a) and incubated at 37°C. Quantitative growth was measured at eight and 24 hours. Control broth, commercial formula, and pasteurized breast milk showed no inhibition with a rapid logarithmic growth to a maximum of 10^8-10^9 CFU/ml at 24 hours. Compared to these controls, fresh breast milk, fresh frozen breast milk, and that frozen for 21 days demonstrated significant inhibition of growth ($p < 0.001$). A trend toward loss of inhibiting activity for group B streptococcus was noted with prolonged freezing of breast milk but not for E. coli. The bacterial growth inhibiting effect of human breast milk may be multifactorial (cells, antibodies, complement, lactoferrin, lysozyme). Although freezing may quantitatively decrease the amount of some of these factors, it cannot be assumed that comparable functional reductions will necessarily result.