HYPEROSMOLALITY OF FORMULAS USED IN THE NICU AFTER THE ADDITION OF ORAL OR PARENTERAL MEDICATIONS, G.I.

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The low birth weight infant graduating from critical to inter-

mediate growing care is oftentimes placed on medications which when added to standard formulas exceed a safe osmotic load (400  $mOsm/kg H_20$ ).

Oral and parenteral preparations of the seven most common medications used in our nursery were added to Similac with Iron (Ross Laboratories, Colombus, Ohio, osmolality 292 m0sm/kg  $\rm H_20$ ) in all possible combinations. These included: Furosemide, Aminophylline, Multiwitamins, Vitamin E, Potassium Chloride, Digoxin and Phenobarbital.

The osmolalities of the formula were measured with added oral or parenteral medications. The parenteral medications added contained the same amount of ordered medication but the smaller volumes used afforded a lower osmolality. The osmolalities of the 127 samples were:

Osmolal.	Oral med. + F.	Parenteral med. + F.	Osmolal.	Oral med.	Parenteral med. + F.
<400	21	58	700-799	23	0
400-499	19	27	800-899	22	ő
500-599	19	41	900-999	12	ñ
600-699	11	1			v

It may be necessary to calculate the osmolalities of medications added to the formula of growing premature infants and consider using parenteral preparations in order to decrease the final osmolality of the material.

OSMOLALITIES OF ORAL AND PARENTERAL MEDICATIONS USED IN THE NEONATAL INTENSIVE CARE UNIT, G.I. Martin, R. Jacks, W. Snider and A. Cohen. (Sponsored by F.Y.K. Mu) The Queen Of The Valley Hospital, West Covina, California. Although the association between hypertonic medications and the development of Necrotizing Enterocolitis is unclear, hyperomoner materials (more than 400 more when 1400 more than 400 more

materials (more than 400 m0sm/kg  $\rm H_20$ ) can cause an osmotic diuresis, loose stools and probable mucosal injury. Osmolalities on the seven most common medications used in our

nursery were measured by freezing point depression and checked with calculations based upon standard equations.

		ORAL		PARENTERAL		
Generic Name	Concen- tration/drug	Volume	Osmo- lality	Volume	Osmo- lality	
F*	3mg	.3mL	3340	.3mL	287	_
A*	2mag	2mL	165	.08mL	102	
M*		.5mL	10,540	1mL	<b>5</b> 820	
Λ*	2 <b>5</b> U	.5mL	4170	.125mI	146	
P*	.65maEq	.5ml	<b>45</b> 20	.4mL	<b>35</b> 80	
D*	10	.2mL	<b>357</b> 0	. 1mL	8440	
P*	5mg	1.25mI	9610	.038mI	16,280	

\*Furosemide, Aminophylline, Multivitamins, Vitamin E, Potassium Chloride, Digowin and Phenobarbital.

It May be necessary to calculate the osmolalities of medica-tions offered to growing premature infants and consider using parenteral preparations.

T1430

CPAP ONLY REDUCES NEONATAL APNEA ASSOCIATED WITH AIRWAY OBSTRUCTION. M.J.Miller, W.A.Carlo, R.J. Martin. Dept.Peds, Rainbow B&C Hosp, CWRU, Cleve,OH Although continuous positive airway pressure (CPAP) is an established treatment for apnea of prematurity, its physiologic mechanism of action is unclear. We therefore investigated the effect of CPAP on obstructive control and mixed experience. mechanism of action is unclear, we therefore investigated the effect of CPAP on obstructive, central and mixed apneas, defined by the presence or absence of upper airway obstruction. 14 preterm infants of GA 28 wk (range 26-32) and WT 1156g (range 680-2000g) were studied during sequential 45 min periods on, off, and again on 4 cm H<sub>2</sub>O CPAP. Ventilation (via nasal mask pneumotar) HP chest wall movement. Transcript and Transcript of the contraction of the contractio and again on a com n20 crar. Ventilation via hasai mass pheumo tachometer), HR, chest wall movement, TcPO<sub>2</sub> and TcPCO<sub>2</sub> were con-tinuously recorded. 252 apneas ≥5 sec (mean duration 13±19 sec), and 170 apneas ≥10 sec (mean duration 17±9 sec) were analyzed.

CPAP markedly reduced both mixed and obstructive appeas ≥5 sec and ≥10 sec. In contrast, central apneas ≥5 and ≥10 sec were entirely unaffected by CPAP.

No. of Apneas ≥5 sec (M±SD)						
	On CPAP	Off CPAP	On CPAP	Off vs. On		
Mixed	$0.3 \pm 0.5$	6.3± 5.5*	0.1± 0.3	* p<0.01		
Obstructive	0	1.9± 2.3**	0.2± 0.6	** p<0.01		
Central	7.3±13.8	7.4±11.9***	8.4±16.5	***NS		
Although minute ventilation was not altered, TcPO2 increased						
Dy IIII mmHg on CPAP, whether or not appea was present We						
conclude that CPAP only relieves appea with an obstructive com-						
ponent, possibly by splinting the pharyngeal airway or reflar-						
ly activating muscles which maintain upper airway patency in						
Dreterm infant	- 6		res allay	paceticy in		

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1430 A CMV (NACMV) IN NEWBORNS (NB). J. McMillan, L. Weiner, H. Lamberson, M. Williams, D. Clark, C. McMahon, E. Bousman, A. Patti. SUNY, Upstate Medical Center, Dept. of Peds and American Red Cross Blood

Medical Center, Dept. of Peds and American Red Cross Blood Services, Syracuse, NY. (Spon. by R. Spitzer).

Weekly CMV cultures as well as admission and discharge CMV serologies were performed on 400 NB in an ongoing NICU study of NACMV. Seven of 106 (6.6%) NB hospitalized for > 4 wks acquired CMV, 3 were symptomatic. Blood from 28 ELISA+ and 13 IgM-FA+ units was administered to the 7 NB who acquired CMV. Forty-eight seronegative(S-) NB received blood from the 28 ELISA+ units. Nineteen of the 48 remained hospitalized >14 days following their last ELISA+ transfusion: 6/19 became culture positive(C+) and seropositive(S+), and 5/19 became S+. A transfusion of 8 ml of blood from 1 ELISA+, IgM-FA+ unit was the only blood product received by one of the S- NB, and he became C+ 4 wks later, at 8 wks of age. Twenty ml of this same unit were administered to wks of age. Twenty ml of this same unit were administered to another S- NB who became C+ at 19 wks of age, 3 wks after the transfusion. In addition, 1 NB who was S+ at birth received 11 In addition, I was with was S+ at DITCH received II ml from the same unit 4 wks before becoming C+ at 8 wks of life. Thus 3 of the 7 NB with NACMV apparently acquired infection from 1 ELISA+, IgM-FA+ unit. However, another NB received 3 ml from this same unit and remained uninfected. Twelve of 34 initially St study NB reverted to St. It appears that seropositivity at birth may not provide long term protection against NACMV. In addition, small volumes from an infectious unit can transmit CMV to more than I susceptible NB, but all susceptible recipients may not become infected.

THE IMPROVED OXYGENATION OF PRETERM INFANTS DURING THE IMPROVED OXYGENATION OF PRETERM INFANTS DURING NON-NUTRITIVE SUCKING DEPENDS ON POSTCONCEPTIONAL AGE. R. Paludetto, S. Robertson, M. Hack, C. Shivpuri, Dept. Peds, Case Western Res. U., Cleveland, OH and School of Med., Naples, Italy
Non-nutritive sucking (NNS) has been reported to stabilize transcutaneous (tc) PO2 in preterm infants during both gavage feeding and secieted ventilation. To evaluate the effect of NNS

feeding and assisted ventilation. To evaluate the effect of NNS in the resting preterm infant, we measured TcPO<sub>2</sub>, heart rate (HR) and resp. rate in 14 healthy, sleeping infants (C.A. at birth 26-33 wk, B.Wt 710-1450g). Each study was begun in active sleep, and comprised 15 min. of data collection divided into 3 equal periods: preNNS, NNS and postNNS. Sucking was recorded with a pacifier adapted to measure sucking pressure, and respiration was detected with a nasal thermistor. Subjects were studied at two weekly intervals until discharge and divided into 4 groups

two weekly intervals until discharge and divided into 4 groups based on postconceptional age. During NNS,  $TcPO_2$  increased  $2.3\pm0.9$  mmHg at 32-33 wk (p<.01) and  $4.0\pm1.5$  mmHg at 34-35 wk (p<.05). In contrast,  $TcPO_2$  did not increase during NNS at either 36-37 or 38-39 wk. HR increased during NNS in all 4 groups although this was only significant at 34-35 wk (p<.01) and 36-37 wk (p<.02), while resp. rate never changed. Time spent in active sleap during the 5 min, enoched did changed. Time spent in active sleep during the 5 min. epochs did not differ between the 3 periods and ranged from 61% to 96%. Thus the increase in TcPO2 during NNS is dependent on the inchange in resp. rate or sleep state. These data suggest that providing an opportunity for NNS may be a beneficial intervention in the care of healthy preterm infants between 32 and 35 wk postconcentional age. wk postconceptional age.

NEAR MISS SUDDEN INFANT DEATH SYNDROME (SIDS) AND 1432 SIDS IN NEWBORN NURSERIES: A NEW HOME FOR AN OLD PROBLEM? Oommen P. Mathew and Jatinder Bhatia (Spon. by David K. Rassin), University of Texas Medical Branch,

Dept. of Pediatrics, Galveston, Texas.
Higher incidence of SIDS in low birth weight infants and infants with bronchopulmonary dysplasia (BPD) has been documen-Increased survival and prolonged hospitalization make low birth weight infants uniquely vulnerable to near miss episodes and SIDS while they are still in the nursery. A two year experience is reported. Patient #1 was a 28 week, 970 gm infant, third of a set of triplets who developed BPD following mechanical ventilation for respiratory distress syndrome (RDS). At 46 weeks ventilation for respiratory distress syndrome (RDS). At 46 weeks postconceptional age infant was found apneic and cyanotic requiring cardiopulmonary resuscitation (CPR). Patient #2, was a sibling of Patient 1, who also developed BPD following mechanical ventilation for RDS. At 58 weeks postconceptional age infant was found apneic and required CPR. Patient #3, a 1340 gm, 35 week infant developed apnea and cyanosis only during feeding at 4 weeks of age. At six weeks of age infant was found apneic and cyanotic requiring CPR. Patient #4 was a 1320 gm, 31 week infant who at 7 weeks of age was found apneic and cyanotic. Resuscitacyanotic requiring CPR. Patient #4 was a 1320 gm, 31 week infam who at 7 weeks of age was found apneic and cyanotic. Resuscitation was unsuccessful. Patient #5 was a healthy term infant found apneic and limp at 23 hours of age. Resuscitation was unsuccessful and postmortem findings were consistent with SIDS. We are reporting these 3 infants with near miss SIDS and 2 infants who were victims of SIDS to heighten the awareness of such a problem in the peuborn surveyies. problem in the newborn nurseries.

 $^{
m l}$ Recipient of NIH Clinical Investigator Award HLO1156.