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WHICH MATURE BABIES NEED TO BE OBSERVED IN THE NURSE-
RY: ANALYSIS OF THE SURFACE TENSION (ST) OF AMNIOTIC
FLUID (AF) LIPID EXTRACT (LE). Chandra M. Tiwary,
James B. Haddock, Richard D. Landes, and Doris Burgess (spons.
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We reported (Ped. Res. 1981;15:1452A) that the mothers whose
AF LE showed reduced ST lowering property delivered babies who
developed complications in the neonatal period. This study in-
cluded babies of all weights. To exclude the impact of premies we
examined the predictive value of ST lowering property of the AF
LE for newborns weighing ≥ 2500 gm. The ST was measured on 64 AF
LE by the standard method. The ST sum was calculated by adding
the volume and the ST (both are the minimum volume (ul) of the
AF LE required to maximally lower the ST (dynes/cm)).

In 28 babies (815,913), the ST sum was ≤ 40 ; 22 were normal and
6 (85,91) showed complications: meconium staining-3, ABO incompat-
ibility-2, hyperbilirubinemia requiring phototherapy-2, and
Down's syndrome-1. In 36 babies (817,919), the ST sum was ≥ 40 ;
19 were normal and 17 (86,911) showed complications: Rh and other
isoimmune hemolytic diseases requiring exchange transfusion-4,
hyperbilirubinemia requiring phototherapy-2, ABO incompatibility-
2, polycythemia requiring partial exchange transfusion-1, hypo-
glycemia-4, possible sepsis-1, and meconium staining-3. The mo-
thers of only 5 babies showed a prenatal condition suggesting a
need for the baby's observation. Conclusion: ST sum value is a
nonspecific indicator of a baby's health. A high value suggests
a need for observation of a ≥ 2500 gm baby; a low value suggests an
absence of complications subsequent to delivery.

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Effect of Prenatal Glucocorticoid on Fetal Rat Lung
Prostaglandin Synthesis. Michael Y. Tsai, Mark W.
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Prenatal Glucocorticoid therapy is increasingly being used for
accelerating fetal lung maturation. Glucocorticoids, however,
are also known to inhibit phospholipase A_2 and thus the synthesis
of prostaglandins (PG). In perinatal rat lung, the major PG is
prostaglandin (PGI_2), a potent vaso- and bronchodilator important
in lung function. To determine the effect of glucocorticoid
therapy on fetal lung PGI_2 synthesis, we measured 6-keto- $PGF_{1\alpha}$
(the stable breakdown product of PGI_2) levels by RIA. Pregnant
rats received 4 doses of dexamethasone (DEX) (0.4 mg/kg) at 12 hr
intervals prior to sacrifice. Table 1 shows the 6-keto- $PGF_{1\alpha}$
levels of fetal lungs from DEX-treated and control mothers (mean
 $\pm$ SEM, 4 fetuses from each of 6 litters for each group).

DEX Treatment	6-keto- $PGF_{1\alpha}$ (pg/mg protein)	
	21 Days Gestation	22 Days Gestation
Control	292 $\pm$ 49	256 $\pm$ 36
0.4mg/kg	443 $\pm$ 49	443 $\pm$ 28

DEX treatment significantly increased 6-keto- $PGF_{1\alpha}$ levels. There
were no significant differences between male and female fetuses
with or without DEX treatment. GC/MS studies confirmed results
obtained by RIA. These results suggest that prenatal DEX en-
hances endogenous levels of 6-keto- $PGF_{1\alpha}$ in fetal lung. Since
 PGI_2 may be important in perinatal lung maturation and function,
the effectiveness of glucocorticoid therapy for accelerating
functional lung maturity may be partly due to the stimulation of
 PGI_2 synthesis.

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ELEVATED CALCITONIN (CT) IN BIRTH ASPHYXIA AND
PREMATURITY: ROLE IN THE PATHOGENESIS OF EARLY
NEONATAL HYPOCALCEMIA (HC) P. Venkataraman, R.C.
Tsang, I. Chen, M. Sperling, Dept. Pediatr., Univ. of Cincinnati
Although CT is stress responsive the role of CT in pathogene-
sis of early neonatal HC is unknown. We studied the thesis that
CT, gastrin, glucagon 1) are higher in cord than mother; 2) rise
postnatally; 3) correlate inversely with gestation; 4) are higher
in birth asphyxia; and 5) elevated CT results in HC; 6) gastrin
and glucagon are CT secretagogues. We studied 64 mother-infant
pairs, gestation 25-42 wks, Apgar 1' 6.2 $\pm$ 2.7, 5' 7.6 $\pm$ 2.2. Cord
Ca, Mg, P (mg/dl), CT, gastrin and glucagon (pg/ml) were mostly
higher than maternal, 10.15 $\pm$ (SEM) 0.18 vs 8.8 $\pm$ 0.16 (p<0.005);
1.95 $\pm$ 0.06 vs 1.8 $\pm$ 0.06 (p<0.05); 5.8 $\pm$ 0.25 vs 3.4 $\pm$ 0.13 (p<0.005);
81 $\pm$ 17 vs 49 $\pm$ 11 (p<0.05); 133 $\pm$ 20 vs 123 $\pm$ 15 (n.s.); 120 $\pm$ 9 vs 78 $\pm$ 7
pg/ml (p<0.005) respectively. In neonates at 24 h CT, gastrin
and glucagon rose to 254 $\pm$ 29 (p<0.005); 172 $\pm$ 28 (n.s.); 216 $\pm$ 17
pg/ml (p<0.005). Serum Ca fell to 8.7 $\pm$ 0.2, 8.7 $\pm$ 0.3 mg/dl at 24,
48 h, (p<0.005). Term cord CT correlated with 1' Apgar, r=-0.4
(p<0.05), at 5', r=-0.8 (p<0.0001). 24 h serum CT correlated
with 24 h serum Ca, r=-0.7 (p<0.0003) and 48 h Ca r=-0.93 (p<
0.0003). Cord CT was higher <32 wks vs term 146 $\pm$ 45 vs 61 $\pm$ 18
pg/ml (p<0.05) and higher with Apgar <6 vs >7 at 1' and 5', 118 $\pm$
37 vs 56 $\pm$ 18 and 266 $\pm$ 72 vs 49 $\pm$ 9 pg/ml resp (p<0.05). Neither serum
gastrin nor glucagon correlated with CT. Thus, 1) cord CT and
glucagon are elevated; 2) CT and glucagon rise postnatally; 3)
cord CT is higher in preterm and asphyxia; 4) high serum CT cor-
relates with low serum Ca. We speculate that elevated serum CT
may result in HC in preterm and birth asphyxiated infants.

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FUROSEMIDE EFFECTS ON NEWBORN RENAL & BONE
CALCIUM METABOLISM, Zhi-Ping Guan, Winston Koo,
Jerry Schutzman, Vicky Neumann, & Reginald C. Tsang,
University of Cincinnati College of Medicine.

Furosemide diuretics are commonly used in neonatal intensive care.
Recent anecdotal reports have appeared of preterm infants who develop
renal calcification & osteopenia on chronic high dose furosemide ther-
apy. The mechanisms for development of these possible complications in
infancy is unclear. We hypothesize that furosemide diuretics result
directly in hypercalciuria, nephrocalcinosis, secondary hyperparathy-
roidism & decreased bone mineral content. Newborn rats were randomiz-
ed from day four into control & treated groups for a 28 day study. Grp.
1, placebo; Grp. 2, daily 5 mg/kg of furosemide; Grp 3, 15 mg/kg of
furosemide. By analysis of variance, urinary calcium increased from 7.81
to 11.25 to 20.35 mg/dl for the three respective groups (p<.05). Urinary
Mg also increased from 13.1 to 14.1 to 19.3 mg/dl. Urinary P did not
increase. Renal Ca ash content of treatment grps. were significantly
increased (6 of 25 & 6 of 26) beyond control 95% limit. Chi-square p<.05.
Bone weight of tibias was decreased from .21 to .17, .16 gs. (p<.01), as
was ash weight .13, .11, .10 gs. (p<.05), in association with decreases in
body weight of 68, 62, 57 gs. Bone Ca & body weight were correlated (p
<.01). Serum Ca, Mg, P, & parathyroid hormone concentrations (mid
molecule 44-68 radioimmunoassay, rat standard, CV 9%), were not
different among grps. Thus, furosemide in newborn rats results in
increased urinary Ca, increased renal Ca content, decreased bone
mineral, decreased body weight & no changes in serum Ca, Mg, P or
parathyroid hormone. We speculate that the effect of furosemide
therapy in the newborn on Ca metabolism is directly related to increased
Ca loss in the urine.

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LONG TERM FOLLOW-UP IN ≤ 1500 GM. BIRTHWEIGHT (VLBW).
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M. G. Robinson), Medical College of Ohio, The Toledo Hospital,
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376 VLBW neonates were admitted to Regional Perinatal Center,
between July 1979 to December 1981. 281 (75%) survived, 127
(45%) were followed up for up to 18 months corrected age and 3
years of age. The overall neonatal mortality was 25%; mean ges-
tational age, 29.8 $\pm$ 2.35 wk.; mean birthweight, 1113 $\pm$ 230 gm.;
SGA, 26%; Apgar ≤ 3 at 1 min., 35%; ≤ 5 at 5 min., 21%; outborn
35%, ventilated 78%. CT scan/ultrasound was done on 77 (61%),
of which 38 (49%) had paraventricular-intraventricular hemorrhage.
Neurologic examination, Bayley Scales of Infant Development,
McCarthy Scales of Children's Abilities were done. Cerebral
palsy or developmental delay (MDI more than 2 standard deviations
below the mean), visual deficits were considered severe handi-
caps. Mean Bayley Score 70-84 were considered suspect.

BIRTHWEIGHT (g)	NO HANDICAP	SUSPECT	SEVERE HANDICAP
500 - 750	5	1	3
751 - 1000	20	9	5
1001 - 1250	26	9	1
1251 - 1500	25	14	9
TOTAL	76 (60%)	33 (26%)	18 (14%)

Our data confirms optimistic results of modern perinatal care.
Additional work is needed to further reduce incidence of handicap.

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LONG TERM FOLLOW-UP IN VERY LOW BIRTHWEIGHT (VLBW)
NEONATES WITH PARAVENTRICULAR INTRAVENTRICULAR HEM-
ORRHAGE. J. G. Urrutia, T. Mathew, E. Brookfield,
M. Satish, S. McQuiston, J. Butterfield, S. ElShafie. (Spon. by
M. G. Robinson) Medical College of Ohio, The Toledo Hospital,
Dept. of Ped., Toledo, Ohio.

78 VLBW admitted to the Neonatal Intensive Care Unit, in the
Regional Perinatal Center, during the period July 1979 to Decem-
ber 1981 had CT brain scan and/or ultrasound. They were followed
at 18-24 months corrected age and neurologic examination and
Bayley Scales of Infant Development were performed. Their ges-
tation (wks.) and birthweight (kg.) in the control group were
G.A. 29.3 $\pm$ 1.9 and birthweight 1.050 $\pm$ 0.2 and paraventricular-
intraventricular group were G.A. 29.2 $\pm$ 2.3 and birthweight
1.052 $\pm$ 1.3. Degree of hemorrhage was graded according to Papile.

(N)	Normal	G I	G II	G III	G IV
Major Handicap	40	8	8	14	8
MDI	4(10%)	1(13%)	0	5(36%)	7(88%)
PDI	88 $\pm$ 17	87 $\pm$ 11	91.5 $\pm$ 24	68.4 $\pm$ 16	59 $\pm$ 17
Mean $\pm$ S.D.	85 $\pm$ 12	86 $\pm$ 8	87.1 $\pm$ 10	77 $\pm$ 14	50 $\pm$ 8.5

Progressive significant motor and mental handicaps were found
with Grade III and IV hemorrhage.