†1278 THE ADVERSE EFFECT OF SUPRAVENTRICULAR TACHYCARDIA ON CARDIAC OUTPUT IN THE NEONATE.

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To assess the impact of supraventricular tachycardia (SVT) on cardiac output in the neoante, we measured noninvasively ascending aortic blood flow ($\dot{Q}_{Ao}$) pre and post cardioversion (cv) in 4 neonates with paroxysmal atrial tachycardia (PAT) or atrial flutter (AF). Ascending aortic blood flow velocity (V_{Ao}) was determined using a portable, 5MHz, pulsed Doppler velocimeter and suprasternal transducer. Aortic cross sectional area (A_{Ao}) was calculated from echographic measurements of the internal diameter of the ascending aorta. Left ventricular output was computed as $\dot{Q}_{Ao}(ml/min)=V_{Ao}(cm/sec)xA_{Ao}(cm^2)x60sec/min$. The infants ranged in age from 1 to 14 days, mean 5 days, and ranged in weight from 2.9 to 3.7kg, mean 3.5kg. 2 patients had PAT and 2 had AF with 2:1 block. Conversion required DC countershock in 2 patients, digoxin in 1, and facial ice application in 1. Summary of the results of cv are shown in the table:

	Pre cv	Post cv	P value
Heart rate (BPM)	248 ± 22(SD)	155 ±13	.002
$\dot{Q}_{Ao}$(ml/min/kg)	116 ± 15	207 ±15*	.001
Stroke volume(ml/kg)	0.47 ± 0.1	1.35 ± 0.2	.001

*normal Q_{Ao} for neonates = 230 ± 50

This study demonstrates that SVT impairs significantly cardiac output and stroke volume in the neonate and emphasizes the impact of successful cardioversion in returning systemic blood flow to normal levels.

1281 NEUTROPHIL RESPONSE TO CHEMOTAXINS IN THE RAT PERITONEUM: A MODEL FOR THE INFLAMMATORY RESPONSE IN NEC.

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Neonates with necrotizing enterocolitis have a peritoneal inflammatory response which has not been examined as a factor in the neutropenia of NEC. The rat peritoneum was used to determine the independent effects of protein and bacterial toxin on neutrophil mobilization.

30 juvenile rats were divided into 3 groups. Casein(10 mg), cholera enterotoxin(1ug) or saline were injected IP in 3 trials. In 2 trials rats were sacrificed at 5 hrs., and in the 3rd at 24 hrs. Peripheral blood(B) peritoneal(P) and bone marrow(B.M.) total WBC counts with differentials were obtained. The distribution of mature neutrophils (polys and bands) in each compartment was determined. Table (B=nx10^3; P,BM=nx10^6).

	Exp. 1(5hrs)			Exp. 2(5hrs)			Exp. 3(24 hrs)		
	B	P	BM	B	P	BM	B	P	BM
Control	2.35	9.11	-	0.88	7.77	32.07	0.48	2.82	30.16
Casein	5.62*	64.78*	-	1.14	37.83*	20.67*	0.65	13.17*	15.62*
Toxin	10.51*	57.28*	-	1.80*	35.77*	16.55*	2.73*	-	15.59*

*signifies p<.01 compared to control

Casein and toxin provoke mobilization of mature neutrophils into the peritoneum. With toxin there is also a fluid response which at 24 hrs was exudative precluding quantitation of cells. Although in 2 experiments bone marrow neutrophils were depleted, stores were adequate to avoid neutropenia. This may not be the case in neonates with NEC. Dietary protein(casein) and bacterial toxin in the peritoneum of babies with NEC may exert a neutrophil chemotactic response.

●1279 EFFECT OF PHENOBARBITAL ON CEREBRAL BLOOD FLOW AND TOTAL BRAIN OXYGEN CONSUMPTION IN NEWBORN PIGLETS.

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The effect of phenobarbital (Pb) on cerebral blood flow (CBF) and total brain oxygen consumption (TBOC) was studied in 3 groups of awake newborn piglets (ages 1-3.5 d). Group I (control n=9) received saline, Group II (n=9) received low Pb dose (15 mg/kg IV) and Group III (n=9) received high Pb dose (45 mg/kg IV). Four CBF and cardiac output measurements per piglet using radioactive microspheres (^{141}Ce,^{85}Sr,^{95}Nb,^{46}Sc) arterial blood gases, 02 content hematocrit and plasma glucose were obtained at 0,15,30,60 min after saline or Pb injections. In all groups, pH, PO2, PCO2, blood pressure, heart rate, temperature and plasma glucose remained unchanged except a 14% decrease ($p<0.05$) in BP and an increase ($p<0.05$) in PCO2 (3 torr) at 60 min in Groups II and III. Total cerebral blood flow (TCBF) in Groups II and III decreased by 13% and 11.5% respectively ($p<0.05$) at 15 min and was significantly lower ($p<0.05$) than Group I but returned to baseline 30 min after drug injection. Similar findings were noted in different areas (cerebrum, thalamus, brain stem and eyes). TCBF and TBOC remained unchanged in Group I. In Group II, TBOC decreased by 35% ($p<0.01$) at 15 min and returned to baseline at 60 min. In Group III, high Pb dose lowered TBOC by 34% at 30 min ($p<0.01$) which persisted to 60 min. We conclude that therapeutic Pb dose produced only a transient decrease in TCBF and TBOC, but a high dose produced a more important and sustained decrease in TCBF and TBOC.

1282 PERITONEAL NEUTROPHIL CELL RESPONSE IN NEC.

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Neutropenia is a well-known consequence of necrotizing enterocolitis in the newborn. Increased peripheral utilization or bone marrow suppression with systemic sepsis have been implicated as mechanisms for this neutropenia. However, many infants with NEC do not have positive blood cultures. We examined peritoneal as well as peripheral leukocyte counts at the time of surgery in 6 neonates with NEC.

Total WBC's(T) were determined by coulter counter. Mature neutrophil(N) and band cell counts(B) were performed and a ratio of B:N+B was determined. All infants had cultures of blood, peritoneal fluid* and stool (rectal) swabs. (T,N,B=nx10^3)

			PERIPHERAL			PERITONEAL			
Pt	Ga/wk	Onset	T	N+B	B:N+B	T	N+B	B:N+B	Orgs*
#1	38	6 da	8.2	4.9	0.92	24.7	22.7	0.50	None
#2	37	23	4.9	2.2	0.97	59.0	47.8	0.37	None
#3	30	9	33.1	29.3	0.55	3.5	2.3	0.15	E.Coli
#4	26	35	5.1	2.5	0.55	12.8	9.5	0.30	None
#5	26	38	7.9	2.4	0.61	13.5	11.2	0.33	E.Coli
#6	37	2	2.5	1.6	0.61	10.2	9.3	0.50	Kleb.

All infants except one(#3) had a substantial mobilization of neutrophils into the peritoneum during the acute episode of NEC. This occurred even in the absence of perforation(#4) and with the perforation walled off(#1). The table demonstrates a preferential mature neutrophil response. Stools were positive for Enterococci(#3) and Rotavirus(#4). No patient had a positive blood culture. Thus neutropenia in infants with NEC may be attributed to consumption of mature neutrophils in the peritoneum.

1280 APNEA AND PERIODIC BREATHING VALUES DURING THE FIRST YEAR IN CONTROL TERM INFANTS.

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Currently, recordings for apnea and periodic breathing (PB)* events are done to assess infants with apparent "life threatening episodes" to determine their management. The following data are from term infants studied at 3 wks to 1 yr. Respiratory measurements were made with strain gauges and nasal thermistors in a quiet hospital room. One day recordings were done at 3 wks to 6 mos; and overnight at 9 mos and 1 yr. Five of the 9 mos. and 13 of the 1 yr. infants were "near miss" for SIDS who had had no apnea or repeat near miss episodes for at least 2 months prior to recording.

Age		3w	6w	3m	4½m	6m	9m	1y
N		(10)	(9)	(9)	(10)	(9)	(7)	(17)
Sleep time	X	15.1	14.4	13.5	13.8	12.2	7.9	7.9
(hr)	S.D.	1.4	1.2	1.1	.8	1.0	.9	1.5
# of PB	X	167	145	59	45	63	54	36
pauses	S.D.	172	200	22	29	43	35	31
# 10 sec.	X	12	8	3	3	3	5	6
pauses	S.D.	11	11	3	5	4	3	7

Prolonged apnea (≥20 s) with oxygen desaturation is clearly abnormal. The clinical significance of shorter pauses and PB is less certain.

*2 or more, >3 s pauses within 20 s.

1283 NEUROPHYSICAL MATURATION IN IDM: EFFECT OF EARLY MATERNAL MANAGEMENT

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Although diabetes in pregnancy is thought to delay maturation of fetuses, its effects on neurophysical maturation have not been examined, especially in regard to the time of initiation of diabetic management. We tested the hypothesis that the diabetic intrauterine environment will retard the rate of fetal neurophysical maturation and that institution of maternal metabolic control before 9 wks of pregnancy (early) will reduce the maturation delay. Gestation was assessed in 104 infants of insulin dependent diabetic mothers (IDM) followed prospectively and in 221 infants of non-diabetic mothers (non-IDM), by standard clinical exam (Ballard, J. et al, 1979). Both groups had reliable obstetrically confirmed menstrual dates. At all gestations neurophysical maturity was advanced by 0.6 weeks in IDM when compared with non-IDM ($p=0.005$). Early institution of diabetic management in pregnancy (n=76) vs later (n=28) was not associated with change in rate of maturation. In the IDM group there was no effect of race, sex, or tobacco, alcohol, or coffee consumption on maturation. Lack of clinical RDS was associated with accelerated neurophysical maturation ($p<0.003$). We conclude that the diabetic intrauterine environment has an accelerating rather than retarding effect on gestational neurophysical maturation; early institution of diabetic management does not affect the rate of intrauterine maturation.