●1453 APNEA AND BRADYCARDIA DURING ORAL FEEDING IN TERM NEONATES.

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Recent studies in infants have demonstrated substantial reduction in minute ventilation during oral feeding. The aim of the present study was to determine the incidence of cyanosis and bradycardia during oral feeding as a result of the reduction in minute ventilation or apnea. Heart rate, sucking pressure, respiratory efforts and airflow were monitored continuously during feeding in 50 term neonates (mean birth weight 3.5 kg, gestational age 39.7 weeks) in the first week of life. Bradycardia (<100/min) occurred in 9 (18%) infants during the continuous sucking phase of oral feeding. Six of these episodes were preceded by apnea and the remaining 3 episodes were associated with hypopnea (marked reduction in minute ventilation). Airway obstruction occurred during most of the apneic episodes (5/6) and two resulted in cyanosis. The apnea and bradycardia resolved spontaneously with continued feeding in all except one infant. The only intervention performed was discontinuation of feeding in this infant. No episodes of isolated bradycardia or aspiration (associated with coughing and/or choking) was seen in any of the infants monitored. Our results suggest that apnea and transient bradycardia occur more frequently than previously recognized in term infants during oral feeding. This presumably reflects the inability of some infants in coordinating the feeding and breathing patterns during the first week of life and should be considered normal unless it persists beyond the neonatal period.

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†1454 ANEMIA BLUNTS THE NEONATAL HOMEOTHERMIC RESPONSE TO ENVIRONMENTAL COLD STRESS(ECS).

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The homeothermic response to ECS includes increased O_2 consumption ($\dot{V}O_2$) with heat production. Anemia may blunt this response by reducing O_2 transport; limiting $\dot{V}O_2$. We tested this hypothesis in four, awake 3-day old piglets during CONTROL(HCT=26.4±0.9%) and ANEMIC(HCT=14.8±0.1%) periods. Measurements of core(Tc) and ambient(Tamb) temperatures, arterial-mixed venous O_2 contents, and cardiac output(CO, microsphere method) were made in a warm environment(W) and after 60 min. of ECS. Following recovery from ECS, an isovolemic plasma exchange transfusion was done to lower the HCT. W and ECS measurements were then repeated as described. The A-VΔO_2, $\dot{V}O_2$ and O_2 extraction (O_2Ex) were calculated from measured values. Results are below (M±SEM):

	CONTROL		ANEMIA	
TIME(min.)	0(W)	60(ECS)	0(W)	60(ECS)
Tc(°C)	39.0±0.2	38.5±0.4	38.8±0.3	37.3±0.5*+
Tamb(°C)	31.9±0.5	19.8±0.6*	32.0±0.7	19.1±0.4*
CO(ml·kg^{-1}·min^{-1})	489±41	603±82	523±68	591±53
A-VΔO_2(ml/dl)	3.0±0.6	4.6±0.6	2.6±0.4	3.3±0.5
$\dot{V}O_2$(ml·kg^{-1}·min^{-1})	14.6±3.4	28.3±0.8*	14.8±4.1	18.1±3.4
O_2Ex(%)	34.8±6.4	54.6±4.5	49.9±5.3	64.8±9.1

*p<0.05 vs. W, +p<0.05 vs. CONTROL for same study period

Although O_2Ex increased during ECS with anemia, $\dot{V}O_2$ and, presumably, heat production were limited with resultant hypothermia. These preliminary data suggest that anemia blunts the homeothermic response to ECS in newborn piglets.

†1455 REGIONAL OXYGEN(O_2) DELIVERY DURING ENVIRONMENTAL COLD STRESS(ECS) IN ANEMIC PIGLETS.

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We studied regional O_2 delivery (DO_2) in 3, awake 3-day old piglets during CONTROL(HCT=25.6±0.8%) and ANEMIC(HCT=14.9±0.1%) periods. Measurements of blood flow($\dot{Q}$,radiolabeled microspheres) and arterial O_2 content(CaO_2) were made in a warm environment(W) and after 60 min.of ECS. DO_2 was calculated from measured values (ml O_2·100g^{-1}·min^{-1}). Preliminary results are below (Mean±SEM):

	CONTROL		ANEMIA	
TIME(min.)	0(W)	60(ECS)	0(W)	60(ECS)
Tamb(°C)	32.0±0.6	20.1±0.7*	31.6±0.9	19.3±0.5*
BRAIN(DO_2BR)	7.7±0.3	8.3±0.5	8.4±1.5	8.0±1.3
HEART(DO_2HT)	31.1±4.2	45.0±14.5	39.4±2.5	47.4±7.6
SKEL. MUSC.(DO_2SM)	4.7±1.3	12.1±1.6*	2.9±0.3	6.7±2.0+
GASTROINT.(DO_2GI)	16.5±1.5	10.8±1.5*	8.5±2.1+	5.6±1.6+Δ
KIDNEY(DO_2KI)	28.4±1.9	30.6±6.2	21.9±8.2	15.5±5.7Δ
ADRENAL(DO_2AD)	30.0±5.0	20.3±2.9	16.1±3.6+	13.3±3.2Δ

*p <0.05 vs. W-same group
+p <0.05 vs. CONTROL for same study period
Δp <0.05 vs. W-CONTROL

We conclude that anemia with ECS:1)blunts the normal increase in DO_2SM 2)augments the decrease in DO_2GI 3)has no effect on DO_2BR due to increased $\dot{Q}$BR 4)decreases DO_2KI and DO_2AD when compared with the normal, steady state(W-CONTROL). We speculate that, unless associated with increased regional O_2 extraction, anemia with ECS may compromise tissue oxygenation of less vital organs such as the kidneys, adrenals and/or gastrointestinal tract.

1456 REGIONAL BLOOD FLOW ($\dot{Q}$) DURING ENVIRONMENTAL COLD STRESS(ECS).

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We studied regional $\dot{Q}$ and its regulation during ECS in seven 3-4 day old piglets by measuring cardiac output(CO), organ $\dot{Q}$, arterial-mixed venous O_2 content difference(A-VΔO_2), O_2 consumption($\dot{V}O_2$), plasma epinephrine(E) and norepinephrine(NE), mean arterial pressure(MAP), and core (Tc) and ambient(Tamb) temperatures. A vascular resistance index(R) was calculated(MAP ÷ $\dot{Q}$). Each animal was studied in a thermoneutral environment (CONTROL) and 30 min. after the nadir of ECS. Results(Mean±SEM):

	CONTROL	ECS
Tc(°C)	38.8±0.1	38.4±0.5
Tamb(°C)	30.8±0.8	18.8±0.6*
MAP(mmHg)	68.3±3.1	79.4±5.1
$\dot{V}O_2$(ml/kg/min)	13.4±1.9	27.6±2.9*
A-VΔO_2(ml/dl)	3.7±0.1	5.5±0.4*
E(pg/ml)	169±41	167±19
NE(pg/ml)	439±39	508±45
CO(ml/kg/min)	405±30	506±51
$\dot{Q}$(ml/100g/min):		
Heart(HT)	238±33	317±41
Skel.Ms.(SM)	41± 5	94± 8*
Brain(BR)	86± 6	89±11
Gastroint.(GI)	149±10	117±12*
Kidney(KI)	284±26	233±17*
Adrenal(AD)	284±34	222±22*
R(% change from CONTROL):		
Systemic		-2.5± 8
Heart		-6.8±12
Skel.Ms.		-48.5± 5*
Brain		+25±9±18
Gastroint.		+60.3±18*
Kidney		+46.1±12*
Adrenal		+61.6±25*

We conclude that ECS results in (1) †$\dot{Q}$SM(†heat production) due to †R (2) †$\dot{Q}$GI, $\dot{Q}$KI, and $\dot{Q}$AD due to †R (3)†A-VΔO_2 with unchanged E and NE. We speculate that changes in R result initially from metabolic autoregulation(†R) followed by altered peripheral sympathetic tone(†R). *p 0.05 vs. CONTROL

1457 AUTOREGULATION OF CEREBRAL BLOOD FLOW (CBF) IN THE EARLY AND LATE POST-ASPHYXIC PERIOD IN NEWBORN DOGS.

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Autoregulation (AR) of CBF in newborns is thought to be easily impaired. We tested AR at 20 minutes (early) after an asphyxic insult in paralyzed ventilated newborn dogs age 1-5d. CBF was measured with microspheres; cerebral perfusion pressure (CPP) was calculated as mean arterial pressure minus sagittal sinus pressure. Baseline (I) measurements in experimental (EXP n=12) and control (CON n=12) groups were followed by a series of 3 x 3 1/2 minute asphyxic insults produced by interrupting ventilation; a 5 minute recovery period separated successive insults. In EXP, AR was then tested at 20 minutes post-asphyxia during hypovolemic hypotension (II) followed by volume repletion (III); CON served as time controls. PaO_2 >80 torr and $PaCO_2$=30-45 torr for all flow studies. All results mean ± SE.

Results:		I	II	III
pH	CON	7.36 ± 0.01	7.20 ± 0.02*	7.23 ± 0.02
	EXP	7.34 ± 0.01	7.21 ± 0.01*	7.21 ± 0.1
CBF	CON	37 ± 3	42 ± 3	39 ± 3
ml/100gm/min	EXP	42 ± 4	40 ± 4	40 ± 4
CPP	CON	55 ± 3	48 ± 2*	47 ± 2
mmHg	EXP	57 ± 2	36 ± 2*†	48 ± 2*

* P<0.05 vs previous measure; †P<0.001 EXP vs CON.

Conclusion: Thus, in the early post-asphyxic period, CBF is independent of CPP. Preliminary results at 60 minutes post-asphyxia (late) shows similar results (EXP n=3, CON n=4). Overall, AR appears to be functional in the post-asphyxic period in newborn dogs.

1458 FAILURE OF ACUTE CEREBROVASCULAR STRESSES TO PRODUCE INTRAVENTRICULAR HEMORRHAGE (IVH) IN THE NEWBORN BEAGLE (NB) MODEL.

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Cerebral microvascular volume loads due to acute increases in arterial transmural pressure (ATMP) are implicated in the genesis of IVH. Previously, we have shown that such volume and pressure loads occur during the hyperemia accompanying acute recovery from asphyxia (AS) in newborn dogs, and that sagittal sinus pressure (SSP) reflects intracranial pressure at all times (CLIN. RES: 31:791, 1983).

We studied AS and volume depletion-repletion (V) in paralyzed anesthetized ventilated NBs<36hrs. AS involved temporary cessation of ventilation (7-10 min); V involved reducing MAP by 40% for 5 minutes, then rapid repletion. Mean arterial pressure (MAP) and SSP were measured, and ATMP calculated as MAP-SSP at I: baseline, II: end asphyxia (AS) or end hypotension (V), and III: peak recovery. The II-III time interval was <30 secs in AS and V. Changes in cerebral vascular volume were inferred from changes in SSP via the cranial compliance. Histopathology was done. All results as mean ± SE.

Results:	AS (n=7)			V (n=7)		
	I	II*	III*	I	II*	III*
MAP(mmHg)	39±2.6	24±4.6	61±6.6*	41±1.7	24±3.2*	61±4.5*
SSP(mmHg)	2.7±0.4	4.1±0.5*	13.6±1.3*	3.6±0.2	1.6±0.1*	8.6±0.8*
ATMP(mmHg)	37±2.6	20±4.6	47±6.1	37±1.7	22±3.2	52±5.0

p < 0.05 vs previous measure; ANOVA.

Despite marked acute ATMP and volume loads, no IVHs were seen.

CONCLUSION: Acute volume loads due to increases in ATMP may not be the cause of IVH.