121

Regulation of Cardiac Output (CO) in Sick Neonates. Heart Rate (HR) or Stroke Volume (SV)? Lindner W, Schaumberger M, Riegel K, Versmold HT*,Department of Pediatrics and Gyn. Ob.*, University of Munich, F.R.G.

Neonates are known to regulate CO by changing HR.Little is known on changes of stroke volume in sick infants.We measured CO,SV,HR and mean arterial blood pressure (BP) by Dopplersonography in 40 sick infants before(A,B) and after (A_T, B_T) therapy.(A):low CO due to respiratory problems(n=16).(B): high CO due to PDA(n=25). (C) and (D): Normal infants(n=40) matched for GA(control).In (A) SV was low and not compensated by a high HR. BP was normal.The rise of CO after treatment(A_T) was due to a rise in SV.In (B) SV was high and normalized after ductus closure (B_T), BP was low and increased,HR did not change. Conclusion: Neonates with low CO and low preload do not compensate low SV by increasing the HR. Preterm infants can increase their SV above normal levels if preload is high and afterload is low. CO in sick neonates is not only regulated by HR but also by SV. Results (mean + SD):

	n	GA wks	CO ml/min/kg	SV ml/kg	mBP mmHg	HR bpm	p <	0.01
A pre	15	33±5	166±28*	1.10±0.28*	35±11	140± 9	*=	A vs A_T
A_T post ther.			259±32	1.78±0.24	38±10	150±12	and	A vs C
B pre	25	28±2	439±63**	2.97±0.48**	30±10	149± 9		
B_T post ther.			260±29	1.71±0.22	40±12	153±12		
C	15	33±3	248±23	1.72±0.19	42±10	139±15	+=*	B vs B_T
D	25	29±2	256±20	1.74±0.18	37± 8	149±18	and	B vs D

122

CARDIAC OUTPUT IN INFANTS OF DIABETIC MOTHERS

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Cardiac output (CO) in 12 infants of diabetic mothers (IDMs) and in 20 normal neonates (control group) was estimated evaluating blood velocity in ascending aorta, using a gated pulsed Doppler velocimeter with a 5 MHz transducer positionated in the suprasternal nocth. CO (mL/min/Kg) was calculated from the equation: CO = SV · HR, where SV = stroke volume (mL/Kg) and HR = heart rate (bpm). Ventricular septal hypertrophy (IVDS > 0.35 cm) was found in 5 of 12 IDMs, but in none control group. In the group with IVDS > 0.35 cm, CO and SV were reduced at comparable HR. Reduced CO in IDMs with hypertrophic cardiomyopathy was related to lower SV, due to decreased left ventricular compliance and limited pre-load.

	Control group (n=20)	IDMs (n=12)	IDMs IVDS > 0.35 (n=5)	IDMs IVDS < 0.35 (n=7)	P
HR	140 ± 14	132 ± 16	133 ± 15	130 ± 16	NS
CO	246 ± 31	212 ± 43	179 ± 33	221 ± 41	0.001
SV	1.78 ± 0.28	1.68 ± 0.44	1.36 ± 0.34	1.87 ± 0.33	0.001

123

PULSED DOPPLER ECHOCARDIOGRAPHIC DETERMINATION OF NEONATAL CARDIAC OUTPUT USING THE APICAL WINDOW

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Cardiac output(CO) can be assessed noninvasively by range-gated pulsed Doppler echocardiography. Simultaneous two-dimensional echocardiography from the apical window allows control of position of the sample volume. To evaluate the accuracy of this method (which is well established in adults) in the neonate, ten healthy newborn infants with gestational ages of 32-36 wks and birth weights of 1670 - 2730 g were studied at postnatal ages of 2 - 10 days.
A 5 MHz mechanical transducer in the Duplex-mode (2D and M-mode/2D and pulsed Doppler) was used. The aortic cross-sectional area was calculated from the aortic root diameter measured by M-mode. Velocity of the left ventricular outflow tract was measured from the apical five-chamber-view. Stroke volume was calculated by the integral of the maximum velocity curve from spectral display obtained by discrete Fourier transformation.
Stroke volume values ranged from 2.3 ml to 6.3 ml (median 4.7 ml). CO corrected for body weight ranged from 199.7 ml/min/kg to 381.4 ml/min/kg (median 283.1 ml/min/kg). Cardiac output was higher in infants delivered vaginally than in those delivered by cesarean section (median 309.1 ml/min/kg vs. 267.0 ml/min/kg).

124

HEMODYNAMIC EFFECTS OF SUBMAXIMAL TREADMILL EXERCISE IN LAMBS WITH AN AORTOPULMONARY SHUNT (S).

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We showed that S lambs at rest could maintain their systemic blood flow (SBF) at the expense of an increased heart rate (HR). Therefore we wondered whether a diminished reserve in HR would limit the S lambs to perform strenuous exercise. We studied 6 7-week-old lambs (12±1 kg; mean±SD) with a Goretex ® graft between aorta (ao) and pulmonary artery (pa) (PBF: SBF ratio ≈3). Six lambs without graft served as controls (C). Variables were recorded at the end of a 10 min. treadmill run at 75% of predetermined VO_{2max}. During exercise HR increased more in C (108±23 to 239±31 bpm) than in S lambs (169±32 to 238±32). Total left ventricular stroke volume (SV_{lv}) did not change in both groups (S: 2.0±0.3 to 2.0±0.3; C: 1.1±0.3 to 1.1±0.1 ml/kg), but the fraction of total SV_{lv} through the shunt decreased per heart beat so that the fraction contributing to SBF (effective SV_{lv}) increased significantly (S: 0.7±0.2 to 0.9±0.3). This enabled S lambs to increase SBF (118±20 to 216±40 ml/min/kg) to such an extent that it was not significantly different from SBF in C lambs (116±10 to 260±20). We conclude that S lambs adapt well to exercise by increasing their effective SV_{lv}.

125

MAXIMAL EXERCISE AND ARTERIOVENOUS OXYGEN DIFFERENCE IN LAMBS WITH AN AORTOPULMONARY SHUNT (S).

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Systemic blood flow (SBF) is maintained by an increased heart rate (HR) in S lambs at rest (SBF: 135±42 and 128±8 ml/min/kg in S and control lambs (C), resp.; HR 189±18 and 139±30 bpm; means±SD). Maximal HR is not likely to be different in S and C lambs. Consequently SBF will be lower in S lambs during exercise at maximal aerobic power (VO_{2max}). VO_{2max} itself will be lower too unless AV oxygen difference is increased. We studied 5 7-weeks-old lambs (12±1 kg) with a Goretex ® graft between aorta (ao) and pulmonary artery (pa) (PBF: SBF ratio ≈3). Another 5 lambs of the same age and weight but without a graft served as controls. Variables were recorded at rest and at the point of exhaustion of a graded max. exercise test. HR was not significantly different during VO_{2max} (S: 284±25 bpm; C: 294±33). Consequently S lambs could not maintain SBF (285±27 ml/min/kg) at the level of C lambs (328±26; $p<0.05$). The limited ability to increase SBF was no stimulus for S lambs to increase their AV oxygen difference more than C lambs (S: 51±3 to 90±10; C: 48±3 to 95±8 $ml O_2/l$). We conclude that S lambs cannot extract more oxygen to increase their VO_2max.

126

THE ROLE OF OXYGEN AFFINITY IN THE ADAPTATION TO CHRONIC HYPOXEMIA IN 5-WEEK-OLD LAMBS.

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A decrease in oxygen affinity of hemoglobin could contribute to the adaptation to chronic hypoxemia in a right-to-left shunt. We studied P50, systemic oxygen delivery (SOD) and oxygen uptake (VO_2) in the 6th week of life in 11 chronically hypoxemic and in 14 normoxemic lambs. Hypoxemia had been experimentally induced in the 2nd week of life by means of an atrial septal defect and a pulmonary stenosis. Hypoxemic lambs had a decreased arterial oxygen saturation (66±7 vs 93±2 %, mean ± SD, $p<0.001$) and an increased hemoglobin concentration (145±14 vs 104±14 $g.l^{-1}$, $p<0.001$). Heart rate was increased (171±28 vs 143±32 beats.min^{-1}, $p<0.05$) but systemic blood flow (SBF) was not different. Also SOD (23±10 vs 23±5 $ml.min^{-1}.kg^{-1}$) and the P50 were not different (5.03±0.43 vs 5.10±0.45 kPa). VO_2 was maintained in hypoxemic lambs (9±3 vs 9±1 $ml.min^{-1}.kg^{-1}$) but mixed venous oxygen saturation ($S^{mv}O_2$) (41±9 vs 58±2 %, $p<0.001$) and oxygen tension (6.0±1.0 vs 7.2±0.5 kPa, $p<0.001$) were decreased. We conclude that P50 does not change in chronically hypoxemic lambs. These results suggest that the minimally attainable $S^{mv}O_2$ will be equal in hypoxemic and control lambs so the amount of oxygen that can be unloaded will be decreased in hypoxemia. We speculate that the ability to increase oxygen-extraction and SBF is limited in hypoxemia. This will impair the ability to meet increased demands for oxygen.