Metabolic rate of neonates with congenital heart disease

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Krauss, A. N., and Auld, P. A. M. (1975). *Archives of Disease in Childhood*, **50**, 539. **Metabolic rate of neonates with congenital heart disease.** Seven infants under one month of age with controlled congestive heart failure showed a mean oxygen consumption of 9.4 ± 1.6 SD ml/kg per min, a mean respiratory quotient of 0.71 ± 0.05 SD, and a mean metabolic rate of 63 ± 12 SD cal/kg per 24 h. This compares with a group of infants with congenital heart disease not in heart failure with $\dot{V}_{O_2}$ of 6.5 ± 1.2 SD ml/min per kg, respiratory quotient of 0.80 ± 0.11 SD, and basal metabolic rate of 45 ± 8 SD cal/kg per 24 h. These differences are significant ($P < 0.001$). The findings of a greater metabolic rate associated with congestive heart failure are thus extended to the newborn period.

Recent studies have shown that undergrown newborn infants have a raised metabolic rate when compared to those normally grown (Jonxis *et al.*, 1968; Sinclair and Silverman, 1966). Infants with congenital heart disease have also been found to have raised metabolic rates in proportion to their degree of growth retardation and heart failure (Lees *et al.*, 1965; Pittman and Cohen, 1964; Stocker *et al.*, 1972). Only a few of these observations have been made on infants under one month of age. The present study was undertaken to provide information about the metabolic requirements of neonates with congenital heart disease during this critical period.

Methods

Fifteen infants ranging in birthweight from 1.25 to 4.8 kg were studied at ages ranging from 9 h to 60 d. 5 infants were studied twice, the remainder once each. The diagnosis of congenital heart disease was made on the basis of heart murmurs, abnormal electrocardiogram or chest x-ray, or cyanosis without pulmonary disease. Profuse sweating was not observed. 12 patients underwent cardiac catheterization. 7 of these were diagnosed as having congestive heart failure on the basis of oedema, tachypnoea, and hepatosplenomegaly. These infants all received digitalis and diuretics before study, with reduction in clinical evidence of failure. 3 infants were studied after a balloon atrial septostomy (Rashkind procedure). Clinical details are presented in the Table.

Metabolic rate was determined by diaferometry using a Kipp-Noyons MG-4 diaferometer calibrated with

O_2 and CO_2 at known flow rates. Infants were studied within one hour of feeding in an environment of $34-46^\circ C$. All were breathing room air when studied. Measurements of O_2 consumption ($\dot{V}_{O_2}$) and CO_2 production ($\dot{V}_{CO_2}$) were recorded for at least 20 min. Results presented in the Table are the average of 4 or more readings during this time period. Respiratory quotient (RQ) was calculated as the ratio of mean $\dot{V}_{CO_2}$ /mean $\dot{V}_{O_2}$. Cal/24 h per kg were calculated using standard values for the caloric equivalent of O_2 at a particular RQ (Lusk, 1924).

Results

The mean $\dot{V}_{O_2}$ of neonates with congestive heart failure and congenital heart disease is 9.2 ± 1.5 SD ml/min per kg. This represents a basal caloric expenditure of 62 ± 11 SD cal/kg per 24 h. For neonates with congenital heart disease not in congestive heart failure the comparable values are $\dot{V}_{O_2}$ of 6.5 ± 1.2 ml/min per kg and metabolic rate of 45 ± 7 cal/kg per 24 h. These differences are significant ($\dot{V}_{O_2}$, $P < 0.001$; cal, $P < 0.001$). The mean RQ of infants in failure was 0.71 ± 0.05 SD, while those without failure had a mean RQ of 0.80 ± 0.10 ($P = 0.05$). All 7 infants in congestive heart failure were studied at 11 days of age or later, while only 2 neonates not in failure were studied beyond the age of 10 days.

Normal infants over 1500 g at birth studied in this laboratory under similar conditions have a mean $\dot{V}_{O_2}$ of 6.3 ± 2.1 SD ml/min per kg at 1-4 days of age, 7.6 ± 1.7 ml/min per kg at one week,

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Case no.	Birthweight (kg)	Gestational age (w)	Study age (d)	Study weight (kg)	Surface area (m ²)	$\dot{V}_{CO_2}$ (ml/min)
<i>Congestive heart failure</i>						
1	1.94	39	25	1.85	0.140	12.3
2			30	1.95	0.143	18.1
3	2.47	40	24	2.81	0.190	20.3
4	3.26	40	11	2.95	0.194	15.5
5	3.50	40	14	2.83	0.189	24.4
6	2.38	40	18	2.40	0.172	14.0
7			22	2.49	0.175	13.9
8	2.55	40	13	2.49	0.173	12.8
9			16	2.42	0.173	18.2
10	2.90	40	15	3.00	0.206	15.2
<i>No congestive heart failure</i>						
11	4.48	40	3	4.08	0.210	16.4
12			7	4.18	0.210	19.2
13	1.25	31	60	1.90	0.147	10.8
14	2.50	40	1/3	2.50	0.171	14.4
15	4.25	40	10	3.94	0.229	18.5
15	3.08	39	2	3.18	0.204	14.1
17	1.39	30	24	1.42	0.122	8.8
18	2.88	40	2	2.75	0.170	14.3
19			5	2.84	0.172	15.4
20	3.22	40	2	3.23	0.207	16.3

*Mean $\pm$ SD; PS, pulmonary stenosis; PDA, persistent ductus arteriosus; VSD, ventricular septal defect.

and 8.6 ± 0.6 ml/min per kg at 2 weeks. Corresponding metabolic rates in cal/kg per 24 h are 44 ± 13 SD, 53 ± 10 , 60 ± 8 (Krauss and Auld, 1969).

Discussion

The results obtained in this study for O_2 consumption and metabolic rate of infants with congenital heart disease not in congestive heart failure are similar to those obtained in this laboratory for healthy neonates of the same age (Krauss and Auld, 1969). The findings of an increased rate in neonates with congestive heart failure confirms the findings of other investigators in older infants, children, and adults, and thus allows these observations to be extended to the newborn period (Brasel, 1968; Lees *et al.*, 1965; Stocker *et al.*, 1972). Though all infants with congestive heart failure showed poor weight gain, demonstrated by their failure to exceed birthweight as late as 30 days of age, the present study cannot answer the question as to the source of growth failure in congenital heart disease. As in all previous studies, the groups reported here are heterogeneous with respect to

cyanosis, respiratory distress accompanying heart failure, and the severity of the congenital heart disease. Both increased myocardial work and the increased work of breathing may contribute to raised metabolic rate in congenital heart disease (Pittman and Cohen, 1964). Cyanosis shows no constant relation to metabolic rate (Stocker *et al.*, 1972), though severe cyanosis in association with metabolic acidosis has been correlated with a decreased $\dot{V}_{O_2}$ (Levison, Delivoria-Papadopoulos and Swyer, 1965). Excessive sweating, which may contribute to heat loss, has also been reported in some infants with congenital heart disease (Taussig, 1960). Serial studies of relatively homogeneous groups of patients are needed to determine the causes of growth failure in congenital heart disease.

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REFERENCES

- Brasel, J. A. (1968). Oxygen consumption and growth. *Human Growth*, p. 474. Ed. by D. B. Check. Lea and Febiger, Philadelphia.

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$\dot{V}O_2$ (ml/min)	Respiratory quotient ($\dot{V}CO_2/\dot{V}O_2$)	$\dot{V}O_2$ (ml/kg per min)	Cal/kg per 24 h	Diagnosis
18.1	0.68	9.8	64	VSD
23.0	0.79	11.7	81	
27.4	0.74	9.8	67	PS, PDA, congenital rubella
24.1	0.64	8.2	53	Transposition, VSD, coarctation
31.4	0.78	11.1	76	Tricuspid atresia
20.2	0.69	8.4	54	Single ventricle
20.3	0.68	8.1	52	
17.5	0.73	7.0	48	Atrioventricular canal, PDA
24.9	0.73	10.3	70	
23.5	0.63	7.8	52	Coarctation aorta
	$0.71 \pm 0.05^*$	$9.2 \pm 1.5^*$	$62 \pm 11^*$	
27.3	0.60	6.7	44	Transposition of great vessels
23.5	0.82	5.6	39	
13.5	0.80	7.1	49	Fallot's tetralogy
18.8	0.76	7.5	51	VSD
20.7	0.90	5.3	38	Transposition of great vessels
15.9	0.89	5.2	37	VSD, Down's syndrome
12.6	0.70	8.9	60	PDA
15.1	0.94	5.5	39	Single ventricle, pulmonary atresia
18.5	0.83	6.5	45	
20.4	0.80	6.3	44	Transposition of great vessels
	$0.80 \pm 0.10^*$	$6.5 \pm 1.2^*$	45 ± 7	
	t = 2.646	t = 4.200	t = 3.991	
	P = 0.05	P < 0.001	P < 0.001	

- Jonxis, J. H. P., Van der Vlugt, J. J., de Groot, C. J., Boersma, E. R., and Meijers, E. D. K. (1968). The metabolic rate in premature, dysmature and sick infants in relation to environmental temperature. *Aspects of Prematurity and Dysmaturity*, p. 201. Ed. by J. H. P. Jonxis, H. K. A. Visser, and J. A. Troelstra. Stenfert Kroese, Leiden.
- Krauss, A. N., and Auld, P. A. M. (1969). Metabolic requirements of low-birth-weight infants. *Journal of Pediatrics*, **75**, 952.
- Lees, M. H., Bristow, J. D., Griswold, H. E., and Olmsted, R. W. (1965). Relative hypermetabolism in infants with congenital heart disease and undernutrition. *Pediatrics*, **38**, 183.
- Levison, H., Delivoria-Papadopoulos, M., and Swyer, P. R. (1965). Variations in oxygen consumption in the infant with hypoxaemia due to cardiopulmonary disease. *Acta Paediatrica Scandinavica*, **54**, 369.
- Lusk, G. (1924). Animal calorimetry: analysis of the oxidation mixtures of carbohydrate and fat. *Journal of Biological Chemistry*, **59**, 41.

- Pittman, J. G., and Cohen, P. (1964). The pathogenesis of cardiac cachexia. *New England Journal of Medicine*, **271**, 403.
- Sinclair, J. C., and Silverman, W. A. (1966). Intrauterine growth in active tissue mass of the human fetus, with particular reference to the undergrown baby. *Pediatrics*, **38**, 48.
- Stocker, F. P., Wilkoff, W., Miettinen, O. S., and Nadas, A. S. (1972). Oxygen consumption in infants with heart disease. *Journal of Pediatrics*, **80**, 43.
- Taussig, H. B. (1960). *Congenital Malformations of the Heart*, revised ed., vol. II. pp. 697, 915, 926. Harvard University Press, Cambridge, Massachusetts.

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