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 $9.4 \pm 1.6$ SD ml/kg per min, a mean respiratory quotient of $0.71 \pm 0.05$ SD, and a mean metabolic rate of $63 \pm 12$ SD cal/kg per 24 h. This compares with a group of infants with congenital heart disease not in heart failure with $V_{O_2}$ of $6.5 \pm 1.2$ SD ml/min per kg, respiratory quotient of $0.80 \pm 0.11$ SD, and basal metabolic rate of $45 \pm 8$ 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°C. All were breathing room air when studied. Measurements of $O_2$ consumption ($V_{O_2}$) and $CO_2$ production ($V_{CO_2}$) 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 $V_{CO_2}$/mean $V_{O_2}$. 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 $V_{O_2}$ of neonates with congestive heart failure and congenital heart disease is $9.2 \pm 1.5$ SD ml/min per kg. This represents a basal caloric expenditure of $62 \pm 11$ SD cal/kg per 24 h. For neonates with congenital heart disease not in congestive heart failure the comparable values are $V_{O_2}$ of $6.5 \pm 1.2$ ml/min per kg and metabolic rate of $45 \pm 7$ cal/kg per 24 h. These differences are significant ($V_{O_2}$, $P<0.001$; cal, $P<0.001$). The mean RQ of infants in failure was $0.71 \pm 0.05$ SD, while those without failure had a mean RQ of $0.80 \pm 0.10$ ($P = 0.05$). 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 $V_{O_2}$ of $6.3 \pm 2.1$ SD ml/min per kg at 1–4 days of age, $7.6 \pm 1.7$ ml/min per kg at one week,

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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 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

### Metabolic Rate of Neonates with Congenital Heart Disease

**Respiratory (ml/kg per 24 h)**

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>V̇O₂ (ml/kg per 24 h)</th>
<th>Cal/kg per 24 h</th>
<th>Respiratory quotient (V̇O₂/V̇O₂)</th>
</tr>
</thead>
<tbody>
<tr>
<td>VSD</td>
<td></td>
<td></td>
<td>20-4 0.71 ± 0.05*</td>
</tr>
<tr>
<td>PS, PDA, congenital rubella</td>
<td>20-5 0.80</td>
<td>44</td>
<td>20-4 0.80 ± 0.10*</td>
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<td>Transposition, VSD, coarctation</td>
<td>20-5 0.80</td>
<td>44</td>
<td>20-4 0.80 ± 0.10*</td>
</tr>
<tr>
<td>Tricuspid atresia</td>
<td>20-5 0.80</td>
<td>44</td>
<td>20-4 0.80 ± 0.10*</td>
</tr>
<tr>
<td>Single ventricle</td>
<td>20-5 0.80</td>
<td>44</td>
<td>20-4 0.80 ± 0.10*</td>
</tr>
<tr>
<td>Atrioventricular canal, PDA</td>
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<td>20-4 0.80 ± 0.10*</td>
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<td>Coarctation sorta</td>
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<td>44</td>
<td>20-4 0.80 ± 0.10*</td>
</tr>
<tr>
<td>Fallot's tetralogy</td>
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<td>Single ventricle, pulmonary atresia</td>
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<td>44</td>
<td>20-4 0.80 ± 0.10*</td>
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<tr>
<td>Transposition of great vessels</td>
<td>20-5 0.80</td>
<td>44</td>
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</tbody>
</table>

**References**


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