Malnutrition and growth failure in cyanotic and acyanotic congenital heart disease with and without pulmonary hypertension

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Abstract

***Aim*—To investigate the effect of several types of congenital heart disease (CHD) on nutrition and growth.**

***Patients and methods*—The prevalence of malnutrition and growth failure was investigated in 89 patients with CHD aged 1–45 months. They were grouped according to cardiac diagnosis: group aP (n = 26), acyanotic patients with pulmonary hypertension; group ap (n = 5), acyanotic patients without pulmonary hypertension; group cp (n = 42), cyanotic patients without pulmonary hypertension; and group cP (n = 16), cyanotic patients with pulmonary hypertension. Information on socioeconomic level, parental education status, birth weight and nutrition history, number of siblings, and the timing, quality, and quantity of nutrients ingested during weaning period and at the time of the examination were obtained through interviews with parents.**

***Results*—There was no significant difference between groups in terms of parental education status, socioeconomic level, duration of breast feeding, and number of siblings ($p > 0.05$). Group cP patients ingested fewer nutrients for their age compared to other groups. 37 of the 89 patients were below the 5th centile for both weight and length, and 58 of 89 patients were below the 5th centile for weight. Mild or borderline malnutrition was more common in group aP patients. Most group cp patients were in normal nutritional state, and stunting was more common than wasting. Both moderate to severe malnutrition and failure to thrive were more common in group cP patients.**

***Conclusion*—Patients with CHD are prone to malnutrition and growth failure. Pulmonary hypertension appears to be the most important factor, and cyanotic patients with pulmonary hypertension are the ones most severely affected. This study shows the additive effects of hypoxia and pulmonary hypertension on nutrition and growth of children with CHD.**

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Keywords: congenital heart disease; pulmonary hypertension; cyanosis; malnutrition; growth failure

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Infants with congenital heart disease (CHD) are prone to malnutrition for several reasons including decreased energy intake, increased energy requirements, or both.[1] The severity of malnutrition can range from mild undernutrition to failure to thrive. This can have a notable effect on the outcome of surgery, increasing morbidity and mortality. Different types of cardiac malformations can affect nutrition and growth to varying degrees. We investigated the effect of several types of cardiac malformations on nutrition and growth, and reviewed the literature in this respect. The separate effects of hypoxia and pulmonary hypertension on nutrition and growth of children have been investigated widely in several studies, but to our knowledge there are not enough data on patients with both cyanosis and pulmonary hypertension.

Patients and methods

Eighty nine patients admitted to our hospital for cardiac catheterisation between February 1996 and January 1997 were included in this study. Their ages ranged from 1–45 months. Patients with a history of prematurity, intrauterine growth retardation, known genetic malformations, dysmorphic features, and neurologic disability were excluded. All patients' cardiac diagnoses were made on the basis of clinical and laboratory examinations including telecardiography, electrocardiography, and echocardiography. These were confirmed by cardiac catheterisation, angiography, or both, in 86 of the 89 patients. Patients were assigned to four groups according to their diagnosis: group aP, acyanotic patients with pulmonary hypertension (patients with left to right shunt and pulmonary hypertension); group ap, acyanotic patients without pulmonary hypertension; group cp, cyanotic patients without pulmonary hypertension; and group cP, cyanotic patients with pulmonary hypertension. Information on socioeconomic level (monthly income), education status of the parents, patients' birth weight, nutrition history (duration of breast feeding, the timing, quality, and quantity of nutrients during the weaning period, and quality and quantity of feeding at the time of examination), and number of siblings were obtained through an interview with the parents. The age appropriateness of the nutrients consumed by each patient was determined by two paediatricians independently, and labelled as "enough" or "not enough". Patients were subjected to a complete physical examination, checking their weight, length, head circumference, and for physical signs of malnutrition, such as skin lesions, and thin and weak hair. Standardised measure-

Table 1 Cardiac diagnoses

Diagnosis	*n*
Group aP	26
VSD	14
PDA	4
VSD + PDA	1
Complete AVSD + PDA	1
Complete AVSD	3
VSD + coarctation of aorta + subaortic stenosis	1
VSD + PDA + AS + MS	1
ASD + PAPVC	1
Group ap	5
Partial AVSD + left AV valve insufficiency	1
AS, bicuspid aortic valve	1
VSD	2
ASD	1
Group cp	42
TGA without PS	5
TGA with PS	4
TF	22
Complex cardiac disease	6
VSD + PA	4
Valvular PS + PFO	1
Group cP	16
DORV + VSD + coarctation of the aorta	1
TGA + VSD with/without PDA	4
Complex cardiac disease	6
VSD + ASD + pulmonary vascular disease	1
DORV + PDA + APW + DSM	1
TAPVC + ASD	2
Truncus arteriosus + VSD	1

VSD, ventricular septal defect; PDA, patent ductus arteriosus; AVSD, atrioventricular septal defect; AS, aortic stenosis; MS, mitral stenosis; ASD, atrial septal defect; PAPVC, partial anomalous pulmonary venous connection; AV, atrioventricular; TGA, transposition of great arteries; PS, pulmonary stenosis; TF, tetralogy of Fallot; PA, pulmonary atresia; PFO, patent foramen ovale; DORV, double outlet right ventricle; APW, aorticopulmonary window; DSM, discrete subaortic membrane; TAPVC, total anomalous pulmonary venous connection.

ments of weight, length, and head circumference were made by one nurse. Malnutrition was described as mild, moderate, or severe when patient weight was 80–90%, 70–80%, and < 70% of ideal weight for length, respectively. When both weight and length were below the 5th centile for age, the condition was described as "failure to thrive". Pulmonary artery pressure, pulmonary vascular resistance index, aortic oxygen saturation, haemoglobin, albumin, and venous pH were measured. Data on cardiac status included presence of cyanosis, pulmonary hypertension, and congestive heart failure. Measurements of pulmonary artery pressure, pulmonary vascular resistance index, and aortic oxygen saturation were available for the patients who underwent cardiac catheterisation. Mean pulmonary arterial pressure above 25 mm Hg was considered to indicate pulmonary hypertension. Table 1 shows the patients' cardiac diagnoses.

Table 2 Patient characteristics

	Groups			
	aP (n = 26)	*ap (n = 5)*	*cp (n = 42)*	*cP (n = 16)*
Age (months)	15.5 (10.6)	27.4 (14.2)	17.9 (11.1)	14.5 (3.8)
(range)	(4–40)	(12–45)	(1–42)	(2–39)
Birth weight (g)	3099 (627)	3060 (386)	3111 (543)	2975 (472)
Weight (kg)	7.6 (2.4)	10.7 (2.9)	8.5 (2.7)	6.8 (3.6)
Length (cm)	73.3 (9.2)	87.0 (12.4)	74.3 (11.3)	69.0 (11.7)
% of IBW for length	81 (8)	87 (5)	90 (11)	83 (20)
Albumin (g/dl)	3.7 (0.5)	3.7 (0.6)	3.5 (0.5)	3.6 (0.5)
Venous pH	7.34 (0.06)	7.36 (0.03)	7.29 (0.05)	7.30 (0.08)
Haemoglobin (g/l)	111 (13)	108 (15)	140 (24)	119 (27)
Mean pulmonary artery pressure (mm Hg)	46.5 (13.9)	22.0 (4.2)	14.8 (5.5)	54.8 (14.8)
PVRi (U)	4.2 (1.9)	1.0 (0.5)	1.3 (1.1)	5.6 (2.8)
Aortic oxygen saturation (%)	90.5 (6.9)	92.1 (4.3)	59.5 (18.9)	61.6 (17.7)

Values are mean (SD).
IBW, ideal body weight; PVRi, pulmonary vascular resistance index.

STATISTICAL ANALYSIS

Comparisons were made between the four patient groups. All were entered into a database management system and were analysed by the SPSS/PC 6.0 computer software program using the Mann-Whitney U, Wilcoxon rank sum W, and χ^2 tests. Differences were considered significant at $p < 0.05$.

Results

The 89 patients had a mean (SD) age of 17.1 (11.8) months. Several characteristics of the patients are shown in table 2.

There was no significant difference between groups in terms of mean age, parental education status, socioeconomic level (monthly income), birth weight, number of siblings, duration of breast feeding, and number of patients in each group with age appropriate feeding in terms of quality ($p > 0.05$). Forty nine per cent of all patients were reported to have inadequate nutrient intake caused by propensity for fatigue or tachypnoea. Low family socioeconomic level may have contributed to this; 73% of all the families were in this category. However, there was no significant difference among the groups in this respect. More patients in group cP had less nutrient intake for their age than the other groups, and the difference was significant ($p = 0.014$). Only 3 of 16 patients ingested an appropriate quantity for their age. Physical signs of malnutrition were present in 15 of 89 patients (16.9%): seven in group cP ($p = 0.04$), four in group cp, and four in group aP. Eleven of these 15 patients failed to thrive, and 10 had moderate to severe malnutrition.

Of the 89 patients, 58 (65.2%) were below the 5th centile for weight, 37 (41.6%) were below the 5th centile for both weight and height, and 24 (27%) were < 80% of their ideal body weight for length (moderate to severe malnutrition). Moderate to severe malnutrition was more common in group cP (9 of 16 patients) ($p = 0.002$). Thirty two patients (36%) had mild malnutrition. The malnutrition observed in groups aP and cp was most often mild, seen in 12 of 26 (46%) and 13 of 42 (31%) patients, respectively. There was failure to thrive in 11 of 26 (42%), 2 of 5 (40%), 17 of 42 (40%), and 9 of 16 (56%) patients in groups aP, ap, cp, and cP, respectively (table 3). Fifty two per cent of all patients were below the 5th centile in length for age (stunting). Eleven of 26 patients in group aP (42%), 2 of 5 in group ap (40%), 20 of 42 in group cp (48%), and 11 of 16 in group cP (69%) were stunted. Stunting was more common in group cP than other groups. Failure to thrive was also significantly more prevalent in group cP ($p = 0.043$). Most of the patients with short stature were also below the 5th centile in weight for age. There was no significant difference between groups aP and cP in terms of severity of pulmonary hypertension (mean pulmonary artery pressure 46.5 (13.9) and 54.8 (14.8) mm Hg, respectively, $p = 0.07$), and there was no significant

Table 3 Nutrition and growth status

	Groups			
	aP (n = 26)	*ap (n = 5)*	*cp (n = 42)*	*cP (n = 16)*
Nutrition				
Normal	4	2	24	3
Mild malnutrition	12	3	13	4
Moderate malnutrition	8	0	3	5
Severe malnutrition	2	0	2	4
Failure to thrive	11	2	17	9

difference between groups cp and cP in terms of aortic oxygen saturation (59.5 (18.9)% and 61.6 (17.7)%, respectively, p = 0.69).

Discussion

It is well known that malnutrition accompanies and contributes to morbidity in CHD. Controversy exists regarding the relative roles of low caloric intake, type of cardiac lesion, malabsorption, and hypermetabolism.[1–4] Patients with CHD and cyanosis, pulmonary hypertension, and congestive heart failure appear to have an increased prevalence of growth failure and malnutrition.[1 4–6] Optimising nutritional status improves surgical outcome and contributes to reduced morbidity. In a large survey of 890 children with various CHD, 55% were below the 16th centile for height, 52% were below the 16th centile for weight, and 27% were below the 3rd centile for both length and weight.[7] In our study malnutrition appears to be more prevalent and more severe, as 65% of the children were below the 5th centile for weight, and 41% were below the 5th centile for both weight and height. Fifty six of 89 patients (63%) were underweight for their length. This might have been because most of the patients referred to our hospital had severe cardiac lesions and were from families of a low socioeconomic level. As shown in table 3 chronic malnutrition, which affects both weight and length, is also an important problem in CHD.

The cause of growth retardation in CHD is multifactorial.[1 3 4 8] Inadequate caloric intake, malabsorption, and increased energy requirements caused by increased metabolism may all contribute. However, inadequate caloric intake appears to be the most important cause of growth failure in CHD.[1 3] A characteristic feeding pattern of children with CHD is defined, with a large variation in caloric intake.[1] When heart failure is mild the infant commonly overfeeds, and fluid and sodium overload disturb cardiac haemodynamics, leading to decompensation of heart failure and decreased intake. As a result, the individual's overall nutrient intake is inadequate. Arterial blood gas analysis of patients with congestive heart failure commonly reveals normal values, but a form of "stagnant anoxia" caused by sluggish capillary blood flow within the tissues, which leads to cellular hypoxia, occurs in congestive heart failure.[4] Anorexia also accompanies malnutrition and further compromises the patient's condition. Dyspnoea and tachypnoea in patients with congestive heart failure lead to propensity for fatigue and decreased intake. Chronic hypoxia is reported to affect growth. Hypoxic hypoxia has been shown to cause anorexia in experimental rats, along with a concomitant decrease in body weight.[9] Chronic hypoxia may contribute to the feeding problem in cardiac patients. Malabsorption is also thought to play a role in cardiac cachexia.[4] It can result from both congestive heart failure and oxygen lack. We did not investigate occurrence of malabsorption in our patients. Children with CHD are known to be in a hypermetabolic state.[10] Heart disease causes an increase in cardiac and respiratory work. Decreased intake caused by anorexia combined with increased respiratory effort results in a greater nutrient deficit. Children with heart disease may need as much as 50% more calories than normal children in order to achieve normal growth.[1] A combination of these factors predisposes the infant to malnutrition and growth failure.

Previous studies have investigated the effect of cardiac lesion type on growth and nutrition, and several have reported that degree of cyanosis is not correlated to severity of growth impairment.[1 6] However, degree of growth impairment was found to be closely associated with severity of the haemodynamic impairment.[1 11 12] Linde and colleagues[6] found a more pronounced retardation in both height and weight in children with cyanosis than in those with acyanotic heart disease, but did not mention the effect of pulmonary hypertension. In contradiction to the report by Linde *et al*, Salzer and colleagues[5] showed that infants with left to right shunt tended to gain less weight and to be leaner than those with cyanotic heart disease. In our study most of the cyanotic patients without pulmonary hypertension were of normal weight for their length or were mildly malnourished (88%). However, 48% were stunted and 40% failed to thrive. Thus, stunting appears to be more common than wasting in cyanotic heart disease without pulmonary hypertension. We also investigated the impact of pulmonary hypertension on growth and nutrition in cyanotic heart disease, and found that cyanotic patients with pulmonary hypertension were the most severely affected group, 81% having malnutrition, 56% having moderate to severe malnutrition, and 56% failing to thrive. Nutrition history gave the impression that these patients consumed less nutrients than the others. Most group cP patients had compensated metabolic acidosis caused by hypoxia, and all had pulmonary hypertension. Both of these conditions can contribute to respiratory difficulty and tachypnoea, and thus limit nutrient intake. In addition, chronic hypoxia, as discussed previously, is an important factor in anorexia and inefficient processing of nutrients at the cellular level.[4 9]

Delay in surgical repair of congenital heart lesions can lead to worsening of nutrition and growth status of patients. Several reports have documented encouraging results of early repair of critical congenital heart defects in symptomatic neonates and infants rather than palliative operations[13] and of primary surgical closure of large ventricular septal defects.[14 15]

It is well documented that malnutrition most commonly affects patients with pulmonary hypertension, and the results of our study also support this. Hypoxia and pulmonary hypertension were investigated separately for their effect on nutrition and growth in previous studies. As evidenced by our investigation, stunting is more common than wasting in cyanotic heart disease. Additionally, we also showed that patients with cyanotic heart disease accompanied by pulmonary hypertension were most severely affected in terms of nutrition and growth. Both moderate to severe malnutrition and failure to thrive were more common in this group. Severity of their cardiac lesions and malnutrition put this group of patients at greater risk for operative morbidity and mortality. Thus, a more intensive nutritional treatment and early corrective surgery should be considered to optimise the outcome.

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