ORIGINAL ARTICLE

Physical Growth of Children with Ventricular Septal Defect

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ABSTRACT We conducted a prospective study on children with ventricular septal defect (VSD) for assessing the physical growth status, establishing the determinants of growth, and determining the effect of natural history on growth. There were 46 VSD patients and 30 controls aged 1-5 years. We divided the subjects into two groups; group A consisted of 32 VSD patients and 16 controls aged 12-35 months, group B comprised 14 VSD patients and 14 controls aged of 36-60 months. A simple hemodynamic scoring system was created to determine the correlation between physical growth and severity of hemodynamic alteration, using 10 findings based on history, physical, and non-inavsive examinations. Body weight and height, and arm circumference were measured every 3 months up to 12 months. The growth status correlated well with the hemodynamic scores. Body weight and arm circumference were more affected than body height. Physical growth disturbance was observed in high score patients at the beginning, and became more evident at the end, of the study. In low score patients arm circumference was slightly affected at the beginning of the study, while body weight was slightly disturbed after 9 months of observation. **[Paediatr Indones 1994; 34:16-25]**

Introduction

All isolated ventricular septal defects (VSD) can be classified into four main groups according to their hemodynamic presentation:[1,2] (1) isolated small VSD with normal pulmonary vascular resistance, (2) moderate VSD with normal pulmonary vascular resistance, (3) large VSD with dynamic pulmonary hypertension, and (4) large VSD with pulmonary hypertension and elevated pulmonary vascular resistance.

Infant and children with VSD frequently have subnormal growth related to physiological severity and age.[3] Normal physical growth is usually seen only

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in isolated small VSD with normal vascular resistance, while in the other three groups some degree of growth disturbance has been reported.[4-6] The important determinants of physiological severity in VSD are several hemodynamics alteration, i.e., : (1) large left to right shunt; (2) congestive heart failure; (3) pulmonary vascular obstruction.[3] Although these three determinants can only be precisely calculated by cardiac catheterization, they can be predicted from clinical, chest X-ray, ECG, and echocardiographic findings.[1,2] Several authors have used those non-invasive findings for making consideration and further work-up.[1,2,7]

The aim of the present study was to: (1) assess the growth status of patients with VSD; (2) establish the determinants of growth using a simple scoring system; (3) determine the effect of duration of illness on growth; and (4) determine the effect of medical treatment.

Methods

The subjects studied were 45 patients with isolated VSD treated at the Outpatient Clinic, Cardiology Division, Department of Child Health, Cipto Mangunkusumo Hospital, Jakarta, Indonesia. Thirty healthy children attending the same hospital served as control. The study was conducted from July 1, 1987 to October 31, 1987.

The inclusion criteria for VSD patients were: (1) established diagnosis of VSD by history, physical examination, electrocardiography, chest X-ray and echocardiography, and (2) age 1-5 years. The exclusion criteria were: (1) presence of associated cardiac and extracardiac anomalies, (2) associated chronic infectious diseases, and (3) prolonged corticosteroid treatment.

This prospective study was conducted by observing all study subjects for one year. To determine the influence of the natural history of VSD, patients were categorized into 2 groups according to their age. Group A consisted of 12-35 month old patients with VSD and their controls, while group B comprised 36-60 month old patients with VSD and their controls.

For the purpose of the study, a simple hemodynamic scoring system was created to establish the association between the growth status and the severity of hemodynamic alteration in VSD. Previous scoring system proposed by Rilantono[7] was considered inappropriate, because her scoring system used body weight as one of the determinants. This hemodynamic scoring system used 10 determinants, 1 score for each item: (1) feeding difficulties and (2) recurrent respiratory infection obtained from history, (3) congestive heart failure and (4) pulmonary hypertension obtained from physical examination; (5) left ventricular hypertrophy and (6) right ventricular hypertrophy from electrocardiogram; (7) increased vascular markings and (8) car- diomegaly from the chest X-ray; and (9) increased left ventricular dimension and (10) left atrial enlargement from the 2-D echocardiogram. Normal values of the electrocardiogram, chest X-ray and echocardiogram in children have been discussed elsewhere.[2] Increased pulmonary vascular markings was considered to be present when on chest X-ray the diameter of the right descending pulmonary artery exceeded the diameter of the trachea.[8]

The total score ranged from 0 to 10 (Table 1). VSD patients with total score of 0-5 were categorized in group I, presumed as small ventricular septal defect with mild to moderate hemodynamic changes, while patients with total score of 6-10 were categorized in group II, presumed as moderate to large ventricular septal defect with moderate to severe hemodynamic alteration.

Table 1. Scoring system using history, physical examination, electrocardiogram, chest X-ray, and echocardiogram as determinants of the severity of hemodynamic alteration in VSD

Determinant	Score
History	
- feeding difficulties	1
- recurrent respiratory infection	1
Physical examination	
- congestive heart failure	1
- pulmonary hypertension (P_2 >)	1
Electrocardiogram	
- left ventricular hypertrophy	1
- right ventricular hypertrophy	1
Chest X-ray	
- increased vascular markings	1
- cardiomegaly	1
Echocardiogram	
- increased LVID	1
- increased LA/Ao	1
Total hemodynamic score	10

LVID = left ventricular internal dimension; LA = left atrium; Ao = aorta.

Instead of the gestational age at birth, birth weight and length were obtained from the medical record. The determinants of the physical growth status in every case were the percentage of P50 Havard of body weight, body height, and upper arm circumference, as proposed by the others.[9,10] The assessment of the physical growth status was performed five times: at the beginning of the study, then after three, six, nine, and twelve months of observation.

Data were presented in narrative and tabular forms. Student t test and Fisher' exact test were used for statistical analyses. The significant level was $p < 0.05$.

Results

Birth weight was obtained in 43 VSD patients and 30 controls, respectively. There was no statistical difference of birth weight between these two groups. The body length at birth was available in 30 VSD patients and 30 controls. There was no statistically difference of birth length between the two groups (Table 2).

The age distribution of VSD patients and controls was depicted in Table 3. Group A consisted of 32 younger patients with VSD and 16 healthy younger children with the age range from 12 to 35 months, while group B consisted of 14 patients with VSD and 14 healthy children aged 36 to 60 months.

The disribution of VSD patients and controls according to the physical growth status is shown in Table 4. Subnormal body weight, body height, and upper arm circumference were detected in 25 (54.3%), 22 (47.8%) and 26 (56.5%) of VSD patients, respectively. Body weight and upper arm circumference of VSD patients were more affected than their body height. None of the controls had abnormal physical growth status.

Table 2. Comparison of birth weight and length of both groups

	VSD		Control		
	n	x (SD)	n	x (SD)	*p*
BW (kg)	43	3,03(1,37)	30	3,12(0,39)	> 0,05
BL(cm)	30	49,0(1,41)	30	49,1(1,58)	> 0,05

BW = body weight; BL = body length; n = number of subjects; x = mean; SD = standard deviation.

Table 3. Age distribution of patients with VSD and controls

Group	Age (mo)	VSD	Control	Total
A	12-35	32	16	48
B	36-60	14	14	28
Total		46	30	76

Table 4. Comparison between growth status parameters in both groups

	VSD	Control	p
Body weight			
normal	21	30	< 0,01
subnormal	25	0	
Body height			
normal	24	30	< 0,01
subnormal	22	0	
UAC			
normal	20	30	< 0,01
subnormal	26	0	

UAC = Upper arm circumference

Table 5. Association between physical growth status and hemodynamic score

Score	Body weight		Body height		UAC	
	N*	SN*	N	SN	N	SN
0 - 5	18	5	18	5	20	3
6 - 10	3	20	6	17	7	16
Total	21	25	24	22	27	19
X^2	17,17 *p* < 0,05		10,54 *p* < 0,05		12,91 *p* < 0,05	

* N = normal; SN = subnormal.

The association between growth status and the severity of the hemodyamic alteration using simple hemodynamic scoring system is shown in Table 5. In low score VSD patients, subnormal body weight, body height, or upper arm circumference was detected in 21.7%, 21.7% and 15.0%, respectively. On the other hand in high score VSD patients, subnormal body weight, body height, or upper arm circumference was detected in 86.9%, 73.9% and 69.6%, respectively. The growth status of the 23 high score VSD patients was significantly different with that of 23 low score VSD patients. Correlations between hemodynamic scores and body weight, height, and upper arm circumference of VSD patients were shown in Figures 1, 2, and 3. It is clear that weight, height, and upper arm circumference were well negatively correlated with the hemodynamic score.

To determine the effect of age on physical growth of the high score VSD patients, we divided patients into two groups (Table 6). Fourteen out of 17 younger VSD patients (82.3%) and all of older VSD patients had subnormal body weight. Eleven out of 17 younger VSD

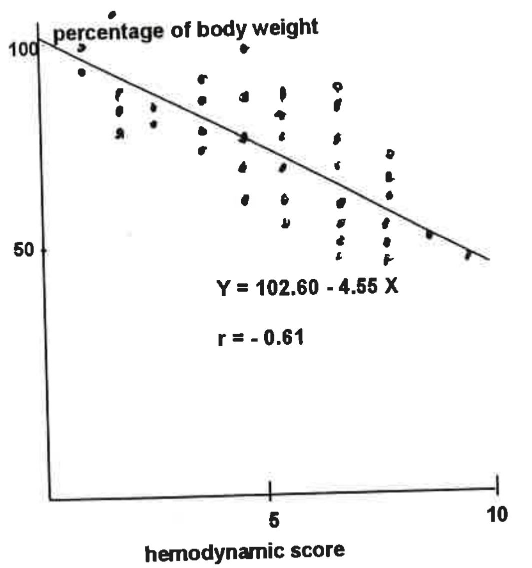

Figure 1. Correlation between tody weight and hemodynamic score in 46 VSD patients

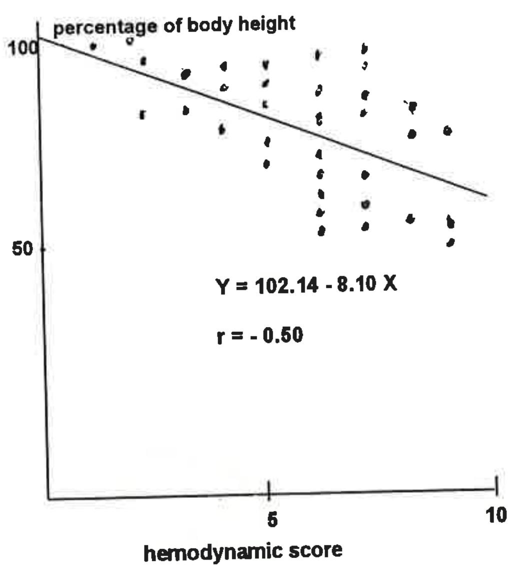

Figure 2. Correlation between body height and hemodynamic score in 46 VSD patients

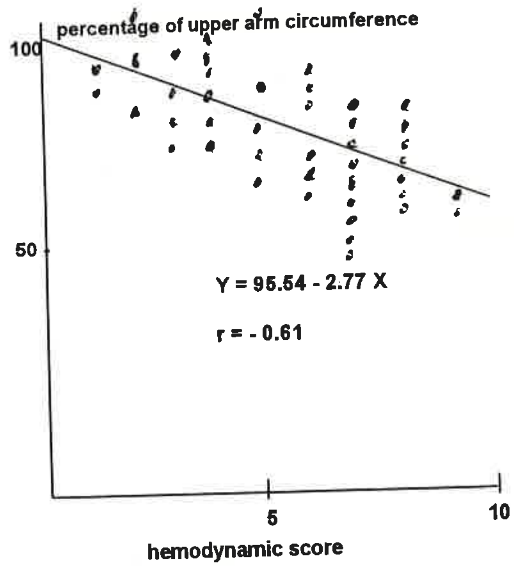

Figure 3. Correlation between upper arm circumference and hemodynamic score in 46 VSD patients

patients (64.7%) and all older patients had subnormal body height. Eleven out of 17 younger VSD patients (64.7%) and 5 out of 6 older patients (83.3%) had subnormal upper arm circumference. Though there was no correlation between physical growth status and the age of the high score patients, the growth disturbance in high score patients seems had occurred in the younger age and worsened at the older age (Table 6).

During 12 months of observation, 10 high score VSD patietns, 5 low score VSD patients, and 10 controls were lost to follow-up. The mean percentages of body weight, height, and upper circumference in 13 high, and 18 low score VSD

Table 6. Correlation between physical growth status and the age of the 23 high score VSD patients

Age (mo)	BW N	BW SN	BH N	BH SN	UAC N	UAC SN
12 - 35	3	14	6	11	6	11
36 - 60	0	6	0	6	1	5
Jumlah	3	20	6	17	7	16
Fisher (p)	0,3840		0,1226		0,3029	

BW = body weight; BH = body height; N = normal; SN = subnormal; UAC = upper arm circumference

patients and 20 controls during 12 months observation were shown in Tables 7, 8, and 9, and Figures 4, 5 and 6. The body weight, height, and upper arm circumference was detected in high score VSD patients at first examination and became more apparent after 12 months of observation. In low score VSD patients, only upper arm circumference was slightly affected at the beginning of the study (Table 9, Figure 6). The body weight was slightly disturbed after 9 months of observation, while the body height was not clearly affected throughout the study (Tables 7 and 8, and Figures 4 and 5).

Discussion

The association of growth and congenital heart disease has received extensive attention in the literature.[11,12] Severe growth disturbance in patients with VSD is only in part due to abnormal postnatal hemodynamics. Intrauterine, genetic factors, and low birth weight also play a role and help explain the incomplete growth

Table 7. Mean percentages of body weight in high score, low score, and controls during 12 months of observation

Interval observ. (month)	Body Weight: High score x (SD) (n=13)	Low score x (SD) (n=18)	Control x (SD) (n=20)
0	64.6 (8.49)	87.8 (13.49) *	90.6 (7.46)
3	64.0 (8.11)	87.5 (13.79)*	90.7 (7.40)
6	63.2 (7.52)	87.0 (13.48)*	91.6 (7.41)
9	61.9 (7.37)	86.5 (13.84)**	91.8 (7.43)
12	61.0 (7.36)	86.3 (13.90)**	92.5 (7.11)

x = mean; SD = standard deviation; *= p > 0.05 ; **= p < 0.05.

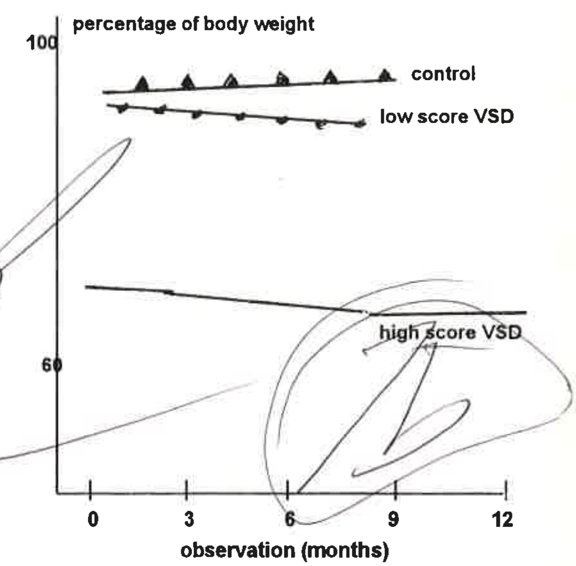

Figure 4. Mean percentage of body weight in 13 high score VSD, 18 low score VSD and 20 control patients during 12 months of observation

Table 8. Mean percentage of body height in high score, low score and control patients during 12 months of observation

	Body height		
Interval observ (mo)	High score x (SD) (n=13)	Low score x (SD) (n=18)	Control x (SD) (n=20)
0	88.3 (5.82)*	95.6 (5.24)	97.2 (4.46)
3	88.1 (8.60)*	95.6 (4.95)	97.1 (4.38)
6	87.7 (6.13)*	95.6 (4.95)	97.1 (4.53)
9	87.4 (6.61)*	95.5 (5.03)	97.6 (4.58)
12	87.3 (6.77)*	95.3 (4.86)	98.0 (4.46)

* = $p < 0.05$; BH = body height; x =mean; SD =standard deviation

Table 9. Mean percentage of upper arm circumference in high score, low score, and control patients during 12 months of observation

	UAC		
Interval observ (month)	High score x (SD) (n=13)	Low score x(SD) (n=18)	Control x (SD) (n = 20)
0	72.7 (4.84)	84.7 (8.82)*	91.5 (4.62)
3	72.5 (5.08)	84.8 (8.56)*	91.9 (4.62)
6	71.9 (5.38)	84.9 (8.40)*	92.2 (4.60)
9	71.2 (6.03)	85.0 (8.28)*	92.6 (4.55)
12	70.6 (6.25)	85.3 (8.12)*	93.0 (4.88)

* = $p < 0.05$; UAC = upper arm circumference; x = mean; SD = standard deviation

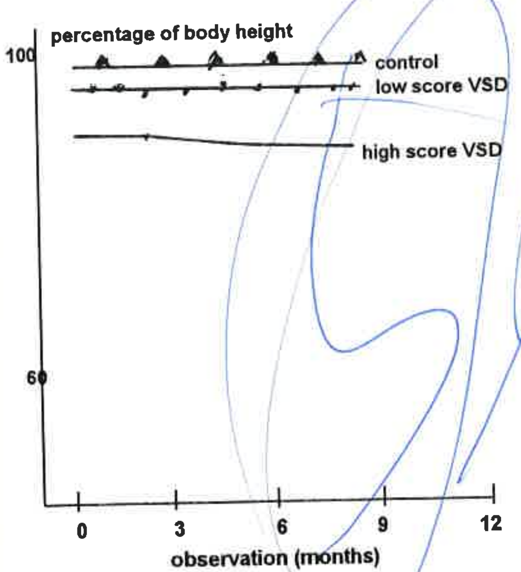

Figure 5. Mean percentage of body height in 13 high score VSD, 18 low score VSD, and 20 control patients during 12 months of observation.

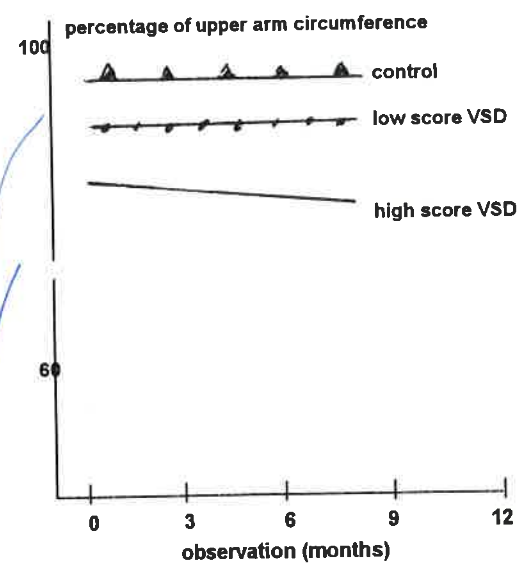

Figure 6. Mean percentage of upper arm circumference in 13 high score VSD, 18 low score VSD, and 20 controls during 12 months of observation.

response after succesful surgery.[3] Several perinatal factors have been presumed to influence the physical growth, but no significant differences are found in perinatal factors except for birth weight.[6] The mean birth weight and mean height of the VSD patients were 3.3 (SD 1,37) kg and 49.0 (SD 1,41) cm (Table 2). These data could exclude the main perinatal factors influencing physical growth in our study.

Infants with linear growth failure and congenital heart disease generally have an associated reduction in weight relative to height which is suggestive of undernutrition.[4] In left to right shunt lesions, weight is more severely affected than height. The results of our study showed that subnormal body weight, body height, and upper arm circuference were detected in 54.3%, 47.8%, and 56,5% of VSD patients respectively (Table 4). It means that body weight and upper arm circumference were more affected than body height. Although these findings were slightly higher than the other reports, the physical growth status were in accordance with the others.[4,11,13]

For the purposes of the study 10 important findings based on history, physical examination, electrocardiography, chest X-ray and 2 D echocardiography were created as the determinants of the hemodynamic changes. Negative correlation was observed between hemodynamic score and the percentage of body weight, body height, and upper arm circumference of the VSD patients (Table 5 and Figures 1, 2, and 3). The physical growth status of those patients with high score (6-10) were significantly different compared with those with the low score (0-5). Therefore we recommended to use the hemodynamic scoring as a parameter to predict the physical growth status in isolated VSD patients.

Levy (1978) reported that admission measurements of physical growth in VSD patients revealed height and weight status to be subnormal and related to physiological severity and age.[3] To ascertain the effect of age on the physical growth of the significant left to right shunt patients, we divided the high score patients into two group. Most of the patients in the two groups had subnormal body weight, body height and upper arm circumference (Table 6). There was no correlation between physical growth status and the age of the high score VSD patients. We presumed that the physical growth disturbances in our high score VSD patients were not affected by the patient age but merely due to left to right shunt and pulmonary hypertension.

The influence of hemodynamic severity on growth failure in patients with VSD has been reported previously by other investigators.[4,5] Infants and children with VSD frequently have subnormal growth, presumably due to a large left to right shunt, congestive heart failure, or pulmonary vascular obstruction.[3] Increased pulmonary artery pressure with large shunts is noted to correlate well with growth failure. There is close asssociation between pulmonary hypertension and poor weight gain [5] Moreover not only the pulmonary/systemic pressure ratio, but also the pulmonary/systemic flow ratio is an important determinant of physical growth in pediatric patients with VSD.[3]

Though there was no correlation between physical growth status and the age in high score VSD patients, the physical growth disturbance in high score VSD patients seemed to have occurred at younger age and worsened at the older age (Table 6). Medical therapy is associated with little change in the subnormal growth status. Most of organ abnormalities observed in the VSD patients suggesting malnutrition as a common etiology.[14] However, Strangway concludes that in children with growth failure associated with congenital heart disease, nutrient intake is not an important factor which limit their growth.[15] That's why it was not surprising that the percentage of body weight, body height and upper arm circumference were affected in high score VSD patients since the first examination and markedly disturbed significantly after 12 months observation (Tables 7,8, and 9 and Figures 4,5, and 6). These findings were in contrast with the report of Bayer in 1969, that although subnormal height and weight status persist in the group treated medically, there is a relative improvement in growth noted in medically treated patients over 2 years.[16]

The natural history of ventricular defect in patients surviving infancy has been reported previously.[17,18] Of the 499 patients with several clinical examinations, 71% remain stable; in 21% changes sugesting some level of closure are noted; in 7% infundibular stenosis begin to evolve, and in 1% pulmonary vascular disease begin to appear or becomes accentuated.[18]

Some interesting findings are seen in our study. Even in low score VSD patients the upper arm circumference was affeted at the beginning and the body weight was affected after 9 months of observation. The subnormal upper arm circumference and body weight in those low score VSD patients were probably not due to severe hemodynamic alteration but might be due to the increased of basal metabolic rate, because of recurrent upper respiratory tract infection. These findings were in accordance with the report of Krieger, that calorie requirements per unit of body weight in his patients are increased because the basal metabolic rate is increased, and not due to the heart disease itself.[19]

Finally, based on all of our findings we suggest that the proposed simple hemodynamic scoring system is of benefit for clinical evaluation and further work-up in isolated VSD patients before cardiac surgery, the definite treatment of all but the most small VSD, is performed.

Conclusions

1. Body weight and upper arm circumfrence were more affected than body height in VSD patients.
2. Negative correlation was observed between hemodynamic scores and the percentages of body weight, height, and upper arm circumference of the VSD patients
3. Physical growth disturbances in our high score VSD patients were not associated with the patient's age.
4. The proposed hemodynamic scoring system is of benefit for making consideration and further work-up in VSD patients.

References

1. Anthony CL, Arnon RG, Fitch CW. Pediatric cardiology. Tennesee: Medical Examination Publishing 1979; 206-25.
2. Fyler D. Ventricular septal defect. In Fyler DC, Nadas' pediatric cardiology, Singapore: Henley & Belfus 1992; 435-57
3. Levy RJ, Rosenthal A, Miettinen OS, Nadas AS. Determinants of growth in patients with ventricular septal defect. Circulation 1978; 57:793-7.
4. Mehrizi A, Drash A. Growth disturbance in congenital heart disease. J Pediatr 1962; 61:418-29
5. Miller RH, Schiebler GL, Grunbar P, Krovetz LJ. Relation of hemodynamics to height and weight persentiles in children with ventricular septal defect. Am Heart J 1969; 78:523-9
6. Kristianson B, Fallstrom SP. Infant with low rate gain. II. A study of an envirement factor. Acta Paediat Scand 1981; 70: 663-8.
7. Rilantono LI, Harimurti G, Firman S, Yusuf AH, Susetyo B. Ventricular septal defect and its problems. Proceeding of the Third National Congress of the Indonesian Heart Association (KOPERKI III), Surabaya 1981
8. Coussement AM, Gooding CA. Obyective radiographic assessment of pulmonary vascularity in children. Radiology 1973; 109:649-54.
9. Samsudin. Cara penilaian keadaan pertumbuhan dan perkembangan fisik anak. 01-10. In: Samsudin, Tjokronegoro A, Eds, Gizi dan tumbuh kembang. Jakarta: Balai Penerbit FKUI 1985;1
10. Husaini YK, Husaini MA, Kunanto G, Karyadi D. Pertumbuhan anak sehat berumur 12 sampai 60 bulan berdasarkan berat dan tinggi badan. Gizi Indonesia 1985; 10:53-6.
11. Umansky R, Hauck AJ. Factor in the growth of children with congenital heart disease. Pediatrics 1962; 30:540-51.
12. Suoninen P. Physical growth og children with cogenital heart disease, pre- and post oprative study of 355 cases. Acta Paediatr Scand (Suppl) 1971; 225:1-45.
13. Chan KJ, Tay JSH, Yip WCL, Wong HB. Growth retardation in children with congenital heart disease. J Singapore Paediatr 1988; 29:185-93.
14. Naeye RL. Organ and cellular developement in congenital herat disease and in alimentary malnutrition. J Pediat 1965; 67:447-58.
15. Strangway A, Fowler R, Cunningham K, Hamilton JR. Diet and growth in congenital heart disease. Pediatrics 1976; 57: 75-86.
16. Bayer L, Robinson SJ. Growth history of children with congenital heart defects. Am J Dis Child 1969; 7:564-8.
17. Bloomfield DK. The natural history of ventricular septal defect in patients surviving infancy. Circulation 29: 914, 1964
18. Corone P, Doyon F, Gaudeau S, et al. Natural history of ventricular septal defect, a study involving 790 cases. Circulation 1977; 55:908-15.