ORIGINAL ARTICLE

HIS Bundle Electrocardiogram in Children with Secundum Atrial Septal Defects and Patent Ductus Arteriosus*

by

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Abstract

The purpose of this study is to compare intra-atrial conduction (atrio-ventricular conduction and intraventricular conduction) in children with secundum atrial septal defect (ASD II) and patent ductus arteriosus (PDA) using His bundle electrocardiogram (HBE).

Six patients with ASD II and seven with PDA, aged ranging from 3 to 11 years, were included in the study. The diagnoses of all cases were confirmed by cardiac catheterization. The results of HBE studies showed that the PA, AH, HV, interval in ASD II were 31.7 ± 7.6 msec; 65.8 ± 16.8 msec; 42.5 ± 2.2 msec; and in PDA were 25 ± 7.8 msec; 77.9 ± 9.4 msec; 40 ± 3.8 msec respectively. The differences were statistically not significant ($P > 0.05$).

The data showed that intra-atrial conduction in children with ASD II was not longer than that in PDA and also there was no difference in atrioventricular conduction and intraventricular conduction, although children with ASD II usually have abnormal ECG, like right bundle branch block.

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Introduction

The intracardiac electrogram or His bundle electrocardiogram was first described by Alan and others using an isolated heart preparation. They were able to record a biphasic potential of the His bundle which they called
tial. Scherlag and associates (1969) later showed that this His (H) potential could be recorded consistently during cardiac catheterization.

His bundle electrocardiogram (HBE) was divided into three components : P — A interval, A — H interval and H — V interval (Anderson et al., 1973).

There have been several subsequent reports on His bundle in adult, but there were less
children (Abell et al., 1972; Brodsky et al., 1971; Roberts and Olley, 1972a; Anderson et al., 1973). His bundle might be expected to differ from that of adult for two reasons (Roberts and Olley, 1972b). (1) The child influenced the P—R interval and this might affect the P—A, A—H or H—V interval
heart rate influenced the P—R interval and thus similarly affected the P—A, A—H or H—V interval.

They also suggested that this conduction was not influenced by cardiac abnormalities.

The purpose of this study is to investigate intra-atrial conduction (A — V nodal
tem conduction) in children with secundum atrial septal defects (ASD II) using His bundle electrocardiogram (HBE), as compared with children with patent ductus arteriosus (PDA).

Material and methods

Six patients with secundum atrial septal defects and seven patients with patent ductus arteriosus were included in this study. The age ranged from 3 to 11 years. The diagnosis of all cases was confirmed by cardiac catheterization. The electrophysiologic studies were obtained during cardiac catheterization in all cases; and were recorded as previously described (Scherlag et al. 1969; Roberts and Olley, 1972a; Anderson et al., 1973). A tripolar electrode catheter was introduced by way of the femoral vein or saphenous vein and was positioned across the tricuspid valve so that His bundle could be obtained. (1) The P—A interval was measured from the onset of the P wave to the first major rapid deflection of the atrial wave in the intracardiac electrogram; it represents the time from the earliest detectable evidence of atrial muscle activation to the time of low right atrial activation, the so called intra-atrial conduction. (2). The A — H interval was measured from the first major rapid deflection of the atrial wave to the onset of the His bundle deflection; it represents the time from the low right atrial activation to the activation of some part of the His bundle or proximal bundle branches, the so called A — V nodal conduction. (3) The H—V interval was measured from the onset of the His bundle deflection to the earliest deflection caused by

the onset of ventricular activation; it represents the time from the activation of the His bundle to the onset of ventricular muscle activation, the so called HIS — Purkinje system conduction.

Results

The vital statistics, physical findings and laboratory data of the patients studied were shown in table 1.

TABLE 1: *Vital Statistics, Physical Examinations and Laboratory Data of Patients Studied*

ASD II GROUP

	Patient	Age (yr)	M/F	Phys. Exam. BP mmHg	RV	LV	Wide fixed Split 2nd HS	Ej. Syst. Mur-mur	Cont. Mur-mur	Lab. Exam. Hb. gm/ 100 ml	Ht. v%	RBC mil/ cmm
A1	IRN	10	F	120/80	+	—	+	+	—	14.8	40	48.6
A2	NRH	6	F	105/60	+	—	+	+	—	15.5	45	4.82
A3	SKM	11	F	100/70	+	—	+	+	—	12.6	38	4.42
A4	MRN	12	F	120/80	+	—	+	+	—	12.4	38	4.24
A5	IFS	11	F	120/80	+	—	+	+	—	16.1	42	5.62
A6	NRM	11	F	110/70	+	—	+	+	—	12.4	36	4.07

PDA GROUP

	Patient	Age (yr)	M/F	Phys. Exam. BP mmHg	RV	LV	Wide fixed Split 2nd HS	Ej. Syst. Mur-mur	Cont. Mur-mur	Lab. Exam. Hb. gm/ 100 ml	Ht. v%	RBC mil/ cmm
B1	HLD	10	F	105/65	—	+	—	—	+	12.6	35	3.78
B2	END	6	M	95/60	—	+	—	—	+	12.6	32	4.38
B3	FRD	3	F	80/50	—	+	—	—	+	12.1	37	4.38
B4	SRY	5	F	95/60	—	+	—	—	+	12.5	35	4.32
B5	DNA	6	F	95/60	—	+	—	—	+	11.5	36	3.90
B6	RKP	4	F	95/60	—	+	—	—	+	11.9	38	4.02
B7	RTS	6	F	90/60	—	+	—	—	+	12.1	36	4.28

All of the six patients with secundum atrial septal defects (ASD II) were female and six out of seven patients with tent ductus arteriosus (PDA) were female too. The age of the patients with ASD II ranged from 6 to 12 years with the average of 10 years, while the patients with PDA ranged from 3 to 10 years with the average of 6 years. All of the patients studied had classical physical findings.

Patients with ASD II showed increased right ventricular activities, widely fixed split of the second heart sounds, soft ejection systolic murmur on the upper left sternal border and middiastolic murmur on the tricuspid valves area. Patients with PDA showed wide pulse pressure, continuous murmur on the infraclavicular areas; six out of seven patients had middiastolic flow murmur on the mitral valves areas. All of the patients were not anemic; the hemoglobin contents, the hematocrit contents and the red blood cell counts of the two groups were not significantly different (Table 1).

TABLE 2: *Electrocardiographic Data of the Patients Studied*

Patient	Age (yr)	P axis	QRS axis	T axis	P-R (msec)	Rate	QRS-T grad.
ASD II GROUP							
A1 IRN	10	+ 30	+ 110	— 20	150	94	130
A2 NRH	6	+ 60	+ 115	0	130	108	115
A3 SKM	11	+ 60	+ 110	+ 15	185	92	95
A4 MRN	12	+ 50	+ 100	+ 30	140	120	70
A5 IFS	11	+ 30	+ 115	— 20	155	90	130
A6 NRM	11	+ 60	+ 140	+ 4	120	108	136
PDA GROUP							
B1 HLD	10	+ 35	+ 70	+ 50	155	82	20
B2 END	6	+ 75	+ 75	+ 55	175	90	20
B3 FRD	3	+ 55	+ 70	+ 75	160	108	5
B4 SRY	5	+ 60	+ 50	+ 60	145	100	10
B5 DNA	6	+ 25	+ 55	+ 45	120	90	10
B6 RKP	4	+ 35	+ 60	+ 35	145	100	25
B7 RTS	6	+ 65	+ 86	+ 47	160	100	39

The heart rate of the ASD II group and the PDA group ranged from 90 to 120 and from 82 to 108 per minute respectively. This was not statistically different. The P—R interval of the ASD II group and the PDA group ranged from 120 to 185 and from 120 to 175 miliseconds respectively. This was not statistically different either. The P axis of the ASD II group ranged from — 30 to +60, the QRS axis ranged from +100 to +140, the T axis ranged from —20 to +30 and the QRS—T axis gradients ranged from 70 to 136. All of the patients with ASD II showed incomplete right bundle branch block (RBBB). The P axis of the PDA group ranged from +25 to +75, the QRS axis ranged from +50 to +86, the T axis ranged from +35 to +75 and the QRS—T axis gradient ranged from +5 to 39.

TABLE 3 : *Mean and Standard Deviation of P axis, QRS axis, T axis and QRS-T gradient of Patients Studied*

ECG Data	ASD II (y ± 2s)	PDA (y ± 2s)	Test
P axis	+ 48.3 ± 29.4	+ 50 ± 36.0	$p > 0.10$
QRS axis	+ 115 ± 26.8	+ 67 ± 24.8	$p < 0.001$
T axis	+ 1.5 ± 39.2	+ 52 ± 25.4	$p < 0.001$
QRS — T grad.	113.5 ± 52.8	18 ± 23.0	$p < 0.001$

The heart rate, the P — R interval and the P axis between these two groups were not statistically different; however the QRS axis, the T axis and QRS—T axis gradient between these two groups were significantly different (Table 3).

TABLE 4 : *Hemodynamic Data of Patients Studied*

	Oxygen Saturation				Pressure (mmHg)					Flow ratio	Shunt ratio	Press. ratio	Resist. ratio	D/
	VC	RA	PA	LA	Ao	RA	P	A	LA	Ao					
A1	83.1	91.1	93.9	105.1		1.5	23	3		100/60(75)	2.0	50	23.5	11.8	ASD II
A2	80.5	89.9	94.3	104.7		2.0	15	2.5		100/75(95)	2 6	61.5	13 4	5	ASD II
A3	74 6	90.8	92.7	102.3		1 0	18	3		100/68(80)	2.9	65.5	18.7	6.5	ASD II
A4	78.9	96.8	96.3	103.5		0.5	34	1.5		90/75(85)	3.4	70.6	38.5	11.3	ASD II
A5	78.7	86.1	85.2	103.5		0.5	50	1		115/80(95)	1.5	33	52	35	ASD II
A6	72.2	81 2	85.7	896.8		0	10	1.5		110/70(84)	2.4	58	10	4	ASD II
B1	84.4	88.1	92.6		105.1	4	10	5		80/50(60)	1.7	41.2	17	10	PDA
B2	83.1	85.5	98.6		105.5	0.5	32	10		125/50(85)	2.9	65.5	26	9	PDA
B3	84 1	79.2	97.7		109.2	2	30	10		125/75(100)	2.2	55	20	9	PDA
B4	88.1	85.8	99.7		110.8	1	34	11		110/50(80)	2.5	60	29	11.6	PDA
B5	92.7	91.3	100.6		110.0	1	22	6		120/75(85)	2.2	54.6	19	8.7	PDA
B6	84.3	83 3	96.6		109.9	3	34	10		100/50(70)	2 0	50	36	18	PDA
B7	73.7	76.5	80.2		100.3	1	11	2		120/75(100)	1.2	16.7	10	8	PDA

The hemodynamic data of the patients studied were shown in Table 4. All of the ASD II group showed oxygen step up at atrial level, five of them with flow ratio of more than 2 : 1 and resistance ratio of less than 12%; while all of the PDA group showed oxygen step up at pulmonary arterial level, five of them with flow ratio of more than 2 : 1 with resistance ratio of less than 20%.

TABLE 5 : *Mean and Standard Deviation of Flow Ratio, Shunt Ratio, Pressure Ratio and Resistance Ratio of Patients Studied.*

Pulmonary-Systemic Ratio	ASD II (y ± 2s)	PDA (y ± 2s)	Test
Flow Ratio	2 5 ± 1.7	2.1 ± 1.7	$p > 0.10$
Shunt Ratio	56.4 ± 26.9	49.0 ± 32 4	$p > 0.10$
Pressure Ratio	26.0 ± 32.4	22.4 ± 17.2	$p > 0.10$
Resistance Ratio	12.3 ± 23.2	10.6 ± 6.9	$p > 0.10$

The flow ratio, shunt ratio, pressure ratio and resistance ratio of these two groups were not statistically different (Table 5).

TABLE 6 : *Mean and Standard Deviation of Right Atrial, Pulmonary Arterial, Left Atrial, Aortic Pressure and Systemic Pulse Pressure of Patients Studied.*

Mean Pressure (mmHg)	ASD II (y ± 2s)	PDA (y ± 2s)	Test
Right Atrium	0.9 ± 2 0	1 8 ± 2.6	$p > 0.10$
Pulmonary Artery	25.0 ± 29.6	24.7 ± 21.1	$p > 0.10$
Left Atrium	2.1 ± 1.7	7.7 ± 6.8	$p < 0.01$
Aortic Pressure	84.8 ± 19.0	82.9 ± 29.4	$p > 0.10$
Systemic Pulse Pressure	33.7 ± 21.4	50.7 ± 33.7	$p < 0.5$

The mean pressure of the right atrium, pulmonary artery and aorta of these two groups were not statistically different. However the mean left atrial pressure and systemic pulse pressure of the ASD II group were less than that of the PDA group. These differences were statistically significant (Table 6).

TABLE 7 : *Duration of P—R, P—A, A—H and H—V Intervals in Milliseconds of the Patients Studied*

Patient	Age (yr)	%L — R	P — R	P — A	A — H	H — V
ASD II GROUP						
A1 IRN	10	50	150	30	50	45
A2 NRH	6	61.5	130	30	50	40
A3 SKM	11	65.5	185	45	90	40
A4 MRN	12	70.6	140	40	50	40
A5 IFS	11	33	155	20	85	45
A6 NRM	11	58	120	25	50	45
PDA GROUP						
B1 HLD	10	41.2	155	20	85	45
B2 RND	6	65.5	175	40	75	55
B3 FRD	3	55	160	52	100	40
B4 SRY	5	50	145	20	80	40
B5 DNA	6	54 6	120	20	60	30
B6 RKP	4	50	145	20	70	40
B7 RTS	6	16.7	160	40	75	40

The P — R interval, P — A interval, A — H interval and H — V interval of the two groups were shown in tables 7 and 8.

TABLE 8 : *Mean and Standard Deviation of P-R, P-A, A-H, and H-V Intervals of Patients Studied*

H. B. E.	ASD II (y ± 2s)	PDA (y ± 2s)	Test
P - R (msec)	146.7 ± 45.4	151.4 ± 34.6	$p > 0.10$
P - A (msec)	31.7 ± 7.6	25.0 ± 7.8	$p > 0.10$
A - H (msec)	65.8 ± 16 8	77.9 ± 9.4	$p > 0.10$
H - V (msec)	42.5 ± 2.2	40 0 ± 3.8	$p > 0.10$

Discussion

Only one patient of the patients studied was a male; it means that ASD II and PDA were more common in female, these findings were previously described by others (Wood, 1971; Nadas and Fyler, 1973). The average age of the PDA group was younger than that of the ASD II group.
tients with PDA had their symptoms earlier and were detected earlier by the physician.

All of the ASD II group showed incomplete right bundle branch block, these findings were similar to the electrocardiographic pattern in large ASD II reported by Martin de Oliveira and Zimmerman (1958). Only one patient with large ASD II had increased pulmonary arterial pressure and resistance ratio of more than 12%, it means that in uncomplicated ASD II the fundamental hemodynamic changes are an increase in blood volume work without significant increase in pressure in the right ventricular chamber.

The sum of P—A, A—H and H—V interval of the two groups was less than the P—R interval measured from the body surface in lead II by a few milliseconds (Table 7). This could be due to the proximity of the extracellular fields produced by ventricular activation to the intracardiac leads to record lead II, so that with the onset of ventricular activation selective changes might occur in specific body areas several milliseconds before potentials are perturbed in lead II (Anderson et al., 1973).

The P — R, P — A, A — H and H — V interval were not influenced by age and shunt ratio (Table 7). Roberts and Olley (1972b) demonstrated that the prolongation in P — R interval which occured with the increase in age occured at the H — V interval, however there was no significant difference in the P — A interval.

There was no differences in the P — R interval and P — A interval between these two groups (Table 8).

These findings were similar to those reported by Roberts and Olley (1972b) in patients with ASD II whose left to right shunt was small and the pulmonary-to-systemic flow ratio was less than 2 : 1. Prolongation of the P — R interval and the P — A interval (intraatrial conduction) was reported by Anderson and associates (1973) with large secundum atrial septal defects. They concluded that any large atrial septal defects of the secundum type altered a large segment of the anatomy of the atrial septum and secondarily increased the distance of the sinus node and the A — V node due to atrial enlargement required
overload. However, we found that the P — R interval and P — A interval were not influenced by interatrial septal defects, even in patients with large defects with the pulmonary-to-systemic flow ratio of more than 2 : 1. There were three specific atrial pathways (internodal pathways) connecting the sinus and A — V nodes which conducted faster than the surrounding atrial muscle (Meredith and

Titus, 1968). It might explain the normal P — R interval and normal intra-atrial conduction in our cases in which the major of these internodal pathways were not affected by the atrial defects. But we suggested that in patients with a wellknown prolonged P — R interval, the prolongation might also occur in the P — A interval due to severe enlargement of the right atrium.

The A—H interval and H—V interval between these two groups were not statistically different (Table 8). These were also reported by others (Roberts and Olley, 1972b; Anderson et al., 1973). It means that the A—V nodal conduction and the His-Purkinje system conduction were not influenced by congenital cardiac abnormalities such as in patients with atrial complete right bundle branch block and volume overload of the right ventricle. The incomplete RBBB presented here did not mean disturbance in conduction of the right branch of the bundle of His itself, but delay in activation of the basal portions of the right ventricle and of the higher part of the interventricular septum. The delay in conduction is due to hypertrophy and dilatation of the region mentioned above.

Conclusion

1. Intraatrial conduction might not be prolonged in patients with large secundum atrial septal defects showing normal P — R interval.
2. A—V nodal conduction and His-Purkinje system conduction were not influenced by congenital cardiac defects.

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