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RESEARCH ARTICLE

Mapping of the Cardiac Conduction System and Evaluation of its Electrophysiologic Properties in Pediatric Patients with Congenital Heart Diseases

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Abstract: Background: Congenital heart defects can interfere with heart conduction system development. Understanding cardiac conduction system and its electrophysiological properties is essential in children with congenital heart disease (CHD). This study aims to map the conduction system and assess its electrophysiologic characteristics in children with CHD. Intracardiac electrophysiological studies were conducted in 41 children aged (2-18 years) with CHD undergoing therapeutic or diagnostic cardiac catheter. Conduction intervals (AH/HV) and atrioventricular nodal (AVN) properties including Wenckebach cycle length (WCL), effective refractory period (ERP), sinus node function including sinoatrial conduction time (SACT) and sinus node recovery time (SNRT) were correlated with anatomical lesions of the patients . Results: We detect significant prolongation of the AH interval in patients with simple lesions affecting the right side of the heart (74.19 ± 20.92 msec) (mean ± SD) P value (0.03) indicating delayed conduction through the AV node. In contrast, an increase in the mean of HV interval was noted predominantly in cases with simple lesions affecting the left side of the heart (46.62 ± 15.45 msec) (mean ± SD), indicating infra-Hisian conduction delay. We also found that HV interval was shorter in patients with complex lesions with left ventricular predominance (39.8 ± 4.32 msec) (mean ± SD) P value (0.03) .There was significant positive correlation between AH and PR intervals , atrioventricular node WCL show positive age dependent correlation . Also, SNRT correlated significantly and positively to cSACT. In patients with dextrocardia or abnormal cardiac situs, catheter positioning required modified mapping approaches due to altered anatomical orientation. Conclusion: Electrophysiologic properties of the heart conduction system in pediatric patients with CHD vary according to lesion itself , its hemodynamic effect and patient's age .Recognizing these patterns can enhance procedural accuracy particularly in complex anatomical variants.

Keywords: Cardiac Conduction System, electrophysiologic Properties, congenital heart diseases.

BACKGROUND

Congenital heart disease (CHD) is considered to be the commonest major congenital anomaly in the first year of life and so it represents a significant health care challenge ***(Mandalenakis et al., 2017).***

CHD can interfere with development of the conduction system of the heart**.** This can occur through anatomical affection of site of conduction system components and the hemodynamic effect of lesion by volume and pressure overload ***(Houck et al., 2020).***

Invasive electrophysiologic testing with programmed stimulation and mapping for full description of the conductive system in children with CHDs and detection of any abnormal hidden pathway that act as substrate for arrhythmia will enable cardiologist and cardiothorathic surgeon to adopt warranted plan to avoid and predict post-operative complications as a consequence of conductive system injury and proper management of arrhythmia.

METHODS

This is a cross section observational study involved 41 children aged (2-18 years) with CHD prepared for diagnostic or therapeutic cardiac catheter according to their own congenital cardiac lesions. The study was conducted in Mansoura University Children's Hospital (MUCH), Cardiology department from April 2022 to March 2024. The study protocol was done to map and describe the cardiac conductive system in pediatric patient with CHD including anatomical site of sinoatrial node (SAN), AVN, His bundle to detect any abnormalities with identification of the electrophysiologic characters of the conductive system including Conduction intervals (AH/HV) and AVN properties including WCL ERP, SAN function including cSACT and SNRT with correlation of these findings with cardiac anatomical lesions. Patients who underwent surgical correction of the cardiac lesion that disrupt the original anatomical lesion and those who had cardiac pacemaker were excluded.

Data collection

All patients in the study were subjected to the followings:

1. **Full detailed history** as demographic data: name, age, sex, medical and interventional history.
2. **Full cardiac examination.**
3. **Baseline 12 leads electrocardiogram** with measurement of HR, PR interval, QRS duration and QTc interval, presence of right or left bundle branch block.

- **Conventional echocardiography** for assessment of original lesion **Atrial septal defect (ASD)** was classified according to pulmonary-to-systemic flow ratios QP/QS*(Pollick C et al., 1988)*
    - Small ASD : QP/QS <2
    - Large ASD : QP/QS ≥2
- **Ventricular septal defect (VSD)** was classified according to size of the defect in relation to size of aortic valve annulus ***(Lopez L et al ., 2018***)
    - Small VSD : <1/3 of aortic valve annulus
    - Moderate VSD : 1/3–2/3 of aortic valve annulus
    - Large VSD : >2/3 of aortic valve annulus

Patent ductus arteriosus (PDA) was classified according to pulmonary end diameter (***Fernando R et al., 2013)***

- Small PDA : 1.5 - 3 mm
- Moderate PDA : 3-5 mm
- large PDA : > 5 mm

- **Pulmonary and aortic valve stenosis** were classified according to doppler gradient into mild, moderate and severe according to 2014 American College of Cardiology (ACC)/American Heart Association (AHA) guidelines ***(Nishimura et al, 2017)***

- Pulmonary valve stenosis (PS):

- Mild PS: pressure gradient less than40 mm Hg (or RV systolic pressure <50% of the LV pressure).
- Moderate PS: pressure gradient 40 to 70 mm Hg (or RV systolic pressure 50% –75% of the LV pressure).
- Severe PS: pressure gradient greater than 70 mm Hg (or RV systolic pressure >75% of the LV pressure).

- Aortic valve stenosis (AS):

- Mild AS : mean Doppler gradient ≤20 mm Hg
- Moderate AS : mean Doppler gradient 20 - 39 mm Hg
- Severe AS : mean Doppler gradient ≥40 mm Hg

- Coarctation of aorta (COA)

Mean Doppler gradient >20 mmHg is often used as the threshold to recommend COA intervention ***(Egbe AC et al., 2023)***

4. **Electrophysiologic study (EPS)** with mapping of the conduction system including measurement of baseline intervals with atrial and ventricular pacing and extrastimulus as following:

a) Sterilization & local infiltration anesthesia using lidocaine 1% of the right or left groins was being performed and loading dose of heparin 70-75U/Kg IV was given.

b) Three 6F catheters (two quadripolar catheters and one deflectable decapolar catheter) were introduced via the right or left femoral vein puncture. In young patients (weight < 15 kilograms) only two 6 F catheters were used, one quadripolar catheter and other deflectable decapolar catheter.

c) Catheter placement and measurement using electrophysiology recording , cardiac stimulator and ablation (Claris) ST.JUDE medical

- ☒ A 6F quadripolar catheter was placed at His bundle area under fluoroscopic guidance through advancement of the tip of catheter across the tricuspid annulus near the septal region to capture signals from Atrial (A) signal, His bundle (H) potential and Ventricular (V) signal.

In one case had severe form of kyphoscoliosis, His bundle electrogram was recdorded retrogradely through femoral artery using deflectable decapolar catheter at aortic cusp.

- ☒ **AH interval** was measured from atrial (A) signal recorded by the His catheter and the start of His (H) potential. Normal range in pediatrics 50-100 msec ***(Pass RH and Ceresnak SR., 2014).***
- ☒ **HV interval** was measured from the His (H) potential to the earliest recorded ventricular activation on surface ECG. Normal range in pediatrics 30-50 msec. ***(Pass RH and Ceresnak SR ., 2014)***

- This quadripolar catheter was then advanced into RV or LV according to the dominant ventricle for ventricular burst pacing and single extra stimulus.

Ventricular pacing:

- **Burst pacing** was done through introducing a specific number of pacing stimuli (train) at a progressively shorter cycle length.This pacing protocol is used for the assessment of the retrograde Wenkebach cycle length (the stimulated cycle at which the 1:1 atrioventricular conduction over the atrioventricular node stops) and the pattern of retrograde atrial activation.

- **Single extrastimulus** was done through introducing a series of paced stimuli delivered from ventricular catheter of around eight paced beats (a drive train), and then an early paced stimulus is delivered (extrastimulus). The drive train and the extra stimulus with a progressive decrescent value are repeating until there is a loss of ventricular capture. This protocol is used for the evaluation of the retrograde conduction over the atrioventricular node, presence of the accessory pathways and ventricular refractoriness.
  - **Effective refractory period (ERP):** is the longest coupling interval at which a premature impulse fails to propagate through the AVN. Normally, ERP is 230-310 ms ***(Alboni P et al., 1985).***
- **Wenkebach cycle length (WCL):** is the longest pacing cycle length at which Wenckebach block in the AVN is observed. Normally, WCL is 500 to 350 ms ***(Ziad F. Issa et al ., 2019)***
- The 6F deflectable decapolar catheter was introduced to the coronary sinus for atrial antegrade burst pacing and single extra stimulus.

Atrial pacing:

- **Burst pacing** is similar to the ventricular pacing. The pacing is delivered from coronary sinus catheter. This pacing protocol is used for the assessment of the antegrade Wenkebach cycle length
- **Single extrastimulus** is similar to ventricular pacing. The stimulation is made at the coronary sinus. This protocol is used for assessment of the anterograde conduction over the AVN, atrial refractoriness, and induction of specific arrhythmias.
- Another 6 F quadripolar catheter was placed in the high right atrium to deliver pacing stimuli close to the sinus node to assess sinus node function by measuring sinus node recovery time (SNRT) and sinoatrial conduction time (SACT). In young patients (weight < 15 kilograms) the same His catheter was used for this purpose.

SNRT

It is a key measurement to assess sinus node function.

- **Atrial overdrive pacing :**Pacing was done at a rate faster than the sinus rate .The protocol includes the stimulation of the atria at a fixed rate for 30 seconds at various CLs with 50 msec decrements (600, 500, and 400 ms) . The stimulation is then abruptly terminated, and the time needed for the sinus node to regain its automaticity is measured.
- **Measure SNRT:** Measure the interval between the last paced atrial stimulus and the first spontaneous Atrial EGM in the HRA recording. Normally SNRT must be less than 1600 ms
- **Calculate the corrected SNRT (cSNRT):** cSNRT= SNRT – base line sinus cycle length, it is approximately 275 msec in pediatrics ***(Samson RA et al., 1999).***

SACT

It evaluates time taken by an impulse to travel from SAN to surrounding atrial tissue .The protocol is obtained by constant atrial pacing using HRA catheter for 6-8 beats , 30 msec shorter than sinus CL

- **Measure SACT :**
  - SACT = (Return cycle length - spontaneous sinus CL) /2
  - Return cycle length: interval from A2 to next sinus P wave. Normal range is 45 to 125 ms ***(Z i a d F. Issa et al., 2019).***
- During our study if any case developed tachyarrhythmia it will be ablated

d) **Standard views:** LAO 40 and RAO 30 were used to define catheter site by fluoroscopy, however in cases with dextrocardia mirror images of these views were taken to facilitate catheter placement at the usual site, then the mirror image view is returned to the standard view for proper identification of catheter site according to patient anatomy.

e) **The patients were classified into 2 groups :**

Group I: simple lesions, subdivided into subgroups A, B

- IA: lesions affecting right side of the heart (secondum ASD and pulmonary stenosis).
- IB: lesions affecting left side of the heart (VSD, PDA, COA and AS).

Group II: complex lesions, subdivided into subgroups C, D

- IIC: lesions with right ventricular predominance (double outlet right ventricle (DORV) and tetralogy of Fallot (TOF)).
- IID: lesions with left ventricular predominance (Tricuspid atresia , double inlet left ventricle (DILV) , unbalanced AVSD with left ventricular predominance)

Analysis of our data in different groups will include:

1- **Surface ECG:** PR interval, corrected QTc interval by the use of Bazett's formula: QTc=QT/ √ RR and QRS duration.

2- **Intracrdiac electrogram:** AH, HV intervals, WCL and ERP of antegrade and retrograde limbs of AVN, dual AVN physiology, sinus node function by cSNRT and SACT.

3- **Fluoroscopic site** for SAN, His bundle, coronary sinus in patients with different lesions guided by their electrical signals.

ETHICAL CONSIDERATIONS

Ethical approval was obtained from Mansoura University, Faculty of Medicine and Health Sciences Institutional Review Board (IRB). All parents of our children or their legal guardians signed an informed consent before their enrollment in this study. All information obtained through a complete data collection sheet.

STATISTICAL METHODS

All data were tabulated in SPSS sheet version 27. Categorical data were

Expressed as number and percent. Continuous data were tested for Normality of distribution using Shapiro Wilk test. Normally distributed

Data were expressed as mean ± standard deviation (SD). Non- parametric

Data were expressed as median and interquartile ranges (IQrs). Appropriate

Statistical tests were used according to data type:
-Descriptive and frequency analysis were used to describe the data.

-Student t- test was used for comparison between 2 variables of Normally distributed data.

-Chi square test was used for comparison between 2 variables of Categorical data

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-Fisher exact test used for comparison between 2 variables of Categorical data when the number of events in one group was less Than 5.
-Pearson correlation test was used to assess the correlations between 2 variables of continuous type.

RESULTS

The current study included 41 patients with congenital heart diseases with mean age 8.6 ± 3.61 years. Most of included patients were males representing 63.4% of patients ***(table 1).***

Different cardiac lesions were reported. The most frequently reported lesions were ASD in 11 patients (26.8 %), DORV in 6 patients (14.6 %), PDA in 5 patients (12.2 %), COA in 5 patients (12.2%), Other diagnosis of lower frequencies included pulmonary valve stenosis in 3 patients (7.3%) , Tricuspid atresia in 3 patients(7.3%) , VSD in 2 patients (4.9%) , 1 patient (2.4%) for each of the following diseases : pulmonary hypertension , DILV , AS , TOF , unbalanced AVSD (left ventricular dominance) and Ebstein anomaly in ***(table 2).***

About 70.7% of patients had simple lesions and 29.3% had complex lesions. Out of 12 complex lesions, 7 had right ventricular predominance (58.3%) and 5 had left ventricular predominance (41.7%). Out of 29 simple lesions, 16 had right side affection (55.2%) and 13 had left side affection\= (44.8%). ***(Table 3).***

A-H interval had value of 64.1 ± 18.3 ms (mean ± SD). The H-V interval value was 44.4 ± 9.88 ms (mean ± SD). Assessment of AV nodal properties by ventricular burst pacing revealed 1:1 VA concentric conduction via AVN with WCL of retrograde limb 288 ± 89.5 ms (mean ± SD).Ventricular extra-stimulus showed decremental conduction via AVN with ERP of retrograde limb 258 ± 88.3 ms (mean ± SD) . Atrial pacing showed 1:1 conduction with WCL of antegrade limb 260 ± 53.6 ms (mean ± SD). Atrial extra-stimulus showed decremental conduction with ERP of antegrade limb 220± 43.8 msec (mean ± SD).

Of note, 8 children did not have retrograde AVN conduction. Dual AV nodal physiology was documented in 2 patients (4.9%) show jumb and Echo while 3 patients (7.3%) showing a jump phenomenon only. Sinus node function was assessed by cSNRT and SACT. cSNRT was 130 ± 47.4 ms (mean ± SD) , SACT was 66.1 ± 25.6 ms (mean ± SD) , All patients demonstrated normal SNRT and cSACT ***(table 4).***

Patients with simple lesions affecting right side of the heart had significantly longer A-H interval 74.19 ± 20.92 (Mean ± SD) than patients with simple lesions affecting left side of the heart 59.84 ± 12.65 (Mean ± SD) (p= 0.03) . Mean H-V interval in patients with simple lesions affecting the left side of the heart 46.62 ± 15.45 (Mean ± SD)

Was longer than H-V interval in patients with simple lesions affecting the right side 41.87 ± 5.26 (Mean ± SD). (Table ***5)***
We noticed that left ventricular predominance complex lesions had significantly lower H-V interval 39.8 ± 4.32 (Mean ± SD) than right ventricular predominance complex lesion 49.43 ± 4.12 (Mean ± SD) (p= 0.003) ***(table 6)***

A- H interval correlated significantly and positively to PR interval (r: 0.45; p= 0.002) ***(figure 1)***, Also, WCL of antegrade limb of AVN correlated significantly and positively to age ***(r: 0.62; < 0.001) (figure 2),*** WCL of retrograde limb of AVN correlated significantly and positively to age ***(r: 0.44; p= 0.013) (figure 2)***

SNRT correlated significantly to cSACT ***(r: 0.95; p< 0.001) (figure 3)***

Table 1. Demographics of the included patients

	Total cohort (n= 41 patients)
Age (years) Mean ± SD	8.6 ± 3.61
Sex No. (%) - Male - Female	 26 (63.4%) 15 (36.6%)

Table 2. Congenital heart diseases diagnosis:

	Total cohort (n=41 patients) No. (%)
Atrial septal defect	11 (26.8%)
Double outlet right ventricle	6 (14.6%)
Patent ductus arteriosus	5 (12.2%)
Coarctation of the aorta	5 (12.2%)
Pulmonary valve stenosis	3 (7.3%)
Tricuspid atresia	3 (7.3%)
Ventricular septal defect	2 (4.9%)
Aortic valve stenosis	1 (2.4%)
Tetralogy of Fallot	1 (2.4%)
Double inlet left ventricle	1 (2.4%)
Ebstein	1 (2.4%)
Pulmonary hypertension 2ry to CHD	1 (2.4%)
Unbalanced AVSD (left ventricular dominance)	1 (2.4%)

Table 3. Classification of congenital heart lesions

	Total cohort (n= 41 patients) No. (%)
According to complexity: **Simple** **Complex**	 29 (70.7%) 12 (29.3%)
Complex lesions site(ventricular predominance) : **Right ventricle** **Left ventricle**	 7 (58.3%) 5 (41.7%)
Simple lesions (left / right side affected): **Right side** **Left side**	 16 (55.2%) 13 (44.8%)

Table 4 .Data of intracardiac measurments in the studied patients

	Total cohort (n= 41 patients)
A- H (msec) **Mean ± SD**	64.1 ± 18.3
H- V (msec) **Mean ± SD**	44.4 ± 9.88
ERP of antegrade limb of AVN (msec) **Mean ± SD**	220 ± 43.8
WCL of antegrade limb of AVN (millisecond) **Mean ± SD**	260 ± 53.6
ERP of retrograde limb of AVN (msec) **Mean ± SD**	258 ± 88.3
WCL of retrograde limb of AVN (msec) **Mean ± SD**	288 ± 89.5

AVN physiology No. (%) - **Dual (Jump &Echo)** - **Dual (Jump only)** - **No Dual**	2 (4.9%) 3 (7.3%) 36 (87.8%)
cSNRT (Seconds) Mean ± SD	130 ± 47.4
SACT(seconds) Mean ± SD	66.1 ± 25.6

AVN: Atrioventricular nodal; ERP: Effective refractory period; WCL: Wenckebach cycle length; SNRT: Sinus nodal recovery time; SACT: Sinoatrial conduction time; A-H: Atrial to Hiss bundle interval; H- V: Hiss bundle to ventricular myocardium interval.

Table 5. Comparison between left and right side affection in simple lesions as regard intracardiac measurements:

	Simple lesions		
	Left side affection (n= 13)	**Right side affection (n= 16)**	**P value**
A-H (msec) Mean ± SD	59.84 ± 12.65	74.19 ± 20.92	***0.03****
H- V (msec) Mean ± SD	46.62 ± 15.45	41.87 ± 5.26	0.26*
ERP of antegrade limb of AVN (msec) Mean ± SD	220 ± 43.59	227.5 ± 50.92	0.68*
WCL of antegrade limb of AVN (msec) Mean ± SD	264.62 ± 51.41	271.87 ± 66.45	0.75*
ERP of retrograde limb of AVN (msec) Mean ± SD	267.77 ± 78.54	254.61 ± 114.28	0.76*
WCL of retrograde limb of AVN(msec) Mean ± SD	297.5 ± 90.98	296.15 ± 113.62	0.98*
cSNRT (seconds) Mean ± SD	141.8 ± 78.75	121 ± 43.37	0.57*
SACT (seconds) Mean ± SD	75.4 ± 40.72	57 ± 21.86	0.33*
AVN physiology No. (%) - **Dual (Jump &Echo)** - **Dual (Jump only)** - **No Dual**	0 (0%) 2 (15.4%) 11 (84.6%)	2 (12.5%) 1 (6.2%) 13 (81.2%)	0.33**

P: Probability: significance <0.05, *Student t- test; ** Fisher exact test.
A-H: atrial electrogram recorded by the His catheter and the start of His electrogram; **H- V**: His bundle electrogram to the earliest recorded ventricular activation on surface ECG; **AVN**: Atrioventricular nodal; **ERP**: Effective refractory period; **WCL**: Wenckebach cycle length; **SNRT**: Sinus nodal recovery time; **cSACT**: corrected sinoatrial conduction time.

Table 6. Comparison between simple and complex lesions as regard intracardiac measurements:

	Complex lesions		
	Left ventricular Predominance (n= 5)	**Right ventricular Predominance (n= 7)**	**P value**
A- H (msec) Mean ± SD	55 ± 18.19	55.57 ± 11.45	0.95*
H- V (msec) Mean ± SD	39.8 ± 4.32	49.43 ± 4.12	***0.003****
ERP of antegrade limb of AVN (msec)	204 ± 35.07	215.71 ± 36.45	0.59*

Mean ± SD			
WCL of antegrade limb of AVN (msec) Mean ± SD	244 ± 28.81	233.33 ± 23.38	0.51*
ERP of retrograde limb of AVN (msec) Mean ± SD	264 ± 15.16	248.33 ± 90.42	0.71*
WCL of retrograde limb of AVN (msec) Mean ± SD	302 ± 43.82	236 ± 30.49	***0.025****
cSNRT (seconds) Mean ± SD	120.67 ± 11.72	138 ± 8.54	0.11*
SACT (seconds) Mean ± SD	69 ± 8.88	69 ± 13.89	0.99*
AVN physiology No. (%) - **Dual (Jump &Echo)** - **Dual (Jump only)** - **No Dual**	0 (0%) 0 (0%) 5 (100%)	0 (0%) 0 (0%) 7 (100%)	0.99**

P: Probability: significance <0.05, *Student t- test; ** Fisher exact test.
A-H: atrial electrogram recorded by the His catheter and the start of His electrogram; **H- V**: His bundle electrogram to the earliest recorded ventricular activation on surface ECG; **AVN**: Atrioventricular nodal; **ERP**: Effective refractory period; **WCL**: Wenckebach cycle length; **SNRT**: Sinus nodal recovery time; **cSACT**: corrected Sinoatrial conduction time.

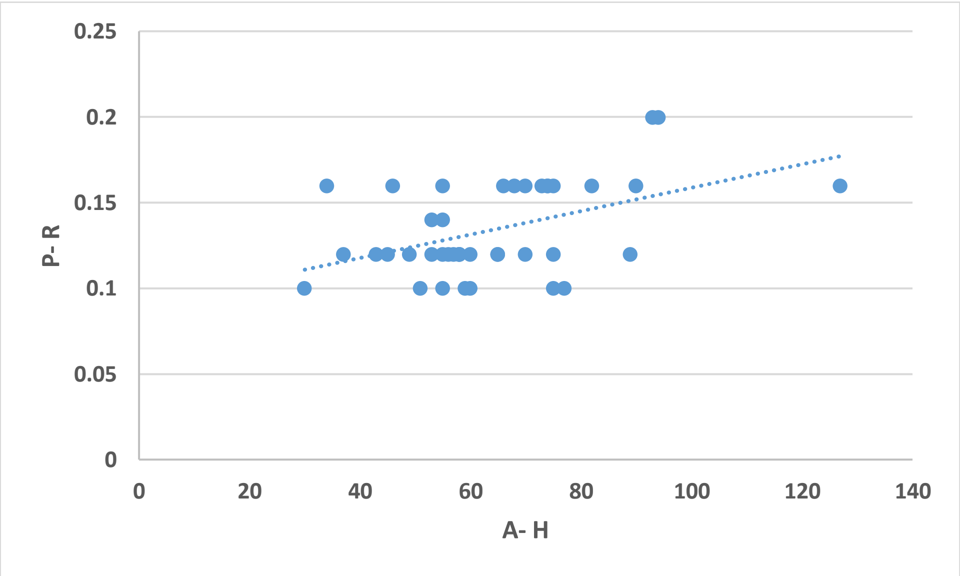

Figure 1: Significant positive correlation between A- H and P- R interval

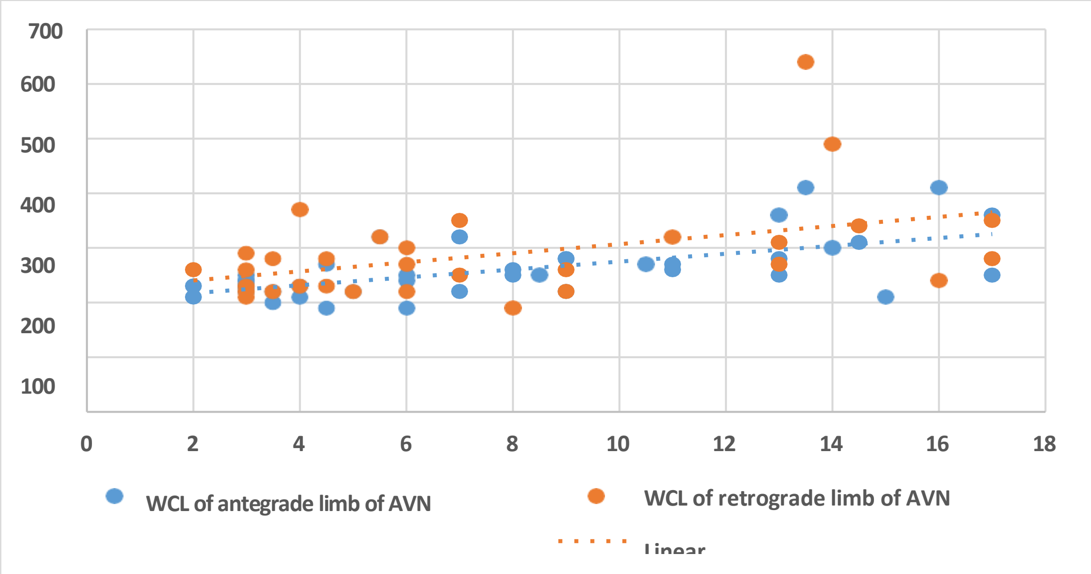

Figure 2: Significant positive correlations between WCL of AVN and age

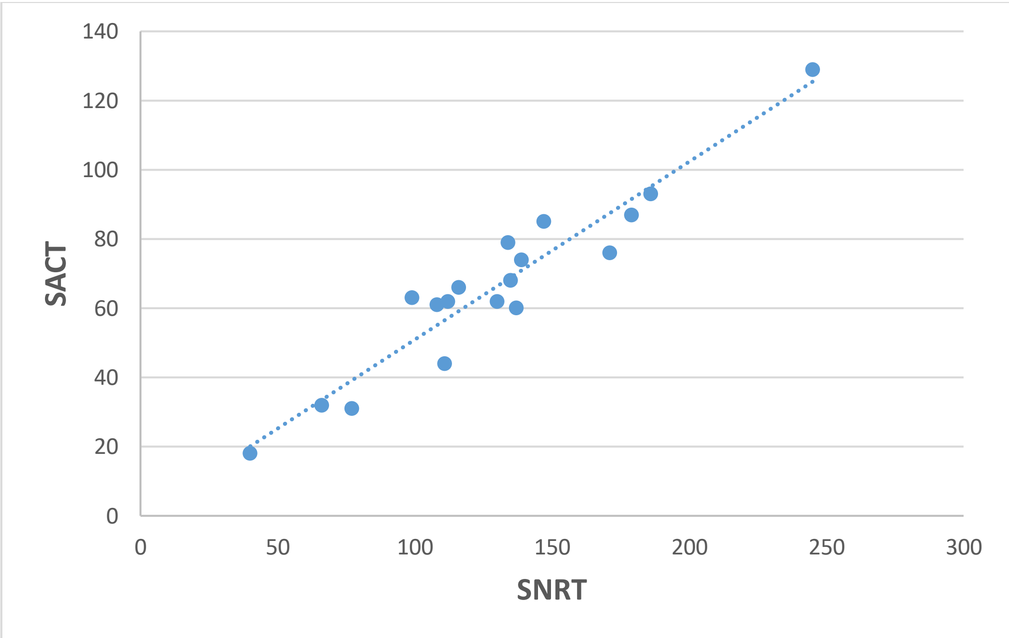

Figure 3: Significant positive correlation between SNRT and SACT

DISCUSSION

CHD is a structural abnormality of the heart and/or great vessels occurring at birth that results in a series of short and long term adverse sequelae ***(van der Linde D et al., 2011)***.It accounts for 3% of neonatal death and 46% of death from all congenital malformations ***(Liu Y et al.,2019).***

The heart is one of the first organs to develop during embryogenesis which is a precisely controlled process. Thus any gene dysregulation result in affecting the whole heart anatomy as well as the conduction pathway ***(Han F et al., 2023).***

The purpose of the electrophysiology study is to describe the electrical properties of the heart based on the anatomy and hemodynamic effect and to determine the sites and mechanisms of any arrhythmias.

We hypothesized that detailed intracardiac mapping for patients with various congenital heart diseases will enable cardiologists and electrophysiologists to detect concomitant cardiac conductive system abnormalities and any hidden accessory pathway. This will help to improve the overall management of the cases.

In our study we mapped and described the cardiac conductive system in pediatric patients with CHD including anatomical site of SAN**,** His bundle, coronary sinus that differ according to lesion itself and assessed the hemodynamic effect of different cardiac lesions on intracardiac measurements with identification of hidden accessory pathway which act as a substrate for arrhythmia.

Our study was conducted among 41 patients with simple or complex CHDs planned for diagnostic or therapeutic cardiac catheter aged 2-18 years, with mean age 8.6 ± 3.61 years. Most of included patients were males representing 63.4% of patients. About 70.7% of patients had simple lesions and 29.3% had complex lesions. Out of 12 complex lesions, 7 had right ventricular predominance (58.3%) and 5 had left ventricular predominance (41.7%). Out of 29 simple lesions, 16 had right side affection (55.2%) and 13 had left side affection (44.8%).

Different cardiac lesions were reported **.**The most frequently reported lesions were ASD in 11 patients (26.8 %), DORV in 6 patients (14.6 %), PDA in 5 patients (12.2 %), COA in 5 patients (12.2%), Other diagnosis of lower frequencies included pulmonary valve stenosis in 3 patients (7.3%) , Tricuspid atresia in 3 patients (7.3%) , VSD in 2 patients (4.9%) , 1 patient (2.4%) for each of the following diseases : pulmonary hypertension , DILV , AS , TOF , unbalanced AVSD (left ventricular dominance) and Ebstein anomaly . Majority of the patients had levocardia 38 patients (92.7%), while 3 patients (7.3 %) had dextrocardia.

Regarding AH interval we observed that patients with simple lesions affecting the right side of the heart had significantly longer A-H interval than patients with lesions affecting the left side. This can be explained that this group includes patients with hemodynamic significant ASD and PS that cause right atrial dilatation. In agreement with our results, ***Bolens M and Friedli B (1984)*** assessed AH interval in cases of secondum ASD before and after surgery and found that AH interval was slightly prolonged before surgery and it decreased significantly postoperatively.

Also , ***Clark E B and Kugler JD (1982)*** conducted a study about preoperative secundum atrial septal defect with coexisting sinus node and atrioventricular node dysfunction and found that five patients had evidence of atrioventricular nodal dysfunction with prolonged AH interval or abnormal atrial pacing rate at which atrioventricular Wenckebach occurred.

A Comparative study of Intracardiac Conduction Intervals in normal children and children with Congenital Heart Disease reported that the A- H interval was found to be significantly prolonged in patients with pulmonic stenosis who had high grade RV pressure overload which affect the right atrium resulting in right atrial enlargement. However, they reported that AH time was normal in patient with secondum ASD and explained that finding that interatrial conduction delay is represented by PA time rather than AH time, This is in contrast to their explanation about the cause of prolonged AH time in cases with pulmonary stenosis which is right atrial enlargement, the same hemodynamic effect of ASD ***(Levin A R., et al 1977).***

A study about His bundle recording in children with normal heart and congenital heart diseases (VSD, TOF, PS, ASD and complex lesions) was conducted by ***Roberts and Olley*** and they reported that no significant changes were detected among different cardiac lesions suggesting that in the studied groups, intervals were not influenced by the lesion. However, they explain there contrary to other studies that report prolonged interatrial conduction in ASD by the small ASD lesions included in their study which cause less hemodynamic effect on the heart ***(Roberts NK and Olley PM., 1972).***

In our study , we found that H-V interval in patients with simple lesions affecting the left side of the heart was not significantly different from H-V interval in patients with simple lesions affecting the right side, however mean value in left sided was longer than right sided simple lesions , this may contributed to size of lesions enrolled in this group which didn't cause significant hemodynamic effect on the heart and this in agreement with Comparative study of Intracardiac Conduction Intervals in Children with Congenital Heart Disease which reported that the H-V interval was prolonged in two groups of patients, both of which had marked hemodynamic insult to the LV either exhibiting high LV pressure overload in patients having sever aortic stenosis or LV volume overload in patients having severe aortic or mitral valve incompetence . The reason for this finding that chronic endocardial trauma due to LV volume or pressure overload may compromise conduction in the left bundle branch (which lies immediately beneath in the subendocardium). Depolarization of the left septum, which is normally the earliest part of the LV to be activated, may be delayed and cause prolongation of the H-V interval ***(Levin A R., et al 1977).***

Our study showed that left ventricular predominance complex lesions had significantly lower H-V interval than right ventricular predominance complex lesion this was explained by three of the five cases included in the group of left ventricular predominance lesion had tricuspid atresia .This come in agreement with Electrophysiologic Studies in Tricuspid Atresia that analyzed his bundle recording in five patients with tricuspid atresia and found that the H-V interval was short in three patients and normal in two patients. The short HV interval was explained by the posterior fibers of the left bundle branch that gave off early and the bundle was shorter than normal. Such an anomalous course of the bundle and early origin of the posterior fascicle can give rise to early activation of the postero-inferior part of the left ventricle ***(Serratto M and Pahlajani DB., 1978).***

Our study showed significant positive correlation between A-H interval and PR interval this is explained by that PR interval spans the time required for the propagating impulse to advance from the atria through the AV node, bundle of His, bundle branches, and the system of Purkinje fibers until the ventricular myocardium begins to depolarize and AH interval represents conduction time from the low right atrium at the interatrial septum through the AV node to the His bundle thus AH time is included in PR interval and any prolongation of AH time should increase PR interval.

There is also significant positive correlation between WCL of AVN and age; this suggests that as patients grow older, the WCL of the AV node tends to increase. This may be explained by several physiological and pathological mechanisms. Firstly, structural and electrical remodeling of the heart occurs progressively with age, particularly in patients with CHD. These changes may include fibrosis, heart chambers enlargement, and alterations in myocardial tissue composition, which can affect conduction properties and lead to prolonged refractory period in the AV node.

Similarly **, DuBrow IW and his colleagues (1976)** studied the influence of age on cardiac refractory periods in man from infant to adult divided the included cases into 6 groups and found that the younger group tended to have shorter values than the older groups ***(DuBrow IW ., et al 1976).***

Our study showed that SNRT correlated significantly to SACT, although they assess different aspects properties where SNRT assesses automaticity while SACT assesses conduction, however in patients with sinus node dysfunction correlation is accepted as the mechanism is common including histopathological changes like fibrosis. .

In our study, we found that all patients with dextrocardia had coronary sinus and His bundle located in abnormal position compared to normal anatomy.

CONCLUSIONS

Patients with CHD often show abnormal conduction patterns due to structural malformations, functional and remodeling changes affecting the conduction tissue. Familiarity with fluoroscopic anatomy in patients with dextrocardia and abnormal situs is important for catheter manipulation.

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List of abbreviation:
AS: aortic stenosis
ASD: atrial septal defect
AVN: atrioventricular node
CHD: congenital heart diseases
COA: coarctation of aorta
CSNRT: corrected sinus node recovery time
DILV: double inlet left ventricle
DORV: double outlet right ventricle
ERP: effective refractory period
PDA: patent ductus arteriosus
PS: pulmonic stenosis
SACT: Sinoatrial conduction time
SAN: sinoatrial node
TOF: tetralogy of Fallot
VSD: Ventricular septal defect
WCL: Wenckebach cycle length

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