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Prevalence of Congenital Heart Defects in Metropolitan Atlanta, 1998–2005

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Abstract

Objective—To determine an accurate estimate of the prevalence of congenital heart defects (CHD) using current standard diagnostic modalities.

Study design—We obtained data on infants with CHD delivered during 1998–2005 identified by the Metropolitan Atlanta Congenital Defects Program, an active, population-based birth defects surveillance system. Physiologic shunts in infancy and shunts associated with prematurity were excluded. Selected infant and maternal characteristics of the cases were compared with those of the overall birth cohort.

Results—From 1998–2005 there were 398 140 births, of which 3240 infants had CHD, for an overall prevalence of 81.4/10 000 births. The most common CHD were muscular ventricular septal defect, perimembranous ventricular septal defect, and secundum atrial septal defect, with prevalence of 27.5, 10.6, and 10.3/10 000 births, respectively. The prevalence of tetralogy of Fallot, the most common cyanotic CHD, was twice that of transposition of the great arteries (4.7 vs. 2.3/10 000 births). Many common CHD were associated with older maternal age and multiple-gestation pregnancy; several were found to vary by sex.

Conclusion—This study, using a standardized cardiac nomenclature and classification, provides current prevalence estimates of the various CHD subtypes. These estimates can be used to assess variations in prevalence across populations, time or space.

Keywords

congenital heart defects; cardiovascular malformations; surveillance; prevalence

Congenital heart defects (CHD) are the leading cause of infant mortality associated with birth defects (1) and can result in chronic disability, morbidity, and increased health-care costs (2, 3). It is therefore important to derive population-based estimates of the prevalence of CHD.

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Most CHD prevalence estimates are based on data from population-based birth defects surveillance systems; a review by Hoffman and Kaplan (4) reported the inter-quartile range of prevalence estimates across 44 international studies for the common forms of CHD. For all types of CHD combined, the inter-quartile range was 60 to 105 CHD per 10 000 births (4). Routine utilization of echocardiography has significantly enhanced the ability to diagnose CHD, including minor abnormalities in asymptomatic infants. Hoffman and Kaplan concluded that much of the observed variability across the 44 studies reflected differences in ascertainment of minor CHD and that recent estimates suggesting an increase in CHD prevalence were attributed to these temporal trends in diagnosis and ascertainment (4).

Another important source of variation is the nomenclature and classification systems used in the prevalence studies, including the International Classification of Disease, 9^{th} *Revision* (ICD-9), the modified version of the British Pediatric Association system; and the International Society of Cardiology. A recent development that should help reduce this variation is the creation of a standard CHD nomenclature, the International Paediatric and Congenital Cardiac Code (5–7), developed by members of the Society of Thoracic Surgeons (STS) and the European Association for Cardio-Thoracic Surgery. The Metropolitan Atlanta Congenital Defects Program (MACDP), a population-based birth defects surveillance system in Atlanta, recently completed a classification of heart defects in its database (8,9). All cases underwent detailed review by pediatric cardiologists to classify CHD according to the standard nomenclature used by the STS congenital heart surgery database. Because there are no published data on the prevalence of CHD based on the STS classification system, we used these MACDP data from the years 1998–2005 to estimate the prevalence of CHD as a benchmark for future studies. As a frame of reference, we compared these data with the inter-quartile ranges published in Hoffman and Kaplan's review.

Methods

MACDP is a population-based surveillance system for major structural birth defects, chromosomal abnormalities, and clinical syndromes established in 1967 by the Centers for Disease Control and Prevention (CDC), Emory University, and the Georgia Mental Health Institute (8). Since that time, the program has conducted surveillance for birth defects among liveborn and stillborn infants greater than or equal to 20 weeks gestation born to residents of the five central counties of metropolitan Atlanta through the use of active case-finding methods and multisource ascertainment. If an estimate of gestational age is not available, the birth weight must be at least 500 grams. Active case finding includes ascertaining birth defects at birthing hospitals using labor and delivery logs, nursery logs, pediatric logs, neonatal intensive care unit logs, postmortem or pathology logs, miscarriage logs, and stillbirth records. A second source of information involves reviewing records from hospitals that a child might visit after being discharged from the newborn nursery. For example, since 1995, records at a local pediatric cardiology center have been routinely reviewed to enhance clinical information about existing MACDP cases. Other sources include reports from cytogenetic laboratories and vital records. Denominator data on the number of live births in the 5 counties were obtained from the Office of Health Information and Policy, Georgia Division of Public Health.

Defect Definitions and Classifications

Records of all infants with a CHD diagnosis in MACDP were previously reviewed and classified by a team of pediatric cardiologists (9). Classification was done to succinctly describe the cardiac lesions and to facilitate CHD monitoring by grouping individual defects based on current understanding of embryonic origins (10). This novel use of STS nomenclature and CHD classification, with potential advantages over standard administrative coding systems, has been outlined previously (9). Importantly, the tri-level classification schema allows for

reorganizing higher aggregation levels (Levels 2 and 3), while maintaining the specificity of STS codes (Level 1). Using the STS nomenclature, most cases (89%) had only one CHD. However, if a case had more than one independent lesion, each CHD was counted separately. Furthermore, infants with transient newborn cardiac conditions and those with shunts associated with prematurity, were classified as physiologically normal.

For the purpose of comparison to previously reported CHD prevalence, the classified CHD aggregates were rearranged into four major groups: 1) left-to-right shunts, 2) cyanotic lesions, 3) left heart obstructive lesions, and 4) right heart obstructive lesions. The definition of these groups, with inclusion and exclusion criteria, is presented in Table I.

Similar to the Hoffman and Kaplan study (4), we also identified a group of infants with "critical" CHD that included all the cyanotic defects listed above, as well as hypoplastic left heart syndrome and pulmonary atresia. Because MACDP cases were not classified based on severity of CHD, we were unable to discern mild from severe occurrences of a particular lesion. Thus, those CHD that have a spectrum of severity, such as valvar aortic stenosis or coarctation, were not included as "critical" CHD.

Statistical Analyses

Prevalence estimates are reported per 10 000 live births. Whereas the numerators for estimates of prevalence for total CHD and critical CHD represent the *number of affected infants and fetuses*, for estimates of prevalence for specific cardiac phenotypes, the numerators represent the number of infants with *specific cardiac defects* regardless of whether the affected infants had one or more independent cardiac defects. We include the interquartile range of prevalence estimates for most of the common forms of CHD used by Hoffman and Kaplan for reference (4) (Table II). Because of the rarity of several of the CHD examined, we used the nonparametric Mann Whitney U test to evaluate whether the distributions of gestational age, birth weight and maternal age for specific CHD phenotypes differed from the respective characteristics of the birth cohort, and Fisher exact test to evaluate whether the proportions of multiple births and males for each phenotype differed from the birth cohort.

Results

During 1998–2005 there were 3240 infants identified with CHD and 398 140 live births in metropolitan Atlanta. The overall prevalence of CHD was 81.4 infants/10 000 births, and the prevalence of critical CHD was 15.6 infants/10 000 births (Table II). Because some infants had more than one independent CHD, the sum of individual CHD prevalence is greater than the total CHD prevalence.

The left to right shunt lesions were the most prevalent group of defects, comprising over half of the total number of CHD (Table II). The most common defect was muscular VSD occurring at a prevalence of 27.5/10 000 births. This prevalence was over twice that of the next two most common cardiac defects (perimembranous VSD at 10.6 and secundum ASD at 10.3/10 000 births). As a group, the prevalence of ASDs was somewhat higher than the range published by Hoffman and Kaplan. Conversely, the prevalence of PDA was on the lower end of predicted at 2.9/10 000. As a group, the full spectrum of AVSD occurred at a prevalence of 4.1/10 000 with complete AVSD comprising roughly half of the total group (2.2/10 000). Of the 88 children with complete AVSD (excluding those with heterotaxy syndrome), 70 (80%) had Down syndrome.

In the cyanotic CHD group, there were two predominant defects: TOF and TGA. TOF was the most common and twice as prevalent as TGA (4.7/10 000 births vs 2.3/10 000 births,

respectively). The prevalence of all other cyanotic defects combined was 5.5/10 000 births (Table II).

Among the obstructive CHD lesions, coarctation of the aorta (4.4/10 000) and HLHS (2.3/10 000) were the two most common left heart obstructive defects, and pulmonary valvar stenosis (5.5/10 000) was a common right heart obstructive defect. All of these were within previously reported estimates. However, the observed prevalence of aortic valvar stenosis (1.1/10 000) and pulmonary atresia with intact septum (0.4/10 000) were below the lower quartiles reported by Hoffman and Kaplan (Table II).

Many infants with CHD were born at a lower gestational age compared with births in the general population (Table III). This was evident for most of the left to right shunt lesions (perimembranous, muscular, and subarterial VSD, secundum ASD, AVSD, and PDA), many of the cyanotic CHD, all of the left heart obstructive lesions, and valvar pulmonic stenosis. Similarly, most of the infants with CHD had significantly lower birth weights relative to the general population. Increased maternal age also appeared to be associated with certain CHD.

Perimembranous and muscular VSD, secundum ASD, AVSD, tetralogy of Fallot, coarctation of the aorta, and valvar pulmonic stenosis were all associated with increased maternal age relative to the overall population. Multiple gestation pregnancy was more common for infants with perimembranous and muscular VSD, secundum ASD, truncus arteriosus, coarctation of the aorta, and valvar pulmonic stenosis. Several cardiac defects were seen more frequently in girls, including muscular VSD, secundum ASD, AVSD, PDA, as well as Ebstein's anomaly and heterotaxy syndrome. Conversely, the diagnoses of TOF, TGA, and TAPVR were more common among boys (Table III).

Discussion

Our study provides prevalence estimates for several CHD which were not previously discussed in the Hoffman and Kaplan review (4) (Table II), including subtypes of septal defects and rare defects such as discordant atrioventricular connections (congenitally corrected TGA), heterotaxy syndrome, and interrupted aortic arch type B, using the STS nomenclature and classification system. The most common defects were muscular VSD, perimembranous VSD, and secundum ASD with prevalence estimates of 27.5, 10.6, and 10.3/10 000 births, respectively.

The CHD prevalence estimates from this study should be interpreted in light of the completeness of case ascertainment. Although our study included cases of heart defects among live births and stillbirths, and thus a more inclusive group of cases than is usually reported on by most other surveillance programs, MACDP data do not include cases of CHD among pregnancy losses less than 20 weeks of age, stillbirths without autopsies, infant deaths with undiagnosed CHD, and cases of CHD first diagnosed beyond 6 years of age (ie, ASD or aortic valve stenosis associated with bicuspid aortic valve). Because some prenatally diagnosed cases may result in pregnancy termination, and the extent of under-reporting of such pregnancy terminations is unclear, some prenatally diagnosed cases of CHD are likely not included in MACDP data (11;12). Accordingly, our prevalence estimates should be viewed as minimum estimates.

Second, routine use of color Doppler echocardiography has increased diagnosis of minor defects (ie, small VSD, milder forms of pulmonary stenosis, and ASD) in asymptomatic infants and children (4). Indeed, recent prevalence studies have shown a significant preponderance of VSD (4). Further, it is likely that the apparent prevalence of VSD will be affected by the age of diagnosis since many small VSD in asymptomatic infants will close spontaneously in the first year of life. Our study not only confirms the predominance of VSD, but also demonstrates

that the muscular VSD subtype is the most common CHD. Many of these muscular VSDs are likely smaller defects diagnosed in newborns that might have been undetected prior to the routine use of color Doppler echocardiography. Furthermore, studies have also suggested that the prevalence of VSD would be even greater if echocardiography is universally performed, rather than only on infants with suspected CHD (13;14).

Because the nomenclature and case classification approach in our study differs from those used in previous studies, and the impact of such differences on prevalence estimates is unclear, comparisons of our prevalence estimates with those in the literature need to be made with some caution. Also, our CHD classification does not distinguish infants with isolated cardiac defects from those with cardiac defects and other non-cardiac anomalies (ie, chromosomal disorders, well-known syndromes, and other associations). Therefore, further classification efforts are needed to differentiate isolated from different types of multiple defects so that prevalence for such subgroups of CHD can be estimated. Lastly, the STS classification codes do not allow for assessment of severity. As such, we are unable to distinguish mild from severe forms of a given CHD phenotype.

The population of the five-county metropolitan Atlanta differs from the population of Georgia and the United States as a whole, particularly with respect to the proportion of residents living in urban areas (8). Because these characteristics may be associated with variations in the occurrence of birth defects (15), our prevalence estimates based on MACDP data are not necessarily generalizable to other populations.

Given the above caveats, in general, the CHD prevalence estimates in this investigation were comparable to those previously reported (4) (Table II). One important difference was a lower prevalence of PDA than expected. This finding could be due to the strict criteria used to exclude PDA in premature or newborn infants (< 6 weeks old) and to exclude PDA when it occurred as an obligate shunt in the setting of critical CHD (9). Therefore, our data likely reflect a better estimate of the true prevalence of PDA as an independent, congenital cardiac defect. Although the prevalence of VSD as a group was within the range of prevalence estimates noted by Hoffman and Kaplan, comparable data in the literature for the VSD subtypes are limited. Ferencz et al noted a higher prevalence for muscular VSD than for perimembranous VSD (16). However, their estimate for muscular VSD was based on a random sample and not all cases of muscular VSD in the population. Our prevalence estimates for both aortic valvar stenosis and pulmonary atresia with intact septum were lower than that reported in the Hoffman and Kaplan review. It is possible that our criteria (Table I) of considering cases with both coarctation of the aorta and aortic valvar stenosis only in the coarctation group might have reduced the aortic valvar stenosis total used for the prevalence calculations. Interestingly, we observed a TOF prevalence that was twice that of TGA (4.7 vs. 2.3/10 000 births). Although both these estimates were within the Hoffman and Kaplan interquartile range, the predominance of TOF relative to TGA has not been universally reported (17), a discrepancy that is probably due to the classification of double outlet right ventricle lesions. Whereas some prevalence reports consider all double outlet right ventricle variants to be a subtype of TGA, our classification distinguished among the variants, and we included tetralogy of Fallot-type double outlet right ventricle in the TOF group (9).

Possible interpretations of associations of CHD phenotypes with infant and maternal characteristics need to keep in mind a couple of methodologic considerations. One is that because multiple statistical tests were performed, some of the observed statistically significant associations might be due to chance. The other consideration is that the sample size for some of the CHD phenotypes was relatively small. Consequently, we may not have had adequate statistical power to detect some real associations for such cases.

We found associations of CHD with low gestational age and birth weight, consistent with previous reports of altered fetal growth associated with certain cardiovascular malformations (18;19). The mechanisms underlying these associations are unclear, although clearly gestational age and birth weight are highly correlated. One possibility is that the presence of CHD might impair normal fetal growth. Another possibility is that both altered fetal growth and CHD represent co-outcomes associated with a common but yet undefined genetic susceptibility or prenatal exposure (ie, alcohol consumption, infection, nutritional status). This investigation confirms the previously reported association of certain CHD lesions with infant sex (16;20). Muscular VSD, secundum ASD, AVSD, PDA, Ebstein's anomaly, and heterotaxy syndrome were all more common in girls, and TOF, TGA, and TAPVR were more common in boys. Suggestive associations were observed between several other cyanotic CHD lesions and infant sex; however due to the rarity of these defects, we can not conclude with reasonable certainty that these associations are true.

Both older maternal age and multiple-gestation pregnancies were associated with approximately one-third of cardiac defects, including some of the most common CHD lesions reported here, a finding which has been noted in previous studies (16;20;21). Two important demographic changes have occurred in Atlanta over time which may have impacted these findings (8). In 1968, roughly 16% of mothers were ≥ 30 years old, compared with 42% in 2005. Older maternal age has long been recognized as a risk factor for having offspring with genetic syndromes, particularly Downs Syndrome. Older maternal age is also associated with the use of assisted reproductive technology, which is a very strong risk factor for multiple gestation pregnancy (22). In MACDP, the percentage of multiple-gestation pregnancies has nearly doubled from 1.8% in 1968 to 3.4% in 2005, likely due to use of assisted reproductive technology (8). During heart classification, cases were not separated on the basis of genetic syndrome. Thus, although our data suggest that older maternal age could be an independent risk factor for CHD, the CHD prevalence reported here is influenced by inclusion of infants with chromosomal disorders. Further work is warranted to examine the independent and joint effects of maternal age, chromosomal anomalies, assisted reproductive technology, and multiple gestation pregnancy on the risk of CHD. Because the classification of heart defects used a standardized nomenclature and a well documented classification system, it will be possible to compare the results of this study with future studies in order to assess prevalence variations across populations, time or space, or in relation to possible interventions.

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List of Abbreviations

ASD
: Atrial Septal Defect

AVSD
: Atrial Ventricular Septal Defect

CHD
: Congenital Heart Defects

HLHS
: Hypoplastic Left Heart Syndrome

IQR

Inter-Quartile Range

MACDP

Metropolitan Atlanta Congenital Defects Program

PDA

Patent Ductus Arteriosus

STS

Society of Thoracic Surgeons

TAPVR

Total Anomalous Pulmonary Venous Return

TGA

Transposition of the Great Arteries

TOF

Tetralogy of Fallot

VSD

Ventricular Septal Defect

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Table 1

Definitions of congenital heart defects with inclusion and exclusion criteria.

Defect Group	Definition
Left to Right Shunts	
Ventricular Septal Defect (VSD)	Includes Type 2 (perimembranous), Type 4 (muscular), Type 1 (subarterial (conoseptal, infundibular, supracristal), and unspecified types of VSD. Excludes VSD with Interrupted aortic arch type B or cyanotic defects
Atrial Septal Defect (ASD)	Includes secundum, sinus venosus, and unspecified types of ASDt > 4mm in size. Excludes patent foramen ovale and obligatory shunts
Atrioventricular Septal Defect (AVSD)	Includes complete, intermediate, and unbalanced AVSD and those associated with tetralogy of Fallot, ostium primum atrial septal defect, and Type 3 (inlet) ventricular septal defect
Complete Atrioventricular Septal Defect	Includes complete atrioventricular septal defect
Patent Ductus Arteriosus (PDA)	Includes term infants (≥36 weeks gestation) with patent ductus arteriosus persisting for ≥ 6 weeks after delivery. Excludes obligatory shunt lesions or if maintained by prostaglandin infusion.
Cyanotic Congenital Heart Defects	
Tetralogy of Fallot (TOF)	Includes typical TOF, TOF with absent pulmonary valve, pulmonary atresia with ventricular septal defect, pulmonary atresia with major aortopulmonary collateral arteries, and TOF-type double outlet right ventricle.
Transposition of the Great Arteries (TGA)	Includes concordant atrioventricular connections and discordant ventricular arterial connections, with or without ventricular septal defect or left ventricular outflow tract obstruction. Also includes double outlet right ventricle with malpositioned great arteries.
Discordant Atrioventricular Connections	Includes discordant atrioventricular and ventriculo-arterial connections (congenitally corrected transposition of the great arteries), with or without ventricular septal defect or outflow tract obstruction; and discordant atrioventricular with concordant ventriculo-arterial connections
Truncus Arteriosus	Includes all truncus arteriosus subtypes. Excludes hemitruncus and pseudotruncus
Total Anomalous Pulmonary Venous Return	Includes all types of total anomalous pulmonary venous return
Tricuspid Atresia	Includes tricuspid valve atresia with normally related great arteries.
Ebstein's Anomaly	Includes Ebstein's anomaly of the right heart with atrioventricular concordance
Single Ventricle Complex	Includes double-inlet left or right ventricle, mitral or tricuspid valve atresia with aortic malposition, or other specified or unspecified types of single ventricle.
Heterotaxy Syndrome	Includes atrial situs abnormalities, situs inversus with or without dextrodardia, situs ambiguous, isolated dextrocardia, and unspecified heterotaxy. Cases with other heart defects and heterotaxy syndrome are only considered in the heterotaxy group, and excluded from the other heart defect groups
Left Heart Obstructive Defects	
Coarctation of the Aorta	Includes coarctation of the aorta with or without aortic valve stenosis, aortic arch hypoplasia, and interrupted aortic arch type A
Valvar Aortic Stenosis	Includes valvar or unspecified aortic valve stenosis, dysplasia, or atresia Excludes isolated bicuspid aortic valve, and aortic stenosis with coarctation
Interrupted Aortic Arch Type B	Includes type B and unspecified type of interrupted aortic arch.
Hypoplastic Left Heart Syndrome (HLHS)	Includes HLHS with or without ventricular septal defect
Right Heart Obstructive Defects	
Valvar Pulmonic Stenosis	Includes valvar and unspecified pulmonary stenosis, or valvar dysplasia
Pulmonary Atresia	Includes pulmonary atresia with intact ventricular septum.

Table II

Congenital heart defect prevalence per 10 000 live births. Comparison of 1998–2005 Atlanta estimates with previous estimates.

	1998–2003 Atlanta		Hoffman & Kaplan[a]
	N	Prevalence	Prevalence IQR
Left to Right Shunts			
Ventricular Septal Defect	1665	41.8	17.6–44.8
Perimembranous Ventricular Septal Defect	423	10.6	-
Muscular Ventricular Septal Defect	1096	27.5	-
Subarterial Ventricular Septal Defect	20	0.5	-
Ventricular Septal Defect NOS[b]	126	3.2	-
Atrial Septal Defect	523	13.1	3.7–10.6
Secundum Atrial Septal Defect	411	10.3	-
Sinus Venosus Atrial Septal Defect	15	0.4	-
Atrial Septal Defect NOS[b]	97	2.4	-
Atrioventricular Septal Defect	163	4.1	2.4–4.0
Complete Atrioventricular Septal Defect	88	2.2	-
Patent Ductus Arteriosus	114	2.9	3.2–7.8
Cyanotic Congenital Heart Defects			
Tetralogy of Fallot	189	4.7	2.9–5.8
Transposition of the Great Arteries	90	2.3	2.3–3.9
Discordant Atrioventricular Connections	10	0.3	-
Truncus Arteriosus	24	0.6	0.6–1.4
Total Anomalous Pulmonary Venous Return	31	0.8	0.6–1.2
Tricuspid Atresia	19	0.5	0.2–1.2
Ebstein's Anomaly	24	0.6	0.4–1.6
Single Ventricle Complex	41	1.0	0.5–1.4
Heterotaxy Syndrome	68	1.7	-
Left Heart Obstructive Defects			
Coarctation of the Aorta	177	4.4	2.9–4.9
Valvar Aortic Stenosis	45	1.1	1.6–3.9
Interrupted Aortic Arch Type B	15	0.4	-
Hypoplastic Left Heart Syndrome	91	2.3	1.5–2.8
Right Heart Obstructive Defects			
Valvar Pulmonic Stenosis	220	5.5	3.6–8.4
Pulmonary Atresia	17	0.4	0.8–1.5
Critical CHD[c]	621	15.6	10.8–15.3
All CHD[c]	3240	81.4	60.2–105.7

[a] Adapted from Hoffman JI, Kaplan S. Incidence of Congenital Heart Disease. *J Am Coll Cardiol*. 2002;39:1890–1900.

[b] NOS = Not otherwise specified

[c] Prevalence for critical CHD and all CHD is based on the number of infants and fetuses, not on the number of defects.

Table 3

Comparison of gestational age, birth weight, maternal age, multiple births, and sex for infants and fetuses with CHD relative to the cohort of all live births in metropolitan Atlanta, 1998–2005.

	Mean Gestational Age	Mean Birth Weight	Mean Maternal Age	Percent Multiple Births	Percent Male
Birth Cohort	**38.8**	**3262**	**28.0**	**3.4**	**50.9**
Left to Right Shunts					
Perimembranous Ventricular Septal Defect	37.1*	2845*	29.6*	6.6*	48.7
Muscular Ventricular Septal Defect	37.7*	3156*	29.3*	6.7*	45.3*
Subarterial Ventricular Septal Defect	36.4*	2930	27.8	5.0	65.0
Ventricular Septal Defect, NOS	35.0*	2415*	28.4	7.1*	53.2
Secundum Atrial Septal Defect	35.7*	2629*	29.2*	9.0*	41.4*
Sinus Venosus Atrial Septal Defect	38.0	3141	27.9	13.3	66.7
Atrial Septal Defect, NOS	36.0*	2631*	27.8	4.1	47.4
Atrioventricular Septal Defect	36.1*	2569*	31.8*	6.1	39.9*
Complete Atrioventricular Septal Defect	36.1*	2550*	32.6*	5.7	40.9
Patent Ductus Arteriosus	38.0*	3056*	28.9	3.5	35.1*
Cyanotic Congenital Heart Defects					
Tetralogy of Fallot	36.9*	2665*	29.2*	5.3	60.3*
Transposition of the Great Arteries	38.5	3173	29.1	3.3	62.2*
Discordant Atrioventricular Connections	38.6	3200	27.2	0.0	70.0
Truncus Arteriosus	36.3*	2550*	27.6	12.5*	58.3
Total Anomalous Pulmonary Venous Return	38.1*	3006*	27.8	6.5	83.9*
Tricuspid Atresia	37.9	2949	28.4	0.0	63.2
Ebstein's Anomaly	36.8*	3160	28.6	0.0	25.0*
Single Ventricle Complex	36.5*	2781*	27.8	4.9	48.8
Heterotaxy Syndrome	37.0*	2921*	27.4	1.5	36.8*
Left Heart Obstructive Defects					
Coarctation of the Aorta	37.7*	2970*	29.4*	8.5*	54.8
Valvar Aortic Stenosis	37.0*	2841*	29.1	6.7	62.2
Interrupted Aortic Arch, Type B	37.8*	2866*	30.1	0.0	53.3
Hypoplastic Left Heart Syndrome	36.1*	2814*	28.0	3.3	49.5
Right Heart Obstructive Defects					
Valvar Pulmonic Stenosis	35.8*	2731*	29.2*	8.2*	46.4
Pulmonary Atresia	37.3	2748*	26.2	11.8	58.8

* $p < 0.05$