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Management of an Associated Ventricular Septal Defect at the Time of Coarctation Repair

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Abstract

Background—Management of a ventricular septal defect (VSD) at time of coarctation of the aorta (CoA) repair remains controversial, with recent studies advocating concomitant repair of both defects. We evaluated the surgical management and mortality for patients undergoing CoA repair associated with a VSD.

Methods—We retrospectively reviewed data submitted to the Pediatric Cardiac Care Consortium of patients undergoing repair of CoA from 1982 to 2007. The cohort was divided into three groups: CoA repair plus VSD closure (group 1); CoA repair plus pulmonary artery band (group 2); and CoA repair without repair of VSD (group 3). Variables reviewed included era, age, and weight at repair, and in-hospital mortality.

Results—There were 7,860 patients who underwent repair of CoA, of whom 2,022 had an associated VSD (25.7%). Mortality after CoA repair with and without an associated diagnosis of VSD was 8.3% versus 2.1% ($p < 0.001$). Mean age at repair for group 1 ($n = 286$) and group 2 ($n = 472$) was 87.4 days and 21.6 days, respectively ($p = 0.004$), and median weight was 3.31 kg and 3.30 kg, respectively ($p = 0.130$). Discharge mortality for group 1 and group 2 was similar, at 8.7% and 9.1%, respectively ($p = 0.852$). Patients with CoA/VSD who had neither VSD closure nor pulmonary artery banding (group 3) had a hospital mortality of 7.9%.

Conclusions—The association of CoA and VSD is common. A strategy of concomitant VSD closure at CoA repair does not result in worse discharge mortality when compared with pulmonary banding with anticipated staged repair of the VSD. These outcomes support continued evaluation of a one-stage approach.

The association of coarctation of the aorta (CoA) with a ventricular septal defect (VSD) in neonates is common. Repair of CoA early in infancy is indicated for most patients. The optimal surgical strategy for the management of a hemodynamically significant VSD at the time of primary repair of coarctation of the aorta (CoA) remains controversial. Historically, a two-stage approach has been favored. The first-stage procedure consisted of CoA repair and pulmonary artery banding (PAB) through a left thoracotomy, followed at a subsequent admission with closure of the VSD through a median sternotomy [1, 2]. Many centers now

favor a single-stage approach, with repair of both the CoA and the VSD through a single incision using a median sternotomy and cardiopulmonary bypass (CPB) with low flow cerebral perfusion or total hypothermic circulatory arrest [3–5].

The aim of this study was to identify evolving trends in surgical management and compare in-hospital mortality rates between the single-stage and two-stage approach for patients undergoing repair of CoA with an associated VSD.

Patients and Methods

Data Source

Data utilized in this analysis were covered by the data use agreement of the Pediatric Cardiac Care Consortium (PCCC) provided from individual member institutions. The study was approved by the Institutional Review Board at the University of Minnesota, with an exception to obtain individual patient consent.

Patient Population

A retrospective review of PCCC data was performed to identify all patients whose data were submitted with the procedural code for repair of CoA between 1982 and 2007. Institutions that participated during this period are presented in Table 1. An evaluation of all available variables was undertaken to identify potential risk factors for in-hospital mortality. As a result of this analysis, a review was then conducted of all patients who underwent primary repair of CoA with a secondary diagnosis code for VSD at less than 12 years of age. With the exception of atrial septal defect and patent ductus arteriosus, all other associated cardiac anomalies were excluded from this analysis. The cohort was then stratified based on the surgical management strategy for the associated VSD. Group 1 consisted of all patients who underwent concomitant repair of both the CoA and the VSD. Group 2 consisted of all patients who underwent repair of CoA with placement of PAB. Group 3 included all patients who underwent repair of CoA without either closure of VSD or PAB.

Data Collection

Patient data available in the PCCC included sex, age at operation, weight at operation, era, in-hospital mortality, postoperative length of stay, and one-incision versus two-incision repairs. The size of the VSD in all patients was stratified into small (less than 4 mm), moderate (4 to 7 mm), and large (greater than 7 mm), and undefined categories. The primary outcome was in-hospital mortality and was defined as all in-hospital deaths after initial CoA repair. A secondary outcome for in-hospital deaths after second hospitalization for staged closure of VSD, PAB removal, or both, was also included.

Categorical variables were reported as absolute numbers and percentages. Continuous variables were expressed as means and standard deviations along with medians and interquartile ranges owing to the positive skew in the data. Between group analyses were conducted using the χ^2 test for categorical variables and the Wilcoxon rank sum test for continuous variables. Multivariable logistic regression was used to evaluate the association between potential risk factors and in-hospital mortality while adjusting for age at repair,

operative weight, operation year, single-stage VSD repair, PAB, and aortic arch diagnosis. Odds ratios along with 95% confidence intervals (CI) were used to evaluate the association between the variables in the model and in-hospital mortality. No adjustments were made for multiple comparisons. All statistical analyses were completed using the R Project for Statistical Computing, version 2.14.1 [6].

Results

The data of 7,860 patients were submitted to the PCCC with the procedural code for repair of CoA. The in-hospital mortality for the entire cohort was 4.2%. There were 2,022 patients (25.7%) who had an associated diagnostic code for a VSD. When comparing the repair of CoA in patients without associated cardiac anomalies (except atrial septal defect or patent ductus arteriosus) to the patients with an associated VSD, the mortality was significantly lower (2.1% versus 8.3%, respectively; $p < 0.001$). A multivariable analysis of the entire CoA cohort revealed several variables that were significant for mortality (Table 2). It is noteworthy that the presence of a VSD in association with the primary repair of CoA was an independent risk factor for in-hospital mortality, and that is the foundation for this overall review. Also, for the entire cohort ($n = 7,860$), there was a significant decrease in discharge mortality when comparing the most recent decade (3.1%) to the immediately preceding period (6.3%, $p < 0.005$).

There were 1,117 (55%) males and a total of 236 patients (12%) considered premature at birth. Sixty-nine percent of patients ($n = 1,389$) underwent repair of the CoA at less than 30 days of age, with an additional 25% ($n = 506$) being repaired before 1 year. Characteristics of the three groups are presented in Table 3. The median age at repair for group 1, group 2, and group 3 was 13.0, 12.0, and 15.0 days, respectively ($p = 0.005$). The median weight at repair for group 1, group 2, and group 2 was 3.35 kg, 3.30 kg, and 3.50 kg, respectively ($p = 0.0130$). The postoperative length of stay was similar for all groups. In group 1, 269 (92.6%) underwent repair through a median sternotomy only. Few patients (7%) underwent a combined approach utilizing both sternotomy and a left lateral thoracotomy. The discharge mortality for groups 1, 2, and 3 were comparable at 8.7%, 9.1%, and 7.9%, respectively ($p = 0.852$). Table 4 stratifies the cohort based on the management strategy undertaken and the era evaluated. Over the 2 decades from the 1990s to the end of 2000, there was a significant decrease in the mortality for the single-stage repair (18.8% versus 4.7%, $p < 0.05$). In addition, during the most recent decade reviewed, mortality for the single-stage strategy was significantly lower than for patients who received palliation with a PAB (4.7% versus 7.0%, $p < 0.05$). This decrease in mortality occurred with a significant increase in the number of single-staged repairs in patients who had the VSD addressed at the time of CoA repair.

The cohort ($n = 2,022$) was broken down based on the size of the VSD at the time of CoA repair and the management strategy used (Table 5). Of patients who underwent a surgical intervention to address the associated VSD, 81.5% had at least a moderate size VSD, and of those undergoing repair of CoA in which the VSD was not addressed, 48.8% had a moderate to large VSD. This percent is likely lower, as 18.4% of patients in group 3 had an unspecified VSD size recorded.

Patients who underwent CoA with PAB (group 2) who returned on a subsequent visit for PAB removal without VSD closure ($n = 59$) had an in-hospital mortality rate of 11.9% ($n = 7$). The median time from discharge to the second admission was 7.46 months (interquartile range, 4.13 to 13.66). Patients who underwent CoA with PAB and returned for VSD closure ($n = 220$) had an in-hospital mortality rate of 4.5% ($n = 10$), but only 1 death of 53 patients (1.9%) occurred in the most recent era (2000 to 2007). There were 445 patients (35%) in group 3 who returned for closure of the associated VSD at a median time of 63.0 days (interquartile range, 0 to 5,958) after primary repair of the CoA. The discharge mortality associated with delayed closure of the VSD was 9.4% ($n = 42$). Clearly, patients may have had subsequent closure of the VSD at institutions that did not participate in the PCCC, implying that the incidence of patients returning for VSD closure is underestimated. Of patients who did return, 69.0% ($n = 307$) had at least a moderate sized VSD at the time of primary repair.

Comment

The optimal surgical management of infants presenting with CoA and an associated VSD remains controversial. When compared with repair of an isolated CoA, the presence of a VSD is associated with increased discharge mortality regardless of the operative strategy employed. The reason for this is likely multifactorial and somewhat related to the era from which the data were collected. This point has been shown in several single-center studies that review experiences over an extended period [5]. Our data reveal similar results, with improvement in overall surgical results from complex repairs of this combination of anatomic defects over the eras studied.

Traditionally, several possible management strategies have been utilized to address an associated VSD at the time of CoA repair. These include a conservative approach to the VSD, treating the resultant heart failure medically; closures at the time of repair; or palliation with a PAB. The decision as to which strategy to employ had been traditionally based on the preoperative studies relating to the size of the VSD, institutional comfort levels with more extensive neonatal repair, and conflicting published information supporting several approaches [7–9].

Brouwer and colleagues [10] published their institutional results of 80 infants less than 3 months of age with CoA and VSD who had undergone repair by either a single-stage single incision ($n = 16$) or a two-stage approach ($n = 64$). The early mortality in the two-stage group was 4.7% compared with 18.8% early mortality in the single-stage single incision group. Freedom from recoarctation in the two-stage group at 5 years was 91.3%, compared with freedom from recoarctation in the single-stage group of only 60% ($p = 0.018$) [10]. In a multi-institutional project conducted by the Congenital Heart Surgeons Society, patients undergoing a two-stage repair demonstrated improved survival compared with a single-stage approach [11]. The study found that a two-stage repair consisting of correction of the CoA with PAB followed by a later operation of VSD closure with removal of PAB, resulted in 2-year survival of 97%. This compared with 2-year survival of 64% for the patients who underwent combined single-stage repair of CoA and VSD. Again, the limitation with interpreting these results has been the era-dependent improvement in the overall experience

with neonatal surgery. Nevertheless, these results set the standard for the management strategy of this combination of defects.

In the present study, the overall discharge mortality for patients undergoing a combined approach was 8.7%, compared with 9.1% for a staged strategy (p 0.85). Indeed, mortality was the lowest among patients undergoing repair concomitantly during the most recent period studied. These findings suggest that mortality for primary single-stage repair was comparable to that for patients who underwent a palliative procedure and anticipated return for closure of the VSD.

Advantages of the two-stage approach include avoiding exposure to cardiopulmonary bypass early in life. Reconstruction of the aortic arch through a median sternotomy will often also require either total hypothermic circulatory arrest or low-flow isolated cerebral perfusion. There is also an obligatory need for blood and blood products when these repairs are carried out in the neonate [12]. By deferring closure of the VSD to a later date while addressing the CoA, these detrimental effects of cardiopulmonary bypass may be avoided. A staged procedure to close the VSD is performed with reduced exposure to the bypass circuit, short periods of cardioplegic arrest of the heart, closure in a larger child with potentially less risk of complete heart block, and shorter postoperative courses.

Disadvantages of a two-staged palliative strategy include the hemodynamic burden of the uncorrected anomaly, scarring of the pulmonary artery requiring reconstruction with artificial material, development of both left and right ventricular outflow tract obstruction, and the postoperative risks associated with any repetitive operative intervention. However, patients presented in this study who underwent CoA with PAB who returned for VSD closure ($n = 220$) had a discharge mortality rate of 4.5% ($n = 10$), but only 1 death in 53 patients (1.89%) occurred in the most recent era (2000 to 2007). Despite this improved outcome in the most current era, the added mortality of a second procedure is still a distinct disadvantage of the two-stage approach.

As previously reviewed, the use of a single-stage approach with repair of CoA and VSD through a median sternotomy historically has had suboptimal results compared with the two-stage approach [1, 2]. However, in more recent published series of patients undergoing a single-stage repair for CoA and VSD at the Children's Hospital of Philadelphia, Gaynor and colleagues [13] presented 25 patients less than 3 months of age who were repaired in a single operation through a median sternotomy with use of total circulatory arrest. Early mortality was 4%, with no late deaths [13]. Other recent experiences with the single-stage approach have also documented improved outcomes with this strategy [4, 14–18]. These studies describe single-stage repairs using deep hypothermia with either total circulatory arrest or selective low-flow cerebral perfusion through the innominate artery [19, 20]. In this study, patients undergoing a single-stage procedure had an overall discharge mortality similar to that of patients who were discharged with either a PAB or medical management of the VSD only.

Several studies have suggested that complete repair of both anomalies can be addressed at the same operative time using two separate incisions [3]. Kanter and associates [21]

reviewed 36 patients who underwent one of three strategies for correction of this combination of defects. Of the 26 patients who had repair of both defects completed during the same operative setting, 15 were repaired through two incisions with a left lateral thoracotomy and a median sternotomy. There were no significant differences in age at repair, size of the proximal and distal aortic arch, or need for prostaglandin between the two groups. The need for total circulator arrest, total bypass time, total cardiac arrest time, and hospital stay were all significantly less in the two-incision group. In our study, fewer than 7% of patients undergoing a single-stage repair had this accomplished through two incisions. We can postulate several reasons for the infrequent use of this approach, including the concerns of not being able to address more proximal hypoplasia of the aortic arch, hemorrhage of a distal suture line with need for systemic heparinization, and infectious complications with two surgical incisions. Given the excellent results with this approach, use of the single-stage two-incision repair may increase in the future. However, the incidence of recoarctation requiring reintervention will still need to be analyzed for all surgical strategies to determine the best approach to consistently achieve optimal long-term outcomes in patients with CoA and VSD.

Study Limitations

This is a retrospective study reviewing three cohorts of patients over three eras and has the inherent limitations of a retrospective multi-institutional database review. These limitations include lack of consistent surgical outcomes between institutions, bias based on individual surgical strategies, and lack of consistent follow-up after primary operative interventions. In addition, no long-term follow-up is available for patients once they are discharged from the institution at which the index operation was performed. Also, inconsistencies in the reporting of the degree of aortic arch hypoplasia became evident during the review, making any conclusions difficult to attain.

In conclusion, the optimal surgical strategy for the management of a hemodynamically significant VSD at the time of primary repair of CoA remains controversial. The presence of a VSD in patients with CoA is associated with higher mortality rates regardless of surgical management strategy. A strategy of primary VSD repair with CoA repair as a single-stage approach does not appear to be associated with worse in-hospital survival compared with a PAB with staged repair. Furthermore, delayed repair of VSD and removal of PAB after initial CoA repair are both associated with additional mortality at the second procedure in a two-stage approach. Our study supports further evaluation of the single-stage approach as a preferred surgical strategy for patients with CoA and VSD in the current era.

DISCUSSION

DR MARSHALL JACOBS (Philadelphia, PA) I rise to congratulate Dr St. Louis and colleagues on a very important investigation and a very clear and informative presentation. You selected an important unresolved clinical question. You relied upon a data source that has the distinct advantage of having collected multiinstitutional data over a period of 25 years.

The management of neonates and infants with coarctation and ventricular septal defect (VSD) certainly remains a matter of controversy. That having been said, it is almost universally acknowledged that a “one size fits all” approach is not likely to be the solution. Personally, I find it very interesting that there is a large body of evidence to support single-stage management of coarctation or arch obstruction in combination with complex anomalies like transposition with VSD or Taussig-Bing variety of double-outlet right ventricle, but with coarctation and isolated VSD, the matter seems somehow to be less clear.

Your analysis of the Pediatric Cardiac Care Consortium (PCCC) data reveals an increase in reliance on a single-stage approach from only 1% in the decade of the 1980s to 26% after the year 2000. Perhaps more importantly, it reveals a significant decline in discharge mortality associated with the one-stage approach to a level that is comparable and arguably lower than that of either the coarctation repair and pulmonary artery (PA) banding approach or the strategy based on coarctation repair with expectant management of the VSD. So based on your data, you are certainly justified in reaching a conclusion that primary VSD closure at the time of coarctation repair does not appear to result in a higher rate of discharge mortality when compared to staged repair. The next logical question then would be, which approach is best suited to which groups of patients?

In that regard, there were a few aspects of your data that I found particularly noteworthy and interesting. One, for example, was the observation that mean age at repair for patients having a single-stage approach was 87 days, whereas the mean age for the group having coarctation repair with PA band was 22 days. I guess I would have expected that the combined single-stage approach would have been more common in neonates with ductal dependency of the systemic circulation, but it appears that the opposite was the case, and I find this puzzling.

In a very nice report by Hal Walters in 2008, babies managed with a one-stage strategy had a median age at discharge of 39 days. Incidentally, they had a 90% freedom from cardiac reintervention of any type at 5 years. But the age at operation for your one-stage repair patients was considerably older; even older than the age at discharge after one-stage repair in some other series.

Also noteworthy was the observation that of 472 patients who underwent coarctation repair with PA banding, 220 returned for eventual VSD closure and 59 for PA band removal without VSD closure. Curiously, the hospital mortality for the second procedure in that latter group was 12%, that is, for PA band removal without VSD closure, and I think that was the highest operative mortality at any stage for any subcohort. But as importantly, if you do the math, the fate of nearly 200 patients with PA bands remains unknown.

During the years when they used to let me deal with patients and not just with data, my own thoughts on the question of the approach for coarctation with VSD went like this: neonates with critical coarctation and a large physiologically nonrestrictive VSD would most often be managed with a single operation through a median sternotomy. Very occasionally, I would resort to a strategy described by Dr Kirk Kanter, namely, coarctation repair through a thoracotomy followed immediately by VSD closure through the front. Babies with severe coarctation but with a physiologically restrictive VSD would undergo coarctation repair, and

the VSD would be managed expectantly. In the group with restrictive VSD, there would be the very rare exception where a severe degree of aortic arch hypoplasia warranted an anterior approach. Under those circumstances, the VSD would be closed concomitantly. With these management schemes, banding of the pulmonary artery was reserved for exceptional circumstances, such as multiple VSDs.

So, there is a question at the end of this discussion. In addition to commenting on some of the notable, curious aspects of the data set, would you tell us if you think that this very nice analysis supports the development of a treatment algorithm that would guide decision making for the management of patients with coarctation and VSD, and if it is does, what is the contemporary role for pulmonary artery banding?

DR ST. LOUIS Thank you, Marshall. Yes, I think it lends support to the idea that an algorithm should be developed. The problem with the data we obtained from the PCCC is that although very rich in certain types of data, it is very poor in others, and trying to assess the preoperative status of these patients, whether they were on prostins, whether they were on dopamine, sort of how they were, is very difficult to do. So it's very hard to come up with any kind of algorithm based on our data, which are plentiful, to understand what the preoperative conditions were and what led to the decision-making process. Some of the material in the PCCC, too, has become more dated and may not apply as well to today's therapy, although I think that the importance of this study was that it tells us that the algorithm some people have proposed from individual institutions is doable and it's supported by the lower mortality rate.

DR JACOBS I think it's a very important study, especially from the historical perspective, and I appreciate your sharing the paper with me.

DR MARCO RICCI (Miami, FL) In the patient cohort of coarctation repair with pulmonary artery banding, do you think that the presence of even mild aortic arch hypoplasia might have played a role in the unfavorable outcome? Sometimes going back to analyze data from databases, it might be difficult to disentangle patients who have truly isolated coarctation of the aorta versus those who have coarctation with some degree of aortic arch hypoplasia, which if left behind could indeed contribute to the unfavorable outcomes.

DR ST. LOUIS I have no doubt; I agree with you. I went back to the database after getting some of this information and tried to weed out those patients with a diagnosis of arch hypoplasia, and as you see from this discussion slide, the numbers are very low. And I think that the reason for that is just that it was very underreported in the data set, and it wasn't coded for over the last 3 decades for arch hypoplasia. But I agree with you.

DR ROSS M. UNGERLEIDER (Winston-Salem, NC) As many of you know, we published a similar review from the STS database several months ago; some of the people in this room are coauthors. And we found the same thing, that having a coarctation with a VSD doubled the mortality, and then if the patient had a VSD as part in association with other complex congenital heart lesions, the mortality was redoubled, and I am curious about that.

First of all, you have lumped all your patients into two buckets, coarct or coarct VSD, but I am wondering if there is a third bucket, which is coarct VSD with other significant defects, because your mortality for your coarct VSD group was actually quite high, and I am wondering if some of that could be explained by other complex congenital heart defects in that group?

DR ST. LOUIS I noticed that, too, that the mortality of 8% for coarct VSDs seems high. We went back and made sure that these were by associated diagnosis codes that were present in the database, and of those 2,000 patients, they were all VSDs. We took out the canals, we took out the truncuses, we took out everything that could confound the data. So truly I believe that that number of 2,000 is VSD diagnosis. We confirmed that in the proper way by going back to the operative notes and saying that there was no other associated anomaly. I think the biggest weakness with this, and we will bring this out in the paper, is the underreporting of the arch hypoplasia, and that is the biggest issue here.

DR UNGERLEIDER And that was the other question, and I think it was just asked, but we also noticed it in our review. We had no specific criteria for coding aortic arch hypoplasia. Some have suggested that a coarctation extending proximal to the left common carotid artery would fulfill the criteria for hypoplastic aortic arch, and that would at least provide an anatomic descriptor for patients to meet this diagnosis. Hopefully databases for the future, including the STS, will give us an opportunity to code patients with that degree of anatomic specificity so we can see if the kind of coarctation and the approach (such as sternotomy versus thoracotomy) impacts eventually on outcome. But very nice, and it's also nice to see how it does certainly agree with the other database review that having a VSD with a coarctation increases mortality. Thank you.

DR JEFFREY JACOBS (St. Petersburg, FL) Congratulations on a great presentation, and I think you have been able to demonstrate the value of the data that are in the PCCC database. It's a database that has multiinstitutional data over a very long period, as was stated by my cousin Marshal. The database allows for an analysis of process and outcomes over varying eras of time because it has data over such a long period of time, and I think there is tremendous value in those data.

Now, the PCCC no longer collects data, and I was just wondering if you could comment briefly on what's going to happen to the data that are in the PCCC and if those data will be available for future analyses such as this one. This is a topic you and I have discussed a lot but I thought we could talk about it briefly.

DR ST. LOUIS That's a great question, Jeff. The PCCC currently exists in an all-digitized format in the University of Minnesota's academic health care system and the financial support for that. We decided several years ago with your assistance that the utility of this database had long been gone and that with the current databases out there, there was no reason to continue to push enrolling new patients in it, we believe. But I think that's a great question. We're still trying to understand it. We utilize it. It's easy for us to access.

We have to give all the credit, mostly to Dr Jim Moller, who developed this. He developed the coding system, he developed the access system, he developed the risk stratification

system with it, and I think he was probably the father of a lot of the stuff that we have done and you have done in the STS. It is just sitting there. It is utilized by a number of our fellows and residents at the University of Minnesota, but the idea of where it goes next is still very much in the air, and I think that remains to be defined.

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Table 1**Participating Institutions (51 Centers)**

All Children's Hospital, St. Petersburg, FL
 Arizona Health Sciences Center, Tucson, AZ
 Arkansas Children's Hospital, Little Rock, AR
 Arnold Palmer Hospital for Children, Orlando, FL
 British Columbia's Children's Hospital, Vancouver, BC, Canada
 Children's Hospital Central California, Madera, CA
 Children's Hospital of Eastern Ontario, Ottawa, ON, Canada
 Children's Medical Center, Dallas, TX
 Children's Mercy Hospital and Clinics, Kansas City, MO
 Children's National Medical Center, Washington, DC
 Children's Hospital and Medical Center, Omaha, NE
 Children's Hospitals and Clinics of Minnesota, Minneapolis, MN
 Children's Hospitals and Clinics of Minnesota, St. Paul, MN
 Cleveland Clinic, Cleveland, OH
 Cook Children's Medical Center, Fort Worth, TX
 Driscoll Children's Hospital, Corpus Christi, TX
 Hospital Nacional de Niños, San José, Costa Rica
 Inova Children's Hospital, Falls Church, VA
 IWK Health Centre, Halifax, NS, Canada
 Kaiser Permanente, Los Angeles, CA
 Kosair Children's Hospital, Louisville, KY
 Le Bonheur Children's Hospital, Memphis, TN
 Legacy Emanuel Medical Center, Portland, OR
 Mary Bridge Children's Hospital, Tacoma, WA
 Mayo Clinic, Rochester, MN
 Medical College of Georgia, Augusta, GA
 Medical University of South Carolina, Charleston, SC
 Mercy Hospital Medical Center, Des Moines, IA
 Meritcare Medical Group, Fargo, ND
 Miami Children's Hospital, Miami, FL
 Nationwide Children's Hospital, Columbus, OH
 Ochsner Medical Center, New Orleans, LA
 OSF St. Francis Medical Center, Peoria, IL
 Rainbow Babies and Children's Hospital, Cleveland, OH
 Royal Hospital for Sick Children, Edinburgh, Scotland
 Sanford Children's Hospital, Sioux Falls, SD
 SSM Cardinal Glennon Children's Medical Center, St. Louis, MO
 St. Francis Health System, Tulsa, OK
 St. Joseph's Children's Hospital, Tampa, FL
 St. Louis Children's Hospital, St. Louis, MO

St. Luke's Boise Medical Center, Boise, ID
University of Alberta Hospital, Edmonton, AB, Canada
University of Florida Medical Center, Gainesville, FL
University of Iowa Hospitals and Clinics, Iowa City, IA
University of Kentucky Medical Center, Lexington, KY
University of Miami, Jackson Memorial Hospital, Miami, FL
University of Minnesota Medical Center, Minneapolis, MN
University of Mississippi Medical Center, Jackson, MS
University of Wisconsin Hospital and Clinics, Madison, WI
West Virginia University, Morgantown, WV
Wolfson Children's Hospital, Jacksonville, FL

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Table 2

All Patients (n = 7,860) From 1982 to 2007

Variable	OR	95% CI	p Value
VSD diagnosis	1.44	1.11–1.88	0.0059
VSD repair	1.69	1.04–2.75	0.0343
Pulmonary artery band	1.70	1.26–2.29	0.0005
Operation weight	0.04	0.02–0.09	<0.0001
Operation year	0.53	0.44–0.65	<0.0001
Aortic arch diagnosis	1.84	1.26–2.67	0.0015

CI = confidence interval; OR = odds ratio; VSD = ventricular septal defect.

Age at Coarctation Repair by Group With Associated In-Hospital Mortality, Operative Weight, Incision Type, and Postoperative Length of Stay

Table 3

Age	n (%)	Mortality n (%)	Op Wt (kg)	1 Incision/2 Incisions	PLOS (days)
Group 1					
30 days	202 (71)	18 (9)	3.1	195/7	22.7
31–365 days	76 (27)	6 (8)	4.1	69/7	19.1
1–12 years	8 (3)	1 (13)	16.1	5/3	6.0
Total	286	25 (8.7)	3.7	269/17	21.3
Group 2					
30 days	381 (81)	33 (9)	3.1	380/1	26.0
31–365 days	90 (20)	10 (11)	3.7	89/1	32.9
1–12 years	1 (0.2)	0 (0)	2.6	1/0	8.0
Total	472	43 (9.1)	3.2	470/2	27.3
Group 3					
30 days	806 (64)	92 (11)	3.2	805/1	23.7
31–365 days	343 (27)	8 (2)	4.5	343/0	14.9
1–12 years	93 (7)	0 (0)	19.2	93/0	6.7
12–18 years	22 (2)	0 (0)	57.2	22/0	7.0
Total	1,264	100 (7.9)	5.7	1,263/1	19.8

Op Wt = operative weight; PLOS = postoperative length of stay.

Table 4

Association of In-Hospital Mortality With Ventricular Septal Defect Management by Era

Decade	Isolated CoA	CoA+VSD Repair (Group 1)	CoA+PAB (Group 2)	CoA+VSD Without Repair (Group 3)
1982–1989	918 (3.1)	3 (66.7)	123 (15.5)	153 (7.8)
1990–1999	2,349 (2.2)	69 (18.8) ^a	235 (6.8)	624 (8.7)
2000–2007	2,226 (1.5)	214 (4.7) ^a	114 (7.0) ^a	487 (7.0)
Total	5,493 (2.1)	286 (8.7)	472 (9.1)	1,264 (7.9)

^a
 $p < 0.05$.

Values are n (%).

CoA = coarctation of aorta; PAB = pulmonary artery band; VSD = ventricular septal defect.

Table 5

Size of Ventricular Septal Defect at Coarctation Repair With Associated Mortality

VSD Size	Totals	Group 1	Group 2	Group 3
Small (<4 mm)	454 (6.1)	26 (0.0)	13 (0.0)	415 (5.5)
Moderate (4–7 mm)	467 (6.7)	74 (5.4)	73 (5.5)	320 (7.5)
Large (>7 mm)	754 (9.6)	159 (8.2)	298 (7.4)	297 (12.5)
Unspecified	347 (9.8)	27 (18.5)	88 (14.8)	232 (6.9)
Total	2,002 (8.3)	286 (8.7)	472 (9.1)	1,264 (7.9)

Values are n (%).

VSD = ventricular septal defect.