Unifocalization of major aortopulmonary collateral arteries in pulmonary atresia with ventricular septal defect is essential to achieve excellent outcomes irrespective of native pulmonary artery morphology

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Objective: Pulmonary atresia with ventricular septal defect and major aortopulmonary collateral arteries is a complex lesion with a high rate of natural attrition. We evaluated the outcomes of our strategy of unifocalization in the management of these patients.

Methods: From 1989 to 2008, 216 patients entered a pathway aiming for complete repair by unifocalizing major aortopulmonary arteries to a right ventricle-pulmonary artery conduit with ventricular septal defect closure. Where ventricular septation was not possible, definitive repair was considered to include pulmonary artery reconstruction and a right ventricle-pulmonary artery conduit or systemic shunt. Native pulmonary artery morphology was classified into confluent intrapericardial (n = 139), confluent intrapulmonary (n = 51), and nonconfluent intrapulmonary (n = 26).

Results: A total of 203 patients (85%) had definitive repair at a median age of 2.0 years. There was no statistically significant difference in survival after complete repair among the 3 morphologic pulmonary artery groups ($P = .18$). A total of 132 patients (56%) had complete repair with ventricular septal defect closure, as a single procedure in 111 patients and a staged procedure in 21 patients. Focalization of major aortopulmonary collateral arteries with proven long-term patency with the right ventricle was associated with a survival benefit compared with 14 patients in whom unifocalization was not possible and who had only systemic shunts. Overall survival was 89% at 3 years after definitive repair. During follow-up, 190 patients required 196 catheter reinterventions and 60 surgical reinterventions.

Conclusion: By using a strategy of unifocalization, intrapericardial pulmonary artery reconstruction, and right ventricle-pulmonary artery conduit, excellent long-term survival can be achieved in this group of patients even in the absence of native intrapericardial pulmonary arteries. (*J Thorac Cardiovasc Surg* 2009;138:1269-75)

☞ Supplemental material is available online.

Pulmonary atresia with ventricular septal defect (VSD) and major aortopulmonary collateral arteries (MAPCAs) is a complex lesion that is estimated to occur in 10 of 100,000 liveborn infants and that has a high rate of natural attrition.^{1,2} Until recently, only a small proportion of infants with this condition would ultimately undergo complete repair, and the outcomes after surgical intervention were disappointing,³⁻⁵ with an estimated 20% chance of surviving to the age of 30 years.¹

In this group of patients, pulmonary arterial blood flow is provided by MAPCAs, which vary in number and origin,⁶⁻⁸ together with the heterogeneous presence of native central PAs. The management of MAPCAs and their incorporation into any definitive repair remains controversial. Without intervention, MAPCAs may stenose and occlude or alternatively induce pulmonary vascular disease as a result of overperfusion.

We believe a key determinant of long-term success is to maximize recruitment of lung segments, and definitive repair should include mobilization and incorporation of native MAPCAs into a reconstructed central and peripheral pulmonary arterial tree, closure of the VSD, and a right ventricle-pulmonary artery (RV-PA) conduit to establish continuity between the PA confluence and the RV. This may be achieved by single or multiple stages via sternotomy or thoracotomies to perform mobilization of MAPCAs, augmentation of hilar vessels, or systemic-to-pulmonary shunts. Such a strategy has been successfully used,⁹ yet is contrary to clinical practice undertaken by others.¹⁰

There remains little evidence that an aggressive surgical approach in this particular patient group confers any long-term survival benefits or functional advantage over

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Received for publication April 26, 2009; revisions received July 2, 2009; accepted for publication Aug 9, 2009; available ahead of print Oct 21, 2009.

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0022-5223/\$36.00

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doi:10.1016/j.jtcvs.2009.08.011

Abbreviations and Acronyms

CI	= confidence interval
MAPCAs	= major aortopulmonary collateral arteries
OR	= odds ratio
RV-PA	= right ventricle-pulmonary artery
VSD	= ventricular septal defect

shunted patients, and most published data are limited to small surgical series with follow-up approaching 10 years. The objectives of the current study were to evaluate the outcomes of our policy that emphasizes MAPCA unifocalization together with intrahilar and intrapericardial PA reconstruction.

MATERIALS AND METHODS

The National Research Ethics Service, United Kingdom, considered this study to be a service evaluation and therefore did not require review by the National Health Service Research Ethics Committee. The data of all patients with pulmonary atresia, VSD, and MAPCAs who underwent surgical intervention at Birmingham Children's Hospital, Birmingham, United Kingdom, between January 1, 1989, and July 31, 2008, were reviewed.

Patients with other intracardiac pathologies accompanied by MAPCAs were excluded from the study. A total of 237 patients entered a treatment pathway aiming for complete repair by unifocalizing major aortopulmonary arteries to an RV PA conduit with VSD closure. When ventricular septation was not possible, definitive repair was considered to include PA reconstruction and a limiting RV PA conduit or systemic pulmonary shunt. Of these 237 patients, complete follow up data were available for 216 (120 female), and this cohort was used for analysis.

Assessment

Echocardiographic assessment was supplemented by cardiac catheterization to delineate the pulmonary blood supply and the characteristics of the MAPCAs. Patients were classified into 3 morphologic subgroups according to the presence or absence of confluent native PAs within the pericardium and at the hilum of the lung.¹¹ This spectrum extends in increasing order of complexity from confluent intrapericardial vessels, through absent intrapericardial but confluent intrapulmonary vessels, to absent intrapericardial and nonconfluent intrapulmonary vessels. The relative importance of each MAPCA was considered with a view to incorporating them into a definitive repair to maximize recruitment of at least 15 individual lung segments. The number of MAPCAs was assessed, and the patients were grouped categorically into less than 5 or more than 5 for the purposes of analysis.

Surgical Approach

The technical aspects of surgical intervention facilitated by hypothermic cardiopulmonary bypass with intermittent circulatory arrest have been described^{11,12} and share several common features with techniques reported by other centers.^{9,13-17}

Follow-up

Dates of definitive surgery were chosen as a common time point from which to derive follow up for all patients. Reintervention was defined as the time from definitive surgery to the first surgical or catheter based intervention because we considered that after such interventions the natural

histories of individual patients were considered to be sufficiently disparate to preclude further useful analysis on a cohort basis.

Statistics

Data are presented as median with interquartile range or mean $\pm$ standard deviation. Survival analysis was performed by the Kaplan Meier method, and data were compared using log rank and Wilcoxon tests. Multifactor analysis of risk factors for survival or death was performed by Cox proportional hazards regression. Factors included in the analysis were age at definitive surgery, gender, DiGeorge syndrome, native PA morphology, the number of MAPCAs, prior surgery at other centers, prior recruitment procedures, or creation of a central shunt or a peripheral shunt. Analyses were performed with SPSS 17.0 (SPSS UK Ltd, Woking, UK).

RESULTS**Patient Characteristics**

A total of 216 patients (120 female) were identified. Native PA morphology was classified into confluent intrapericardial ($n = 139$), confluent intrapulmonary ($n = 51$), and nonconfluent intrapulmonary ($n = 26$). A total of 123 patients had less than 5 MAPCAs, and 24 patients had more than 5 MAPCAs (69 patients missing information). Twenty-five patients (11%) had clinically overt DiGeorge syndrome; genetic testing for 22q11 deletion was performed selectively after informed parental consent was obtained. Before definitive surgery, 50 patients underwent unilateral ($n = 14$) or bilateral ($n = 36$) MAPCA recruitment procedures. Sixty-one patients had expanded polytetrafluoroethylene modified Blalock-Taussig or end-to-side central "Mee" systemic-pulmonary shunts. Sixteen patients had undergone procedures at other institutions before referral.

Surgical Algorithm

Median age at definitive surgery was 2.0 years (interquartile range, 0.8–5.5 years). Median follow-up time was 2.3 years (interquartile range, 0.1–6.7 years). There were 13 early (6%) and 13 late (6%) deaths. Establishment of RV-PA continuity with a valved conduit and VSD closure was possible in 158 patients (73%); single-stage basis in 104 patients (48%) and multiple-stage basis in 54 patients (25%). At last follow-up, the VSD remained open in 21 patients with an RV-PA conduit. For 37 patients, the definitive procedure was PA reconstruction with systemic-pulmonary shunting without RV-PA conduit or VSD closure (Figure 1).

Follow-up

Overall survival was 89% at 3 years. Figure 2 shows the changing distribution of patients by outcome category after definitive surgery. Multifactor analysis of risk factors for survival or death performed by stepwise Cox proportional hazards regression selected only RV-PA conduit as statistically significant ($P < .05$). Single-factor analyses for survival are shown in Table 1.

FIGURE 1. Flow chart summarizing the surgical treatment of 216 patients. MAPCA, Major aortopulmonary collateral arteries; PA, pulmonary artery; RV, right ventricle; VSD, ventricular septal defect.

Impact of Native Pulmonary Artery Morphology

By taking the cohort as a whole, no significant differences in the likelihood of survival were identified in patients after definitive surgery irrespective of native PA morphology (Figure 3). By comparing nonconfluent intrapulmonary and confluent intrapericardial native PAs, the 2 morphologic extremes of the spectrum, there was a nonsignificant tendency to increased risk of death in the former group (odds ratio [OR], 2.46; 95% confidence interval [CI], 0.94–6.47; $P = .07$). By looking closer at the subset of patients who had complete repair including RV-PA conduit and VSD closure, there were significant differences in survival in regard to native PA morphology ($P = .01$).

Achievement of Complete Repair

As is evident from the results of survival factor analysis in Table 1, when considering factors in isolation there is little to choose between the presence of an RV-PA conduit or VSD closure as factors associated with survival, but clearly they are strongly interrelated variables. The presence of an RV-PA conduit was associated with improved survival compared with systemic-pulmonary shunting alone (OR, 0.41; 95% CI, 0.17–0.97; $P = .04$). In all patients, VSD closure was protective against death (OR, 0.44; 95% CI, 0.20–0.98; $P = .04$) (Figure 4).

Of the 3 potential combinations of VSD and RV-PA conduit status, the results show that achieving a complete repair with a septated heart and an RV-PA conduit confers a survival

advantage compared with patients with a shunted circulation with neither VSD closure nor RV-PA conduit (OR, 0.38; 95% CI, 0.16–0.92; $P = .03$) (Figure 5). There were no significant differences in survival between those patients who had their VSD closed on a single- or multiple-stage basis (OR, 0.57; 95% CI, 0.26–1.23; $P = .15$) (Figure 6).

Reintervention

During the follow-up period, 190 patients underwent 196 therapeutic catheter and 60 surgical reinterventions

FIGURE 2. Scatter plot showing distribution of patients by outcome category after definitive surgery.

TABLE 1. Single-factor survival analysis for death, years to death, or end of follow-up

Factor		OR	95% CI	P value
Age at surgery	y	0.99	0.91 1.09	.92
Gender	Male vs female	1.09	0.50 2.35	.83
DiGeorge	Yes vs no	0.12	0.03 0.48	.05
Previous intervention at other center	Yes vs no	1.49	1.35 6.30	.61
Previous shunt	Yes vs no	1.02	0.43 2.43	.96
Any recruitment	Yes vs no	1.69	0.68 4.2	.26
RV PA conduit	Yes vs no	0.41	0.17 0.97	.04
VSD/RV PA conduit status	Open/conduit vs open	0.64	0.17 2.5	.52
	Closed/conduit vs open	0.38	0.16 0.92	.03
1 stage	1 stage vs multistage	0.57	0.26 1.23	.15
VSD closed	Closed vs open	0.44	0.20 0.98	.04
VSD by stage	Closed/multistage vs open	0.56	0.21 1.48	.24
	Closed/1 stage vs open	0.38	0.15 0.93	.03
Pulmonary atresia morphologic group	Confluent	1.53	0.61 3.83	.37
	intrapulmonary vs confluent intrapericardial			
	Nonconfluent pulmonary vs confluent intrapericardial	2.46	0.94 6.47	.07

OR, Odds ratio; CI, confidence interval; VSD, ventricular septal defect; RV-PA, right ventricle-pulmonary artery. Stepwise analysis selected only RV-PA conduit for multifactor analysis; therefore, these data are not included because the result is already in the single-factor column.

(Tables E1, E2, and E3). Ninety-one patients had at least 1 catheter, and 49 patients had further surgery, with 9 patients having more than 1 surgical reintervention. Single-factor analysis showed that definitive surgery at a younger age (OR, 0.95; 95% CI, 0.90 1.00; $P = .05$) and nonconfluent intrapulmonary arteries (OR, 1.68; 95% CI, 1.03 2.73; $P = .04$) were associated with increased reintervention. Only age at surgery was selected for multifactor analysis with other factors not adding extra association at the P less than .05 level.

FIGURE 3. Kaplan-Meier estimated survival of all patients after definitive surgery according to native PA morphology.

DISCUSSION

This work adds to the increasing body of evidence that children with pulmonary atresia VSD MAPCAs can achieve excellent outcomes by using an approach that emphasizes maximal recruitment of pulmonary vasculature and establishment of RV-PA continuity. These benefits are also applicable to those lacking intrapericardial native PAs, a subgroup who may have previously been declined surgery.

As popularized by Mee, the principle of recruiting pulmonary parenchyma by unifocalizing MAPCAs to a hilar manifold has been developed. Contemporary series using this approach in pulmonary atresia VSD have reported favorable outcomes.¹¹ There remains, however, limited long-term follow-up data on cohorts of patients treated in this way.

Assessment of Native Pulmonary Artery Anatomy

Extensive central/intrapericardial PA reconstruction is a key feature of our approach, and we have shown that native PA morphology is not a significant determinant of death after definitive repair.

Other groups¹⁸ have used surrogates for adequacy of pulmonary blood supply, including the McGoon ratio and Nakata index, but we have not found this to be useful.¹¹ We prefer to use a practical classification system adapted from one originally proposed by Tchervenkov and Roy.¹⁹ In our own clinical experience, we identified and classified patients into 1 of 3 groups according to intrapericardial and hilar vessels.¹¹ We rely on subjective assessment of angiographic images and aim to recruit 1.5 lungs or 15 bronchopulmonary segments using a combination of native hilar vessels and incorporating MAPCAs where appropriate.

Initial management of the right ventricular outflow tract has evolved over time to favor reconstruction over the use of systemic-to-pulmonary shunts that others have shown to be associated with increased mortality independently of other factors.^{14,20} The reasons for this are unclear but may

FIGURE 4. Kaplan-Meier estimated survival of all patients after definitive surgery according to VSD status. VSD, Ventricular septal defect.

FIGURE 5. Kaplan-Meier estimated survival of all patients after definitive surgery according to VSD and RV-PA conduit status.

include suboptimal vascular recruitment of pulmonary parenchyma.

Achievement of “Complete” Definitive Repair

Other groups consider the natural history of MAPCAs to be sufficiently unfavorable,¹⁰ such that they avoid incorporating them into any definitive correction. However, this approach risks suboptimal segmental perfusion and depends on the optimistic hope that the native intrapulmonary vasculature will collateralize sufficiently. The assumption that MAPCAs occlude or lack growth potential is based on a review of historical angiographic images that may be flawed.¹⁰

Furthermore, advocates of an early central shunt before a “definitive” repair without incorporating MAPCAs also risk excluding a significant number of patients from what we would consider prognostically beneficial surgery. By definition, a central shunt is only possible in the presence of intrapericardial PAs, thereby excluding patients without the prerequisite luminal patency and continuity.

We believe a key determinant of long-term success is to maximize recruitment of lung segments at an early age, incorporating MAPCAs where necessary, and to establish RV-PA continuity. The current report supports this philosophy. By emphasizing intervention at an early age, problems such as stenosis and occlusion that accompany chronic exposure of MAPCAs to systemic arterial pressure can be avoided, irrespective of the vessels’ embryologic origin.^{21,22}

Ventricular Septal Defect Closure Confers a Survival Advantage Irrespective of Single or Multiple Stages

The establishment of the RV-PA continuity with a valved conduit is protective, as is VSD closure, be it on a single- or multiple-stage basis. VSD closure was undertaken by single stage in 104 patients (48%) and multiple stages in 54 patients (25%), and was protective (OR, 0.85; 95% CI, 0.56–1.29; $P = .56$) compared with leaving the VSD open. There was no significant survival advantage of single-stage VSD closure over multiple-stage closure; thus, as long as the VSD is closed, it seems not to matter if it is performed in a single stage or multiple stages. This was clarified by using a compound of the potential combinations of uni-

FIGURE 6. Kaplan-Meier estimated survival of all patients after definitive surgery according to VSD and whether VSD closure was achieved by 1 stage or multistage repair.

focalizing a PA supply to the RV with or without VSD closure. The results demonstrate that achieving a septated heart with an RV-PA conduit had a survival advantage over a shunted circulation.

In addition, delayed VSD closure at a later stage was not associated with an increased risk of death compared with primary 1-stage repair. This lack of a significant difference in survival between groups in whom VSDs were closed initially or at a later stage is reassuring; if this step is omitted because of initial operative considerations, there does not seem to be a penalty associated with later closure.

Limitations

This is a retrospective surgical series. No accurate account could be made for patients who were reviewed but then did not subsequently undergo surgery. Unfortunately, we do not have access to exercise testing facilities to obtain an objective record of patients’ functional status.

CONCLUSIONS

By using a strategy of unifocalization, intrapericardial PA reconstruction, and RV-PA conduit, excellent long-term survival can be achieved in patients with pulmonary atresia VSD MAPCAs even in the absence of native intrapericardial PAs. MAPCAs are there to be recruited, not ignored. There remains an ongoing requirement for reintervention.

The authors thank their colleagues in the Departments of Pediatric Cardiology (Drs Chikermane, Desai, Dhillon, de Giovanni, Miller, Stümper, and Wright) and Pediatric Intensive Care, Birmingham Children’s Hospital for the ongoing care of these patients.

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Discussion

Dr Yves d'Udekem (Melbourne, Australia). We reviewed all the results with Christian Brizard, and all the comments and questions were discussed. What is your median time of follow up?

Dr Mussa. Three years.

Dr d'Udekem. It was actually 2.3 years in your article. And the reason for that is probably because of one of the flaws of your study. You stopped the follow up at the first intervention, which is a problem, because if you have an early intervention you don't know about the outcome of these patients.

In the follow up study of the Melbourne patients that we did with similar approaches, similar technique, even similar surgeons at times, we gathered a median time of follow up of 15 years. Approximately 15% of the patients who had a definitive repair died after the repair. The median time to death was 4 years, which means half of the patients died 4 years after the repair. If your follow up is approximately 2.5 years, then you may miss half, or who knows, maybe three quarters of the deaths after repair. And having a study for which your conclusion is based solely on survival, it's a very annoying fact.

Disputes are often due to miscommunication. I think if we want to progress in that debate of MAPCAs and unifocalization, we have to clarify some terminology and in particular the term "unifocalization." When you go into the lung's hilum, you dissect a confluent intrapulmonary artery, and you put a shunt there or you put a tube that you connect to the RV, I bet that you're going to call that unifocalization. I think I would call it rehabilitation of the native PAs. I think that we all agree that you have to recruit these intrapulmonary native PAs. But the real question is, what happened to the collateral arteries that you detach from the aorta and incorporate in that pulmonary arborization?

Do you have an idea of what proportion of patients underwent translocation of collateral arteries; and if you know that, do you have angiographic control studies of these translocated collateral arteries?

Dr Mussa. Well, you remember from our presentation that we had reintervened on approximately all of our patients. But in terms of reintervention on the MAPCAs, there weren't many. Like I said, only 1 stent and 2 balloons. There were some surgical interventions of the proximal portions of those, but I don't know the exact numbers of those, but we intervened on approximately 30 main PAs.

Dr d'Udekem. I'm afraid it's difficult to reintervene on a collateral if it's not existent anymore; that's my concern and why I think we need to have these angiographic data.

In your conclusion, I quote, you said, "MAPCAs are there to be recruited." We thought that was a very firm statement expressed like that. We have looked at your results, and we thought there may be some problems with that statement.

First, as I mentioned, the conclusion is based on survival, and we're not sure exactly yet about the survival. It may be excellent, but I think that remains to be demonstrated.

Second, you may want to use more equipoise in the interpretation of your results. For example, if you look at the characteristics of your patients, by the end of the study you have 50 of 216 patients left with a palliation. I think that's an important proportion. You may as well conclude in your study that you achieve a good survival in these patients if you leave a lot of them palliated. Or if you have only collateral translocations in, I don't know, 5060 patients, which

I guess it is, you can say, okay, you have better survival if you rehabilitated native PAs. Obviously these are just suggestions.

I'm wondering if these patients are not sicker than they appear to be, and, especially about what you say, they have a high rate of intervention, 80% reintervention at 5 years. So I'm sure you don't do unnecessary reintervention. So one can suspect that a lot of these patients may have elevated right ventricular to left ventricular pressure ratio, and I think that question should be solved.

There were angiographies for every single one of those undergoing these 196 reinterventions. It is difficult to show that translocated collateral arteries disappear. It might be much easier to demonstrate angiographically that these translocated collateral arteries remain patent. So if that is the case, it should be relatively easy to show angiograms with beautiful pulmonary arborization after translocation of the collateral arteries.

Do you have further data analysis or further evidence, particularly angiographic, to substantiate the statement that "MAPCAs are there to be recruited," or do you want to soften up a little bit that statement?

Dr Mussa. I'll start with the RV LV pressure ratio that you asked about first. Certainly that's an excellent question. It's data that we are looking at, and we have yet to come to a conclusion as to what our RV LV pressure ratios are doing over time.

In terms of angiographic follow up to examine the MAPCAs that we have recruited, unfortunately, a lot of our patients are located all over Europe, and it's difficult to get angiographic data on all of those patients to see exactly how those MAPCAs have fared over time. So I'm sorry, we don't have that data.

Dr Frank Hanley (*Stanford, Calif*). Congratulations on a phenomenal series of patients. Just to weigh in on this question of whether MAPCAs are valuable or not, we have an approximate

21% incidence of completely absent intrapericardial PAs, meaning not a string like, not even a cord like structure, but completely absent, including absence of confluent hilar areas. That 21% incidence translates into more than 100 patients with unifocalized vessels and no intrapericardial PAs. With 8 year mean follow up, and follow up up to 15 years, that subgroup is no different than the larger population. So it doesn't play out as a risk factor for either RV LV pressure ratio or survival.

The important message is, if patients can survive up to 15 years after unifocalization and repair when there were no true PAs, only collaterals, with the same outcome as those who have true PAs, it is unequivocal that collaterals are of value. This is the case when there are absent PAs. It follows that collaterals will be of benefit in any case where the PAs, if present, do not arborize normally.

Dr Mussa. Thank you.

Dr James Tweddell (*Milwaukee, Wis*). Among the patients who were unifocalized and in whom the VSD was left open, you used 2 strategies to supply pulmonary blood flow, an RV PA conduit or a Blalock Taussig shunt. How did you make that decision? And within that group, did you notice any difference in the likelihood of subsequent VSD closure between those 2 strategies?

Dr Mussa. First, I would just say that of those patients with the VSD left open and the use of the Blalock Taussig shunt, the use of shunts was largely historical in this series and was performed early on more than now. Can I just get your second question again, please?

Dr Tweddell. Among those 2 strategies of supplying pulmonary blood flow in the patients whose VSDs were open, did you identify a difference in the likelihood of subsequent VSD closure between those 2 strategies?

Dr Mussa. The simple answer is no, we didn't.

TABLE E1. Single-factor survival analysis for years to intervention, death, or end of follow-up

Factor		OR	95% CI	P value
Age at surgery	y	0.95	0.9–1.00	.05
Gender	Male vs female	1.01	0.71–1.45	.93
DiGeorge	Yes vs no	1.28	0.76–2.18	.35
Previous intervention at other center	Yes vs no	0.64	0.26–1.58	.33
Previous shunt	Yes vs no	1.16	0.78–1.74	.46
Previous recruitment	Yes vs no	1.02	0.63–1.66	.94
RV-PA conduit	Yes vs no	1.00	0.63–1.60	.99
VSD by conduit	Open/conduit vs open	1.27	0.60–2.71	.53
	Closed/conduit vs open	0.93	0.56–1.54	.77
One stage	1 vs multistage	0.90	0.63–1.29	.56
VSD closed	Closed vs open	0.85	0.56–1.29	.56
VSD by stage	Closed/multi vs open	1.12	0.69–1.82	.65
	Closed/1 stage vs open	0.73	0.47–1.15	.18
Group	Confluent	1.10	0.73–1.68	.63
	intrapulmonary vs confluent			
	intrapericardial			
	Nonconfluent	1.68	1.03–2.73	.04
	intrapulmonary vs confluent			
	intrapericardial			

OR, Odds ratio; CI, confidence interval; VSD, ventricular septal defect; RV-PA, right ventricle-pulmonary artery. Only age at surgery was selected for multifactor analysis in stepwise Cox proportional hazards regression; thus, these data are not shown. The other factors did not add extra association at the $P < .05$ level.

TABLE E3. Individual components of surgical reoperations (n = 60)

	n
RV-PA conduit change	35
PA augmentation	30
VSD closure	54
Further MAPCA recruitment	3
Pulmonary artery band	2
Resection of obstructive infundibular muscle bar	1
Repair of RV-PA conduit false aneurysm	1

RV-PA, Right ventricle-pulmonary artery; VSD, ventricular septal defect; MAPCA, major aortopulmonary collateral arteries.

TABLE E2. Catheter reinterventions (n = 196)

	n
Pulmonary artery balloon dilatation	145
Pulmonary artery stent	11
MAPCA balloon dilatation	2
MAPCA stent	1
MAPCA embolization	12
Balloon dilatation conduit	2
Pacemaker	5
Stenting of systemic-pulmonary shunt	1
Dilatation of fenestrated VSD patch	1

MAPCA, Major aortopulmonary collateral arteries; VSD, ventricular septal defect.