SURGERY FOR CONGENITAL HEART DISEASE

TRANSATRIAL-TRANSPULMONARY TETRALOGY OF FALLOT REPAIR IS EFFECTIVE IN THE PRESENCE OF ANOMALOUS CORONARY ARTERIES

Christian P. R. Brizard, MD^a
Carlos Mas, MD^a
Young-Sang Sohn, MD^b
Andrew D. Cochrane, FRACS^a
Tom R. Karl, MD^a

Objectives: The study's object was to analyze the outcomes of transatrial-transpulmonary repair in children with tetralogy of Fallot and anomalous coronary artery crossing the right ventricular outflow tract. **Methods:** The transatrial-transpulmonary approach was used in 611 consecutive repairs, 36 (5.9%) of which were associated with a surgically relevant coronary artery anomaly. The median age and weight of the patients at repair were 23 months (2.8-170 months) and 9.9 kg (5.2-41 kg), respectively. Anomalies included left anterior descending coronary artery from right coronary artery or single right coronary artery (n = 22), right coronary artery from left coronary artery or left anterior descending coronary artery (n = 8), and large right coronary artery conal branch (n = 6). Diagnosis was established before the operation in 25 of 36 cases by angiography (n = 24) or echocardiography (n = 1). The approach was successful in 34 cases, in 25 of which placement of a limited transannular patch was necessary. Two patients had a right ventricle-pulmonary artery conduit as a result of proximity of the coronary branch to the pulmonary arterial anulus and inability to adequately relieve the right ventricular outflow tract obstruction. **Results:** There have been no early or late deaths. Mean right ventricle-pulmonary artery gradient at last follow-up was 19 mm Hg (95% confidence interval 14.5-24 mm Hg), compared with 15 mm Hg (95% confidence interval 12.5-17.5 mm Hg) for patients with normal coronary arteries ($P = .3$). Actuarial freedom from reoperation at 120 months was 96.5% (95% confidence interval 79.8%-99.5%) and was also similar between patients with and without coronary artery abnormalities ($P = .92$). **Conclusions:** Surgically important coronary anomalies in tetralogy of Fallot can be dealt with through the transatrial-transpulmonary approach in most cases without major alterations in technique. Outcomes are similar to those of other patients with tetralogy of Fallot. The presence of anomalous coronary arteries does not impart incremental risk after this surgical strategy. (J Thorac Cardiovasc Surg 1998;116:770-9)

From the Victorian Paediatric Cardiac Surgical Unit, Royal Children's Hospital, Melbourne, Australia,^a and the Korea University Medical Center, Guro Hospital, Seoul, Korea.^b

Read at the Seventy-eighth Annual Meeting of The American Association for Thoracic Surgery, Boston, Mass, May 3-6, 1998.

Received for publication May 8, 1998; revisions requested June 19, 1998; revisions received July 10, 1998; accepted for publication July 16, 1998.

Address for reprints: C. P. Brizard, MD, Cardiac Surgical Unit, Royal Children's Hospital, Flemington Rd, Parkville, 3052, Victoria, Australia.

Copyright © 1998 by Mosby, Inc.

0022-5223/98 \$5.00 + 0 12/6/93476

Tetralogy of Fallot was among the first congenital cardiac abnormalities to be repaired.¹ The surgical strategy has been dominated worldwide by the technique initially described, the trans-right ventricular approach. In 1963 an alternate strategy for repair, combining transatrial and transpulmonary approaches, was described by Hudspeth and colleagues.² This technique has since been favored by some centers.³⁻⁷ The functional benefits include a lower incidence of arrhythmia and lower incidence and severity of early and late post-operative dysfunction of the right ventricle (RV).⁸⁻¹²

Coronary anomalies occur in between 5% and 12% of patients with tetralogy of Fallot.¹³⁻¹⁵ The most commonly encountered patterns are left anterior descending coronary artery (LAD) from right coronary artery (RCA), RCA from left coronary artery (LCA), RCA from LAD, large conal artery from RCA, and single RCA. These abnormalities of epicardial distribution are generally not associated with myocardial ischemia. Their main importance is related to surgical repair, at which time coronary injury could be inflicted during the ventriculotomy or the right ventricular outflow tract (RVOT) resection. One technical advantage of the transatrial-transpulmonary approach is the possibility of undertaking repair even when a major coronary artery branch crosses the infundibulum, precluding a conventional transventricular repair.^{5,6} We have been using the transatrial-transpulmonary approach since 1980 for all tetralogy repairs. Among the 611 patients so operated on in our unit, 36 had an anomalous course of a coronary artery that would have precluded conventional transventricular repair. The purpose of this article is to review our experience with the transatrial-transpulmonary approach in this subset of patients with tetralogy of Fallot. Specifically, we addressed the following questions:

1. What is the incidence of surgically important coronary artery anomalies among our patients with tetralogy of Fallot?
2. Could the anomalous coronary artery be diagnosed in an accurate and sensitive manner with the imaging techniques employed?
3. Was the transatrial-transpulmonary repair appropriate for these cases?
4. How do patients with anomalous coronary arteries compare with the rest of the tetralogy of Fallot cohort with respect to operative and late risk, functional and hemodynamic outcomes, and risk of reoperation?

Patients and methods

During the study period (1980-1997) 611 consecutive patients with tetralogy of Fallot underwent repair in our unit by way of the transatrial-transpulmonary approach. Included in this group were patients with double-outlet RV of tetralogy type and all patterns of pulmonary artery (PA) hypoplasia, stenosis, and discontinuity. Patients with pulmonary atresia and ventricular septal defect were not included in this series because our policy for their treatment has been placement of a RV-PA valved conduit, making any anomalous coronary artery a less critical issue. The hospital records and the angiographic and echocardiographic studies of the patients with confirmed anomalous coronary arteries were retrospectively studied. We excluded the patients who had abnormal coronary anatomic features but could have undergone operation through a classic transventricular approach without limita-

Fig 1. Surgical technique. Schematic drawing of the surgeon's view of the transatrial resection of the infundibular stenosis.

tions specifically imposed by the coronary anatomy (single left coronary ostium with a course of the RCA that was either posterior to the great arteries or between the 2 great arteries). The incidence of surgically relevant coronary anomalies was 5.9% (36/611, 95% confidence interval [CI] 1.1%-20.4%). There were 21 male patients (58%), and the median age and weight at operation were 23 months (2.8-170 months) and 9.9 kg (5.2-41 kg), respectively. Associated anomalies were present in 14 patients (39%).

Twenty patients had undergone 22 previous operations, including right modified Blalock-Taussig shunt (n = 11), left modified Blalock-Taussig shunt (n = 2), central shunt (n = 1), Waterston shunt (n = 3), and bilateral Blalock-Taussig shunts (n = 2). One patient had undergone a trans-right ventricular repair with a conduit insertion in another institution. She underwent a transatrial-transpulmonary RVOT resection at the time of reoperation for conduit obstruction, and her conduit was removed then.

The technique for repair was uniform for all the cases throughout the study, as previously described elsewhere.⁶ We employed bicaval cannulation and moderate hypothermia (28°-34°C) with cardioplegic arrest. The infundibular obstruction was resected by working through the right atrium and the PA anulus. The parietal and septal extensions of the infundibular septum were excised up to the PA anulus (Fig 1). All fibrous thickening of the endocardium and obstructive trabeculations in the RVOT were resected. The resection also included thinning of the anterior free wall of the infundibulum when it was hypertrophied. The pulmonary valve and anulus were incised whenever necessary to create an outlet of

Fig 2. Surgical technique. Modified infundibulotomy in the presence of LAD originating from the RCA. **A**, Anatomy. **B**, Opening of the anulus and the infundibulum, following the course of the anomalous artery. **C**, Transannular patch inserted.

adequate diameter. When the anomalous vessel was impeding the relief of the infundibular stenosis, the incision was made parallel to the vessel and toward the interventricular septum (in the case of an LAD from the RCA, Fig 2) or toward the right atrioventricular groove (in the case of an anomalous RCA from the LAD or the LCA) to maximize the opening of the RVOT. The pulmonary leaflets were retained whenever they were pliable. If the pulmonary leaflets were rigid and likely to impose a significant obstacle in the RVOT, they were sometimes completely resected and an autologous pericardial or homograft monocusp was included in the reconstruction of the RVOT. In most cases an outlet of a caliber 2 mm larger than the mean normal diameter—normalized for body surface area according to the study of Rowlatt, Rimoldi, and Lev,¹⁶ as modified by Kirklin and Barratt-Boyes¹⁷—could be

created. Even if this was not the case, no further attempt was made to enlarge the RVOT. We did not routinely measure either the pressure ratio between the RV and left ventricle or the RV-PA gradient after the repair, because we observed early in our experience that the RV pressure usually dropped significantly a few days after transatrial-transpulmonary repair. Valved homograft conduits were used when the anulus was diminutive and the anomalous coronary artery prevented sufficient enlargement. Even for patients with tetralogy of Fallot without anomalous coronary arteries, however, we commonly use a valved homograft repair for patients with extremely small pulmonary anuli who would require significant transannular extension, particularly in patients with diminutive PAs.

Follow-up data for the 36 patients were collected by our-

Fig 3. Echocardiograms. **A**, Subcostal view. Large vessel (white arrow) in front of the infundibulum (white asterisk). **B**, Short-axis parasternal view (same patient). RCA has early bifurcation into 2 major branches. One (white arrow) is directed to the left, toward the RVOT.

selves or our referring cardiologists. All preoperative echocardiograms and all angiocardiograms were reviewed for this study. We confirmed the positive diagnosis of anomalous coronary artery and tried to evaluate the minimal distance between the anulus and the anomalous vessel. The long-term follow-up, echocardiographically derived RV-PA gradient, and probability of reoperation were compared with those previously obtained for our entire cohort of patients with tetralogy of Fallot in 1992.⁶ The incidence of implantation of RV-PA conduits was compared with that of our entire tetralogy of Fallot series available in our database to November 1997.

Sample means and proportions are expressed with 95% CIs, with continuity correction for the upper and lower limits

Fig 4. Left anterior oblique angiogram. LAD (white arrow) arising from RCA.

of the latter. Alternately, data are presented as a median and range. The comparison of the 2 groups was conducted with the Mann-Whitney test for continuous variables and the Fisher exact test for dichotomous variables. Survival curves and reoperation risks were calculated with Kaplan-Meier formulas. Comparison of reoperation risks was done with the log rank test. No multivariable analyses were performed because of the scarcity of events. Pulmonary annular diameters were standardized against body surface area with z values. Statistical studies were done with the use of Prism version 2 (GraphPad Software, San Diego, Calif).

Results

Diagnosis. The diagnosis of anomalous coronary artery was made at echocardiography in 2 of 29 cases (Fig 3), at angiography in 24 of 35 cases (Figs 4, 5, and 6), and at operation in the remaining 10 cases. One patient underwent echocardiography only.

Anatomy. The anatomic variations were LAD from RCA ($n = 21$, 58%), single RCA ($n = 1$), RCA from LAD ($n = 2$, 5.5%), RCA from LCA ($n = 6$, 16.5%), and major infundibular branch from RCA ($n = 6$, 16.5%), including 1 patient with the RCA wedged between the 2 great arteries. The median distance between the vessel and the pulmonary anulus, as noted

Fig 5. Left anterior oblique angiogram. RCA (white arrow) arising from LCA.

at the operation and recorded in the operative notes for 18 patients, was 10 mm (7-13 mm).

Surgery. Thirty-four patients underwent repair without a conduit by the transatrial-transpulmonary technique, and 2 had a monocusp inserted. Twenty-five patients (69.4%, CI 51.6%-83%) had a limited transannular patch placed. Two patients (5.5%, CI 0.9%-19.9%) had a pulmonary homograft inserted in the RVOT. The mean z value for the pulmonary anuli was -1.65 (CI -2.23 to -1.07) for those who did not receive a conduit. The z values were -5.2 and -3.9 for the 2 who received a conduit. The higher probability of RVOT conduit implantation in this group of patients than in a group of patients with tetralogy of Fallot without anomalous coronary artery (11/587, 1.8%, CI 0.9%-3.4%) is probably due to chance ($P = .19$).

Follow-up. The thirty-six patients have been followed up for a total of 2312 patient-months, with a median follow-up of 52 months (1-175 months). Thirty-three patients have undergone echocardiography in the last 24 months. Two patients had their last echocardiograms 11 and 7 years after their operations, 8 and 5 years ago, respectively. Both are now adults. One overseas patient was no longer available for follow-up 20 months after his operation. He was well with no medication and had a low RVOT gradient on echocardiography at that time.

There were no in-hospital (CI 0%-12%) or late deaths. There was 1 late reoperation for RVOT obstruction; the same approach was used, with adequate relief of the gradient. The actuarial freedom from reoperation at 5 years was 96.9% (95% CI 79.8%-99.5%), with 16 patients remaining at risk (Fig 7). There was no difference in the

Fig 6. Right anterior oblique angiogram. Single RCA.

risk for reoperation between the groups of patients with and without anomalous coronary arteries ($P = .92$). At latest follow-up, all patients were in New York Heart Association functional class I, and 1 patient was receiving diuretics. The mean echocardiographically derived RVOT gradient was 19 mm Hg (CI 14.5-24 mm Hg) at latest follow-up, similar ($P = .3$) to that seen in patients with normal coronary arteries (15 mm Hg, CI 12.5-17.5 mm Hg). One patient was also operated on for tracheoesophageal fistula repair as a neonate. Severe gastroesophageal reflux led to repeated chest infections and elevated PA pressure, and she has a systemic pressure in the RV with no RVOT gradient. One patient has a nearly systemic pressure in the RV, with a 70-mm Hg RVOT gradient. His RV is not dilated, and reoperation is not contemplated at this time. The 2 patients with pulmonary homografts were operated on 40 and 17 months ago. Pressure gradients are 20 and 45 mm Hg, respectively.

The retrospective review of the 29 preoperative echocardiograms did not allow more positive diagnoses of anomalous coronary artery. The retrospective examination of all available preoperative angiograms (30/35) led to the same positive diagnosis made before the operation for all the patients who had an anomalous coronary artery found before the operation. Ten patients had false-negative diagnoses after preoperative angiography; in 2 of these cases the retrospective examination was able to demonstrate LAD from the RCA. Overall, the anomalous coronary artery could be found retrospectively in 22 of the 30 angiograms available. On 14 angiograms the pulmonary anulus and the

Fig 7. Kaplan-Meier freedom from reoperation curve. *Reop*, Reoperation.

Table I. Mean age (in months) at repair according to the anatomy and divided into 2 time spans

	Repair before 12/89			Repair after 1/90			P*
	N	Mean	95% CI	N	Mean	95% CI	
Normal coronary arteries	279	31.8	27-36	297	23.9	20.6-27	.0045
Abnormal coronary arteries	4	51.8	29-74	22	21	14-28	.0137
P	.083			.5			

*Unpaired *t* test with Welch's correction.

anomalous vessel were visible on the same frames (most of the time on the lateral angiogram of the RV). The minimum distance between the 2 elements was measured. Eight of the corresponding operative notes quoted the intraoperative estimation of this same distance. There was a significant correlation between these figures (Spearman $r = 0.92$, $P = .002$).

The mean ages at repair for the patients with normal and abnormal coronary artery anatomy are presented in Table I. Results are presented in 2 time spans, before and after 1990. With the same cutoff point in 1990, the probability of an initial palliative procedure was higher in the earlier group (12/14 patients vs 8/22 patients, $P = .008$), and the delay between the palliation and the repair was longer (42 vs 12 months, $P = .013$). Also, no bilateral Blalock-Taussig anastomoses were performed in the later group.

Discussion

This series of patients with anomalous coronary arteries demonstrates the same postoperative and long-term results after repair as seen in the remainder of our cohort with tetralogy of Fallot. We believe that the transatrial approach is the best strategy for this anatomic

condition. In most cases it allows adequate relief of the infundibular obstruction without the insertion of an RV-PA conduit and provides good long-term hemodynamic and functional results. The adequacy of this approach has been highlighted by other authors.⁵ Other series dealing with this specific anatomic condition cite in-hospital mortality rates between 8% and 29%,^{14,15,18} with an incidence of RV-PA conduit implantation as great as 40%.¹⁴ The reference role of these publications should not be overestimated, however, because they belong to another era of cardiac surgery.

Two points are important in our series. First, we use the transatrial-transpulmonary approach for all our tetralogy repairs and have done so consistently for longer than 18 years. This experience is beneficial when more demanding resections are to be performed. Second, because we do not routinely measure the RV-PA gradient or the pressure ratio between the RV and left ventricle, we are less likely to use a conduit in patients with high RV pressure in the immediate postoperative period. This attitude is based on our early experience, in which we found a significant fall in RV-PA gradient in the days after tetralogy repair. Other authors have had the same experience.^{5,19}

Many different surgical techniques to deal with anomalous coronary arteries have been described. The left internal thoracic artery to LAD bypass has been described either as an elective procedure or after accidental injury of the anomalous vessel.^{20,21} Also described is a turned down flap of anterior pulmonary wall sutured to the edge of the right ventriculotomy and subsequent patching of the anterior aspect of this pathway,²² which is a variation of the double-barrel technique. Boncheck²³ described the complete mobilization of the initial portion of the LAD with the ventriculotomy and the patch going under it. All these procedures have their drawbacks. We did not have to use any of these procedures, although they might have been suitable in the 2 cases in which we performed conduit repair. Because we believe that a large opening of the pulmonary anulus, in combination with its prolongation on the infundibulum, leads to free pulmonary regurgitation, we have elected to use a pulmonary homograft in these cases regardless of the coronary anatomy. However, the probability of conduit implantation is low, and not significantly different from that for patients with normal coronary anatomy. The probability of conduit implantation is far less than in the series of tetralogy repairs with the trans-right ventricular approach.

In our series of patients with tetralogy of Fallot, it has been accepted that an angiogram would be part of the protocol for preoperative investigations. Consequently no thorough attempt was made to make a positive diagnosis of the anomalous coronary artery during the echocardiogram. The tapes that have been reviewed for this study can only show what was registered at the time of the initial echocardiography, which is closely related to the way that the echocardiographer conducted this examination. In our series a preoperative true-positive echocardiographic diagnosis of anomalous coronary artery was rare, and the incidence of false-positive diagnoses was unknown but could have been high. Two studies in the literature demonstrated that the echocardiogram is a highly efficient tool in this field.^{24,25} During an echocardiographic study, the diagnosis of an anomalous coronary artery depends on the identification of the vessel in several views. This requires considerably more skill and time than for a routine tetralogy examination, and implies that a systematic approach to the diagnosis of coronary anatomy in patients with tetralogy of Fallot is required. This is probably why retrospective screening of the preoperative echocardiographic tapes of the patients included in this series did not result in more diagnoses of anomalous coronary arteries. Echocardiography, when performed as a study complementary to angiocardiology, has to be done

differently than when it is to be the sole preoperative investigation or when the indication and technical details of the catheter study depend on its findings.

Angiocardiology failed to detect the anomalous coronary artery in 10 cases. The views used in most cases were left anterior oblique and right anterior oblique with 20-degree skew, which are conventional for tetralogy of Fallot in our institution. Carvalho and associates,²⁶ in the retrospective study included in their article in 1993 that used the same classic views, found 9 false-negative diagnoses of anomalous coronary artery in 10 patients who had the anatomy demonstrated during their operations. Much higher sensitivity is achieved with angiograms when special views or injection sites are used.^{26,27} If the distance between the anomalous vessel and the pulmonary anulus could be predicted, this information would be extremely beneficial. We believe that we have been able to determine it retrospectively in some of our cases. This distance can also be estimated on an echocardiogram.^{24,25}

On the basis of the literature and the previously mentioned data, what should be the strategy for diagnosis of anomalous coronary artery? Is a preoperative diagnosis of such an abnormality a necessity? As we have seen in this series, preoperative diagnosis of anomalous coronary artery is not necessary to achieve a good relief of infundibular obstruction. In a small proportion of cases, however, in which the anomalous coronary artery crosses at the level of a very small pulmonary anulus, an accurate preoperative diagnosis is highly preferable. This would allow planning of special surgical techniques or palliation. Although in our unit patients with tetralogy of Fallot still routinely undergo catheterization, most of the data that are necessary for surgical indications could be acquired with the echocardiographic study. As surgeons we would like to have more specifically oriented angiograms performed in selected cases on the basis of the echocardiographic data. Numerous patients could be operated on without angiograms, providing that echocardiography had ruled out significant likelihood of an anomalous coronary artery in the presence of a diminutive pulmonary anulus. Ultimately the diagnosis of anomalous coronary artery will always remain an intraoperative issue, even bearing in mind the extremely rare intramyocardial LAD from the RCA.²⁸

Initially we thought that the repair should be delayed as long as possible in the presence of an anomalous coronary artery to allow a technically easier procedure. The rationale was that there would be some growth of the anulus in the interim, possibly with development of an intermediate chamber between the infundibular stenosis and the pulmonary anulus. If this is true, it has

been difficult to demonstrate, and any potential technical benefits have been counterbalanced by the hypertrophy of the RV. The real impact of this approach on the age at repair is difficult to determine. There was no statistical difference in the age at repair between the 2 anatomic groups. Furthermore, we have been using less palliation in our patients with tetralogy of Fallot and generally are operating earlier on patients who have and have not received palliation. Our current policy is to perform repair between 4 months and 1 year. Younger patients usually receive palliation unless they have exceptionally favorable anatomic characteristics.

Conclusions. On the basis of this experience, we can make the following inferences:

1. Surgically important coronary artery anomalies may be encountered in 6% of patients with tetralogy of Fallot.
2. Neither angiography nor echocardiography was 100% sensitive for preoperative diagnosis of the coronary abnormality, so the diagnosis and identification remain the surgeon's responsibility.
3. Most cases of anomalous coronary artery in tetralogy of Fallot can be dealt with through the transatrial-transpulmonary approach, limiting the transannular patch to the area between the pulmonary anulus and the anomalous vessel.
4. The outcome of this approach with respect to early and late mortality risk, need for reoperation, and functional and hemodynamic status is similar to that for patients without an anomalous coronary artery.

The patients in this series were operated on by several different surgeons. They were Roger Mee, Tom Karl, Bill Brawn, Andrew Cochrane, Christian Brizard, Ash Pawade, and Shunji Sano.

REFERENCES

1. Lillehei CW, Cohen M, Warden HE, Read RC, Aust JB, DeWall RA, et al. Direct vision intracardiac surgical correction of the tetralogy of Fallot, pentalogy of Fallot and pulmonary atresia defects: report of first ten cases. *Ann Surg* 1995;142:418-45.
2. Hudspeth AS, Cordell AR, Johnston FR. Transatrial approach to total correction of tetralogy of Fallot. *Circulation* 1963;27:796-800.
3. Pacifico AD, Sand ME, Barger LM Jr, Colvin EC. Transatrial-transpulmonary repair of tetralogy of Fallot. *J Thorac Cardiovasc Surg* 1987;93:919-24.
4. Binet JP. Correction of tetralogy of Fallot with combined transatrial and pulmonary approach: experience with 184 consecutive cases. *J Card Surg* 1988;3:97-102.
5. Dietl CA, Torres AR, Cazzaniga ME, Favaloro RG. Right atrial approach for surgical correction of tetralogy of Fallot. *Ann Thorac Surg* 1989;47:546-51.
6. Karl TR, Sano S, Pornviliwan S, Mee RB. Tetralogy of Fallot: favorable outcome of nonneonatal transatrial, transpulmonary repair. *Ann Thorac Surg* 1992;54:903-7.
7. Kawashima Y, Kitamura S, Nakano S, Yagihara T. Corrective surgery for tetralogy of Fallot without or with minimal right ventriculotomy and with repair of the pulmonary valve. *Circulation* 1981;64(suppl 2):II147-53.
8. Atallah-Yunes NH, Kavey RE, Bove EL, Smith FC, Kveselis DA, Byrum CJ, et al. Postoperative assessment of a modified surgical approach to repair of tetralogy of Fallot: long-term follow-up. *Circulation* 1996;94(9 suppl):II22-6.
9. Norgård G, Gatzoulis M, Moraes F, Lincoln C, Shore DF, Shinebourne EA, et al. Relationship between type of outflow tract repair and postoperative right ventricular diastolic physiology in tetralogy of Fallot. *Circulation* 1996;94:3276-80.
10. Stellin G, Milanese O, Rubino M, Michielon G, Bianco R, Moreolo GS, et al. Repair of tetralogy of Fallot in the first six months of life: transatrial versus transventricular approach. *Ann Thorac Surg* 1995;60(6 suppl):S588-91.
11. Dietl CA, Cazzaniga ME, Dubner SJ, Pérez-Baliño NA, Torres AR, Favaloro RG. Life-threatening arrhythmias and RV dysfunction after surgical repair of tetralogy of Fallot: comparison between transventricular and transatrial approaches. *Circulation* 1994;90(5 Pt 2):II7-12.
12. Miura T, Nakano S, Shimazaki Y, Kobayashi J, Hirose H, Sano T, et al. Evaluation of right ventricular function by regional wall motion analysis in patients after correction of tetralogy of Fallot: comparison of transventricular and nontransventricular repairs. *J Thorac Cardiovasc Surg* 1992;104:917-23.
13. Meng L, Eckner DA, Lev M. Coronary distribution in tetralogy of Fallot. *Arch Surg* 1965;90:363-6.
14. Humes RA, Driscoll DJ, Danielson GK, Puga FJ. Tetralogy of Fallot with anomalous origin of left anterior descending coronary artery: surgical options. *J Thorac Cardiovasc Surg* 1987;94:784-7.
15. Hurwitz RA, Smith W, King H, Girod DA, Calwell RL. Tetralogy of Fallot with abnormal coronary artery: 1967 to 1977. *J Thorac Cardiovasc Surg* 1980;80:129-34.
16. Rowlatt UF, Rimoldi HJ, Lev M. The quantitative anatomy of the normal child's heart. *Pediatr Clin North Am* 1963;10:499-588.
17. Kirklin JW, Barratt-Boyes BG. Cardiac surgery. New York: Churchill-Livingstone; 1993. p. 3-60.
18. Berry BE, McGoon DC. Total correction for tetralogy of Fallot with anomalous coronary artery. *Surgery* 1973;74:894-8.
19. Antunes MJ, Castela E, Sanches MF, Melo AS. Preservation of the pulmonary annulus in total correction of tetralogy of Fallot: decreasing transannular gradients in the early follow-up period. *Eur J Cardiothorac Surg* 1991;5:528-32.
20. Bhutani AK, Koppala MM, Abraham KA, Balakrishnan KR, Desai RN. Inadvertent transection of anomalously arising left anterior descending artery during tetralogy of Fallot repair: bypass grafting with left internal mammary artery [letter]. *J Thorac Cardiovasc Surg* 1994;108:589-90.
21. Cooley DA, Duncan JM, Gillette PC, McNamara DG. Reconstruction of coronary artery anomaly in an infant using the internal mammary artery: 10-year follow-up. *Pediatr Cardiol* 1987; 8:257-9.
22. van Son JA. Repair of tetralogy of Fallot with anomalous origin of left anterior descending coronary artery. *J Thorac Cardiovasc Surg* 1995;110:561-2.
23. Bonchek LI. A method of outflow tract reconstruction in tetralogy of Fallot with anomalous anterior descending coronary artery. *Ann Thorac Surg* 1976;21:451-3.
24. Berry JM Jr, Einzig S, Krabill KA, Bass JL. Evaluation of coro-

nary artery anatomy in patients with tetralogy of Fallot by two-dimensional echocardiography. *Circulation* 1988;78:149-56.

25. Jureidini SB, Appleton RS, Nouri S. Detection of coronary artery abnormalities in tetralogy of Fallot by two-dimensional echocardiography. *J Am Coll Cardiol* 1989;14:960-7.
26. Carvalho JS, Silva CM, Rigby ML, Shinebourne EA. Angiographic diagnosis of anomalous coronary artery in tetralogy of Fallot. *Br Heart J* 1993;70:75-8.
27. Dabizzi RP, Caprioli G, Aiazzi L, Castelli C, Baldrighi G, Parenzan L, et al. Distribution and anomalies of coronary arteries in tetralogy of Fallot. *Circulation* 1980;61:95-102.
28. White RI Jr, Frech RS, Castaneda A, Amplatz K. The nature and significance of anomalous coronary arteries in tetralogy of Fallot. *Am J Roentgenol Radium Ther Nucl Med* 1972;114:350-4.

Discussion

Dr John W. Brown (*Indianapolis, Ind*). Your superb results leave little to criticize, with no early and late deaths and a late postoperative gradient lower than 20 mm Hg. You used an RV-PA conduit in only 2 of 36 patients. When one compares these results with those reported in the literature, one is even more impressed; the literature gives an operative mortality rate for this lesion of between 8% and 29%, and conduits are used in as many as 40% of cases in many series. However, these literature reports are old.

Our approach in Indianapolis to this cohort of patients with tetralogy of Fallot has been somewhat different, but our results are comparable. We have operated on 540 patients with tetralogy of Fallot in the last 20 years. Of these, 16 patients have had this anomaly, which is about half the incidence that you report. All but 1 underwent repair through a transventricular approach. A single oblique ventriculotomy, parallel and below the anomalous coronary artery, was performed in 75% of our cases (12/16), and a second transannular ventriculotomy, superior to the anomalous coronary artery, was performed in the other 4 cases. Care is taken to leave at least 5 mm of myocardium above, below, and underneath the anomalous coronary. Polytetrafluoroethylene monocusps are routinely used in all cases of tetralogy Fallot in which a transannular incision is made.

We likewise have no early or late deaths, and only 1 of our patients received a conduit. The postoperative RV-PA gradient is less than 27 mm Hg in all cases. I am not sure that you can conclude that the transatrial-transpulmonary approach is the best technique for this anomaly; however, your results are at least comparable to ours. I do not think that the anomalous coronary artery precludes the use of a ventricular approach to this lesion. I have 3 questions.

First, 20 of your 36 patients had shunts before repair in this series and you now advocate repair between 4 and 12 months. Is there a place for shunting in this group of patients with anomalous coronary arteries?

Second, 75% of your patients (25/36) had transannular incisions, yet only 3 had a monocusp used. What is your current indication for insertion of a monocusp in patients with tetralogy of Fallot?

Third, the degree of tricuspid regurgitation is somewhat higher in our experience when a transatrial approach is used

for the closure of the ventricular septal defect. Have you seen any increased incidence of tricuspid regurgitation with this approach?

Dr Brizard. Thank you for your comments. The first question was about the shunt and whether there is any place for shunting in our current policy. We prefer a nonneonatal transatrial-transpulmonary repair, so we still have a place for shunting in our approach to tetralogy of Fallot. We would perform a shunt in patients who have very small PAs or other conditions that would preclude repair during the neonatal period. As I concluded in our comments, there is not a major role for shunts in this particular anatomic setting.

Regarding the monocusp and its indications, we try to retain all pulmonary valvular tissue as long as it is pliable. If the valvular tissue is not pliable, it is likely to provoke more gradient than competence, and in that case we would excise all leaflet tissue. That would be the only indication for a monocusp. If we use a monocusp, we prefer autologous pericardium, which may be treated with glutaraldehyde or not according to the surgeon's preferences. I prefer to treat with glutaraldehyde.

With respect to the tricuspid valve regurgitation, we have no comparison because we do not use the transventricular repair, but the tricuspid valve is systematically tested and repaired with 1 or 2 sutures for commissural plasty after the transatrial closure of the ventricular septal defect.

Dr Yasunaru Kawashima (*Osaka, Japan*). In your introduction you mentioned that this transatrial-transpulmonary repair was reported in 1963 by Hudspeth and colleagues, but to the best of my recollection they performed this procedure only through the right atrium.

Anyway, as a person who initiated this transatrial-transpulmonary tetralogy repair 20 years ago, I am delighted to know that this procedure can deal with surgically important coronary anomalies in tetralogy of Fallot.

However, I am surprised that you have such a large number of surgically relevant coronary artery anomalies. In our experience with more than 500 cases, we have seen such anomalies in only about 1% of our patients. There may be some racial differences, as we see in the location of the ventricular septal defect.

You mentioned that 3 patients had RV-PA conduits placed because of proximity of the coronary artery to the PA anulus. I wonder whether there is any possibility of avoiding this by extending the pulmonary arteriotomy down to the left lateral aspect, because there must be no important coronary artery in cases in which the LAD comes from the RCA.

Dr Brizard. I apologize because the text of the abstract in the abstract book is slightly different from the presentation. There were actually only 2 conduits in the series.

With respect to the incidence of anomalous coronary arteries, our figure of 5.9% is consistent with the rest of the literature, according to both angiocardiographic and anatomic studies.

In reply to your last remark, the slide in color (Fig 2, B) showing the technique of the modified infundibulotomy is exactly what you described.

Dr Christo Tchervenkov (*Montreal, Canada*). I suggest that some other techniques can be applied to avoid placement

of a conduit in these cases. We have had perhaps a few more than half a dozen patients, neonates and young infants, in whom the aberrant LAD was actually extremely close to the pulmonary valve anulus. For these severely hypoplastic pulmonary valve anuli we have been able to transect the main PA above the anulus, closing the proximal end, and translocate the main PA onto the ventriculotomy incision after mobilization of the branch PAs, finally completing the repair with a transannular patch. In some cases this technique allows you to avoid the use of a conduit.

Recently we treated a patient with double-outlet RV and aberrant LAD that coursed rather obliquely and lower, further away from the incision. I could not use this technique, so in that case I preserved and enlarged as much as possible the native pathway and then placed a conduit on top of that, effectively creating a double-outlet RV to treat a double-outlet RV.

Among cases in which you used the conduit, do you have any in which you preserved the native pathway? Do you have any information regarding what happens to that native pathway in the presence of an extracardiac conduit?

Dr Brizard. In the 2 patients who had conduits placed, the native pathway is patent and the flow is visible on echocardiography. It is not uncommon, and a few cases have been reported in the literature. I cannot answer regarding the future of this native pathway.

With respect to the alternate techniques, I think that the definitive answer for this surgical problem is a bypass from the left internal thoracic artery to the LAD. This has always been reported in cases of accidental transection of the LAD or the anomalous coronary artery. The big step would be to perform this electively in cases with an extraordinarily small anulus.

ON THE MOVE?

Send us your new address at least six weeks ahead

Don't miss a single issue of the journal! To ensure prompt service when you change your address, please photocopy and complete the form below.

Please send your change of address notification at least six weeks before your move to ensure continued service. We regret we cannot guarantee replacement of issues missed due to late notification.

JOURNAL TITLE:

Fill in the title of the journal here. _____

OLD ADDRESS:

Affix the address label from a recent issue of the journal here.

NEW ADDRESS:

Clearly print your new address here.

Name _____

Address _____

City/State/ZIP _____

COPY AND MAIL THIS FORM TO:

Periodical Subscription Services
Mosby, Inc.
11830 Westline Industrial Dr.
St. Louis, MO 63146-3318

OR FAX TO:

314-432-1158

M Mosby

OR PHONE:

1-800-453-4351
Outside the U.S., call
314-453-4351