Intention-to-Treat Analysis of Pulmonary Artery Banding in Conditions With a Morphological Right Ventricle in the Systemic Circulation With a View to Anatomic Biventricular Repair

David S. Winlaw, MBBS, MD, FRACS; Simon P. McGuirk, BMedSci, MRCS; Christian Balmer, MD; Stephen M. Langley, MD, FRCS; Massimo Griselli, MD, MS, FRCS; Oliver Stümper, MD, PhD, MRCP; Joseph V. De Giovanni, MD, FRCR, FRCPC; John G. Wright, MA, FRCR, FRCPC; Sara Thorne, MD, MRCP; David J. Barron, MD, MRCP, FRCS; William J. Brawn, FRACS, FRCS

Background—Some patients with a morphological right ventricle (mRV) in the systemic circulation require early intervention because of progressive systemic ventricular dysfunction or atrioventricular valve regurgitation. They may be eligible for anatomic repair (correction of atrioventricular and ventriculoarterial discordance) but require prior training of the morphological left ventricle (mLV).

Methods and Results—Forty-one patients with congenitally corrected transposition of the great arteries or a previous atrial switch procedure embarked on a protocol of pulmonary artery (PA) banding with a view to anatomic repair. All had an mRV in the systemic circulation and a subpulmonary mLV that was not conditioned by either volume or pressure load. Two patients were not banded, and 39 were followed up for a median of 4.3 years (range, 25 days to 12.6 years). Sixteen patients achieved anatomic repair, with 3 in the early stages of the training protocol. After 2 years, 12 patients were not suitable for anatomic repair and persisted with palliative banding; 8 were functionally improved; and 4 died, underwent transplantation, or required debanding. PA banding improved functional class but did not improve tricuspid regurgitation in the long term for patients not achieving anatomic repair. mLV function was a critical determinant of survival with a PA band as well as survival after anatomic repair. Patients >16 years were unlikely to achieve anatomic repair.

Conclusion—PA banding is a safe and effective method of training the mLV before anatomic repair. It is also an effective palliative procedure for those who do not attain this goal. (Circulation. 2005;111:405-411.)

Key Words: heart defects, congenital • transposition of great vessels • surgery

In the current era, the majority of patients with a morphological right ventricle (mRV) supporting the systemic circulation have congenitally corrected transposition (ccTGA), with a diminishing number having undergone an atrial switch procedure for dTGA. Some patients with a systemic mRV have a good quality of life and live to an advanced age. However, some patients develop progressive ventricular dysfunction and/or severe tricuspid, i.e., systemic atrioventricular (AV), valve regurgitation early in life and require surgical intervention.

We have studied patients with a systemic mRV and intact ventricular septum who present early in life with systemic ventricular dysfunction or AV valve regurgitation. Unlike those with a ventricular septal defect (VSD), the morphological left ventricle (mLV) is not volume loaded and has been working against the low-resistance pulmonary circulation. Typically, the mLV has a reduced ventricular volume and wall thickness and may be “squashed” by the volume-loaded mRV. This is associated with malposition of the interventricular septum and may contribute to the development of systemic AV valve regurgitation by distorting the subvalvar tension apparatus.

The surgical options for symptomatic patients with a systemic mRV and systemic ventricular dysfunction and/or AV valve regurgitation include systemic AV valve replacement or correction of discordant AV and ventriculoarterial connections. Anatomic correction (placement of the mLV in the systemic circulation with the use of an arterial procedure, with or without atrial switch) may be superior to the conventional repair that leaves the mRV in the systemic circuit.1,2 It is unlikely that the mLV could effectively support the systemic circulation before undergoing a period of prior training, achieved by application of a pulmonary artery (PA) band to increase mLV afterload.3 PA banding is reportedly
TABLE 1. Patient Characteristics

<table>
<thead>
<tr>
<th></th>
<th>Previous Atrial Switch</th>
<th>ccTGA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age at banding, y, median (range)</td>
<td>16.2 (10.0–24.2)</td>
<td>2.8 (1.8 mo-39.1)</td>
</tr>
<tr>
<td>Weight at banding, kg, median (range)</td>
<td>54.5 (28.2–75)</td>
<td>15.4 (5.2–89.5)</td>
</tr>
<tr>
<td>Preband NYHA status, median (range)</td>
<td>II (II–III)</td>
<td>II (II–IV)</td>
</tr>
<tr>
<td>Preband moderate or severe mRV dysfunction, n (%)</td>
<td>16 (80)</td>
<td>5 (24)</td>
</tr>
<tr>
<td>Preband moderate or severe TR, n (%)</td>
<td>9 (45)</td>
<td>20 (95)</td>
</tr>
<tr>
<td>Procedures before PA band, n, median (range)</td>
<td>1 (1–3)</td>
<td>0 (0–2)</td>
</tr>
</tbody>
</table>

Abbreviations are as defined in text.

Methods

Patients

Between 1989 and 2002, 41 patients were referred for PA banding to train the mLV. Two groups were defined (Table 1). No patients had anomalies associated with sufficient pressure or volume load to condition the mLV.

Group 1: Previous Atrial Switch

Twenty patients had previously undergone an atrial switch for dTGA. Five patients in this group had venous pathway obstruction, and 3 had a coexisting baffle leak.

Group 2: ccTGA

Of the 21 patients referred with ccTGA, 3 had previously undergone “conventional repair” with closure of a VSD but subsequently developed AV valve regurgitation and ventricular dysfunction. Two patients had hemodynamically insignificant VSDs. Moderate or severe Ebsteinoid malformation of the tricuspid valve was present in 4 patients. One patient had dextrocardia.

Selection Criteria

All patients were symptomatic and had objective evidence of hemodynamic abnormality (such as systemic AV valve regurgitation or mRV dysfunction). It has been our institutional practice to restore the mLV to the systemic circulation when this can be achieved with acceptable risk for an individual patient. This cohort included all patients identified in this manner from our patient population and some from other UK and European centers where this strategy is not used. This study details the outcome of patients for whom there was an intention to band the PA with a view toward further surgery.

Procedure and Clinical Protocol

The aim of the banding procedure was to increase the measured LV pressure, such that the ratio between mLV and mRV (PmLV/mRV) was 0.6, taking account of how the patient’s age, underlying diagnosis, or baseline LV pressure. In 33 patients, a catheter was introduced via the left subclavian vein and advanced across the mitral valve into the mLV to directly measure pressure. In cases where venous pathway obstruction or unusual anatomy prevented direct measurement (n=10), a pressure-measuring needle was inserted into the proximal PA to obtain an estimate of mLV systolic pressure (10 patients). Transesophageal echocardiography was used to observe ventricular function, AV valve regurgitation, and septal commitment. Epicardial echocardiography was used in 4 patients instead of transesophageal echocardiography.

Access was obtained via median sternotomy, although 2 patients in group 1 who were banded as adults were approached by thoracotomy. Silicone-coated Dacron tape was used to reduce the measured circumference of the PA by half and secured with polypropylene sutures. After a period of hemodynamic stabilization and observation of PmLV/mRV, the band was tightened further, and an estimate was made of the point at which ventricular function and systemic pressures were impaired by the band. At that point, the band was loosened by 1 mm. If hemodynamics and saturations were stable, this position was accepted. PA circumference was reduced from 85.4±25.3 to 41.3±12.6 mm (mean±SD) by a median of 53% (range, 33% to 66%). Postoperatively, dobutamine was used up to 10 μg·kg⁻¹·min⁻¹. Sedation was maintained for 6 to 12 hours before extubation. mLV pressure was continually monitored, and the catheter was removed on the morning after surgery. Rebanding was considered if the PmLV/mRV was <0.6, taking account of how the ventricle performed during the initial banding and the measured PmLV/mRV pressure on the first postoperative day. If the function of the mLV appeared satisfactory or good yet mLV/mRV pressures were <0.6, the band was tightened either that day or at some point in the following week. Late rebanding was performed in those who fulfilled similar criteria at subsequent cardiac catheterization.

Assessment of Ventricular and Valve Function

The principal method of assessing ventricular and valve function was transthoracic echocardiography. To avoid interobserver error and bias arising from knowledge of the clinical situation, a single cardiologist (C.B.) retrospectively reviewed echocardiograms at every time point (preband/early and late postband). Ventricular function and AV valve regurgitation were evaluated qualitatively, as routinely performed in our department. Band velocity and TR jet velocity were quantified objectively. Findings were scored according to a nil/mild/moderate/severe scale. Commitment of the interventricular septum was assessed as being to the LV, noncommitted, or to the RV. The thickness of the mLV posterior wall in diastole (LVPWd) was measured in the parasternal long-axis view immediately below the level of the tips of the mitral leaflets. z scores for LVPWd were calculated from normal data of Daubeny et al. Ventriculography at cardiac catheterization was also used to qualitatively assess ventricular function.

Follow-Up

This was a retrospective study, which involved a review of medical records, including cardiac catheter records, surgery reports, and outpatient correspondence, as well as a structured telephone interview with the treating cardiologist, local medical practitioner, and/or patient.

Patients were routinely recatheterized within 6 to 12 months for a formal measurement of the PmLV/mRV, ventricular cineangiography, assessment of band placement, and PA distortion. Valvar regurgitation was considered “significant” when graded as moderate or severe. Patients thought to be effectively trained achieved progressive increases in PmLV/mRV with near-systemic pressures in the mRV, significant increases in LVPWd, and normal mLV function. Duration of training was defined as the period between PA banding and anatomic repair, debanding, transplantation, death, or the end of the observation period (December 2002), whichever arose first.

For the purposes of this study, failure of training was defined as not achieving an anatomic repair within 2 years of PA banding. This figure was arbitrarily defined and reflects our clinical experience, as...
well as being within the 75% confidence interval (CI) of the median
time to operation in those achieving anatomic repair. Only 4 patients
were switched after this time, with 2 successful long-term survivors.
A failure of PA banding was also defined as death, debanding, or
transplantation within 2 years. Patients with <2 years follow-up after
PA banding (n=5) were excluded from the detailed analysis.

Two patients referred for PA banding were not banded at
operation and were excluded from the analysis: (1) A band could not
be safely applied to a 16-year-old female with a previous atrial
switch because of dense adhesions and (2) a 39-year-old man with
cctGA and severe TR was referred for PA banding because of
severe TR. There was no intraoperative improvement in TR after
application of the band, so debanding and tricuspid valve replace-
ment were performed. It was thought that the likelihood of his
undergoing successful mLV training and anatomic repair was low on
the basis of his age.

Data Analysis
Data were analyzed by ANOVA with SPSS for Windows (version
10, SPSS Inc). Continuous variables are expressed as mean±SD or
median and range, and comparative univariate analyses were per-
formed with the t test, Mann-Whitney U test, or Wilcoxon signed-
rank test, as appropriate. Binomial or ordinal data are expressed as
percentages, and comparative univariate analyses were performed
with the χ² test, 2-sided Fisher exact test, or binomial logistic
regression, as appropriate. A probability value P<0.05 was taken to
represent a statistically significant difference between groups.

The effect of preoperative, operative, and postoperative variables
on outcome was tested by univariate and multivariate analyses. Univariate analyses of early-outcome measures were made with the χ² test, 2-sided Fisher exact test, and binomial logistic regression. Variables with P<0.1 were included in a stepwise logistic regression model. Results of these multivariate analyses have been expressed as odds ratios (ORs) with 95% CIs for variables with P<0.05.

Actuarial survival, freedom from reoperation, and freedom from
reintervention were estimated by the Kaplan-Meier method. These
results have been expressed as probability estimates ±SEM. Uni-
variate analyses of actuarial outcome measures were made with the
log-rank test. Variables with a probability value P<0.1 were
included in a stepwise Cox regression analysis. Results of these
multivariate analyses are expressed as likelihood-ratios with 95% CI
s for variables with P<0.05.

Results
Follow-Up
Follow-up was complete, with a median follow-up of 4.3
years (range, 25 days to 12.6 years). For detailed outcome
data, see Figure 1.

Procedural Results
PA Banding
Two patients had a prolonged intensive care course after PA
banding. One patient developed acute renal failure and
jaundice in the setting of impaired ventricular function. The
other patient had repeated supraventricular tachycardias. Both
patients recovered and went on to successful anatomic repair.

Debanding
One patient required early removal of the PA band (deband-
ing) on the day of operation. A 2.8-year-old had previously
undergone a conventional repair (Rastelli procedure for
cctGA and VSD) and was referred for banding of the LV-PA
conduit in the setting of early, severe mRV failure. This band
was removed during the evening of the procedure during an

Figure 1. Schema of outcomes. PAB indicates PA band; TVR, tricuspid valve regurgitation. All other abbreviations are as defined in text.
episode of hemodynamic instability associated with supraventricular tachycardia. The patient was discharged from hospital with severe RV dysfunction but re-presented in cardiogenic shock and died 3 weeks after the conduit banding.

Two patients required late debanding: (1) An 11-year-old girl with ccTGA with VSD who had undergone VSD closure elsewhere was referred for PA banding in the context of mRV failure and severe TR. Banding produced a rise in $P_{mLV/mRV}$ from 0.5 to 0.8; however, severe TR persisted. Seven months later, the patient underwent debanding at the time of systemic AV valve replacement and is currently well. (2) A 20-year-old male who had undergone an atrial switch and subsequent repair of a baffle leak was referred for banding because of a failing mRV. He was banded to a $P_{mLV/mRV}$ of 0.7, but this fell to 0.5 on the first postoperative day, so he was rebanded to a $P_{mLV/mRV}$ of 0.8. He was debanded at 10 months in the face of deteriorating mLV function without evidence of other benefit from the band. He died of congestive cardiac failure 2 months later.

**Rebanding**

Eight patients required tightening of the band (rebanding) early after the initial procedure. A further 5 patients required late banding. The median interval between initial PA banding and rebanding was 200 days (range, 181 to 508 days). No patients requiring early rebanding underwent late rebanding.

Of the 13 patients who were rebanded, only 4 reached anatomic repair (30%), and 5 died or underwent transplantation (38%). Three of 4 patients who had undergone anatomic repair died or underwent transplantation. Of the late rebandings, only 1 of 5 is a long-term survivor, despite 3 of 5 having achieved anatomic repair. Sixty-two percent of those requiring rebanding had undergone a previous atrial switch.

**Age at Banding**

There were 12 patients banded after the age of 16 years (range, 17.0 to 39.1). Of these, 8 have a palliative PA band, 3 died or underwent transplantation, and 1 underwent anatomic correction. Those achieving anatomic correction were younger at the time of banding (median, 5.9±1.4 years; range, 0.1 to 22.7 versus 15.0±2.1 years; range, 0.9 to 29.1; $P<0.01$).

**Effects of PA Banding**

**Changes in $P_{mLV/mRV}$ After Banding**

There was no difference in preband $P_{mLV/mRV}$ according to pathology or age. PA banding was associated with a marked increase in mLV pressure. The $P_{mLV/mRV}$ rose acutely from 0.43±0.14 to 0.72±0.13 ($P<0.05$). There was a relative drop in mLV pressure during the early postoperative period. $P_{mLV/mRV}$ values observed on arrival in the intensive care unit and the first postoperative day were lower than those achieved during surgery ($P<0.05$ for both). $P_{mLV/mRV}$ was higher at subsequent time points than during the first 24 hours postoperatively ($P<0.05$).

Figure 2 demonstrates $P_{mLV/mRV}$ according to whether the patient achieved anatomic repair within 2 years of banding.

**Changes in Echocardiographic Parameters With Banding**

PA banding was associated with a late reduction in mRV dilatation ($P<0.05$), an early and a sustained reduction in mLV compression ($P<0.05$), and an early and a sustained change in commitment of the interventricular septum to the mLV ($P<0.05$; Table 2).

We did not demonstrate early or late improvement in significant TR ($P=0.26$) in the group as a whole. PA banding was associated with a late reduction in significant TR in the subgroup that subsequently underwent anatomic repair ($P<0.05$). In this group, the proportion of patients with significant TR fell from 70% (n=14) preoperatively to 53% (n=9) late after PA banding. However, in the group that did not achieve anatomic repair, there was no sustained improvement in TR ($P=0.16$). It is noted that 50% of the atrial switch cohort did not have significant TR before banding. Separate analysis of the ccTGA cohort showed no alteration in significant TR after PA banding.

**Changes in LVPWd**

Overall, PA banding increased the LVPWd mean±SD z score from −0.95±1.81 before banding to 0.58±2.02 early after banding, and this was change was sustained to the late observation time point (0.96±2.28, $P<0.05$). There was no difference in LVPWd z score between the early and late time points in the group as a whole. PA banding was associated with a late reduction in LVPWd ($P<0.05$).
points, nor was there a difference between those switched versus those following a palliative course at any time point.

Changes in Functional Status
There was an overall improvement in New York Heart Association (NYHA) functional class from a median of II (II–IV) to I (I–III) at the late time point \((P < 0.05)\). Functional class improved in 67%, was unchanged in 27%, and worsened in 9%. Improvements were observed regardless of outcome: 65% of those achieving anatomic repair were improved versus 67% of those with a palliative PA band were improved at the late time point.

Changes in mLV Function
PA banding was associated with the development of LV dysfunction. Sixty-five percent \((n = 20)\) of patients with normal mLV function before PA banding developed mLV dysfunction early after operation, and 30% of these \((n = 6)\) had significant mLV dysfunction. These changes persisted to the late time point.

Six patients with preexisting mild LV dysfunction underwent banding. One underwent anatomic repair after rebanding but died of ventricular failure while awaiting transplantation. Three have died or have undergone transplantation, 1 is scheduled for transplantation, 1 has NYHA class III symptoms, and 1 has undergone debanding and systemic AV valve replacement. LV end-diastolic pressure was not altered by PA banding (data not shown).

Duration of Training
The duration of training for the group that achieved anatomic repair was a median of 14.1 months (range, 8 days to 4.7 years).

Survival
Survival of the Entire Cohort
Actuarial survival for the entire group \((n = 41)\), mean \pm SEM) was 92\pm5% at 2 years and 76\pm8% at 5 and 10 years. There was no difference in actuarial survival between those who achieved anatomic correction and those who did not \((P = 0.94)\; \text{Figure 3}\).

Survival With a PA Band
Overall survival of patients with a PA band was then examined to evaluate the safety and efficacy of the intervention in this group of patients to the point of anatomic repair, death, debanding, or transplantation. In this way, actuarial survival for the group was unaffected by the inclusion of survival data after anatomic repair, which could otherwise be expected to falsely improve overall survival. Actuarial survival for the group was \((\text{mean} \pm \text{SEM}) 91\pm5% at 2 years and 83\pm9% at 5 and 10 years (Figure 3).

Univariate analysis identified preoperative mLV dysfunction (mild versus none) as an important factor associated with an increased risk of mortality or transplantation \((P < 0.05)\). Notably, the following factors did not influence survival with a PA band: (1) diagnostic group (atrial switch versus ccTGA, \(P = 0.63\)); (2) indication for intervention (RV dysfunction versus TR, \(P = 0.44\)); (3) age <16 or \(\geq 16\) years, \(P = 0.40\); and (4) severe preoperative RV dilatation (vs moderate dilatation, \(P = 0.34\)). Survival with a PA band was better in patients who subsequently went on to anatomic repair. In those who do not achieve repair, the survival was 79\pm11% at 2 years and 71\pm12% at 5 and 10 years after banding. Univariate analysis identified 3 factors associated with failure of the training strategy (death, transplantation, or debanding): (1) mild LV dysfunction before PA banding; (2) development of significant mLV dilatation and dysfunction; and (3) postoperative development of late TR.

### Table 2. Effect of PA Banding

<table>
<thead>
<tr>
<th>Observed Parameter</th>
<th>Before Band ((n=39))</th>
<th>Early After-Band ((n=35))</th>
<th>Late After-Band ((n=30))</th>
</tr>
</thead>
<tbody>
<tr>
<td>Moderate or severe mRV dilatation</td>
<td>100%</td>
<td>91%</td>
<td>87%*</td>
</tr>
<tr>
<td>Moderate or severe mRV dysfunction</td>
<td>51%</td>
<td>31%*</td>
<td>39%</td>
</tr>
<tr>
<td>Moderate or severe systemic AV valve regurgitation</td>
<td>69%</td>
<td>47%</td>
<td>57%</td>
</tr>
<tr>
<td>mLV compression</td>
<td>85%</td>
<td>14%*</td>
<td>6.7%*</td>
</tr>
<tr>
<td>mLV dilatation</td>
<td>0%</td>
<td>17%</td>
<td>19%</td>
</tr>
<tr>
<td>mLV dysfunction</td>
<td>0%</td>
<td>17%*</td>
<td>20%*</td>
</tr>
<tr>
<td>Septal commitment to LV</td>
<td>14%</td>
<td>54%*</td>
<td>60%*</td>
</tr>
<tr>
<td>LVPWd, z score, mean \pm SD</td>
<td>(-0.95 \pm 1.81)</td>
<td>(0.58 \pm 2.02^*)</td>
<td>(0.99 \pm 2.24^*)</td>
</tr>
</tbody>
</table>

Abbreviations are as defined in text.
*Significant vs preband value.

Figure 3. Kaplan-Meier survival curve of events include death and transplantation. Patients were excluded from analysis at point of anatomic repair if this occurred. Values shown are mean \pm SEM.
high-resistance systemic circulation. It is acknowledged that pulmonary vascular bed while the mRV contends with the low-resistance coronary supply, and relative coronary insufficiency has been cited as a potential cause of progressive RV failure, particularly in cases where RV hypertrophy coexists.

There are intrinsic differences between the shape and structure of the 2 ventricles that relate in part to their different embryological origins. The mRV has a tripartite crescentic shape, which may be less suited to sustained, high-pressure contractile function. It has a relatively low ejection fraction and reduced myocardial functional reserve, and it is associated with a relatively poor functional adaptation to pressure and volume overload. In addition, the RV has only a single coronary artery supply, and relative coronary insufficiency could be stabilized and in some cases, improved by PA banding. This has been observed by other groups in the management of patients after previous atrial switch for dTGA. It is perhaps too early to say whether these changes are sustained, and we cannot easily explain why a longer-term benefit is seen in those not undergoing anatomic repair. Some suggest that by banding the PA, a leftward shift in septal commitment is achieved, and a long-term reduction in TR is observed. Our findings do not support this statement—we saw no sustained improvement in TR for the group as a whole, only in those going on to anatomic repair. It is not clear why improvements in functional status are not mirrored by sustained improvements in hemodynamic variables such as TR. This may simply relate to changing patient expectations and lifestyle compensations.

All of the patients in this series were symptomatic and had either systemic AV valve regurgitation or mRV dysfunction that mandated surgical intervention. In recent years, the threshold for intervention has been reduced—how to effectively train the mLV remains topical, despite a diminishing number of patients presenting with systemic mRV dysfunction after previous atrial switch. Young patients train quickly, and in infants, the response to banding is very fast, with a doubling of LV mass in only 1 week, although some of this may represent myocardial edema. At the other end of the spectrum, there may be a point at which it is no longer possible to effectively train the ventricle. In our group, older patients did benefit from banding but were unlikely to some patients may do well for a long period of time with this circulation; however, our group of patients developed early problems, with a combination of TR, mRV dilatation, and ventricular failure. It is not known why these problems occur early in some and late in others, but most investigators agree that the genesis of the problem is progressive commitment of the interventricular septum to the mRV, which then affects the tension apparatus of the systemic AV valve, resulting in valvar incompetence.

Discussion

There is considerable debate regarding the best approach to patients with an mRV in the systemic circulation. The majority of new cases in this situation have ccTGA. For patients in whom there is a coexisting VSD, many centers now follow a strategy that restores the mLV to the systemic position, either with a double switch procedure (atrial and arterial switch) or an atrial switch with a Rastelli reconstruction where necessary. Good results have emerged from many centers using this approach, and it represents a serious alternative to conventional repair, wherein the VSD is closed without correcting the AV and ventriculoarterial discordance.

The patients we have described in this study are a rare and more complex group. In the absence of a VSD or subpulmonary obstruction, the mLV works against the low-resistance pulmonary vascular bed while the mRV contends with the high-resistance systemic circulation. It is acknowledged that univariate analysis identified 3 factors associated with the likelihood of anatomic repair: (1) younger age (<16 years); (2) higher PmLV/mRV on arrival in the intensive care unit, at day 1, and late postoperatively; and (3) leftward change in septal commitment after PA banding.

Survival After Anatomic Repair

Survival of patients undergoing anatomic correction after a period of LV training was 84±9% at 1 and 2 years and 76±11% at 5 and 9.5 years after anatomic correction. There was no difference in survival between patients undergoing LV training before anatomic repair with those undergoing a double switch without first requiring training (P=.3, Figure 4). Results for the untrained cohort have been previously published. Importantly, mLV dysfunction before anatomic repair was a risk factor for death after anatomic repair (OR=7.1; 95% CI, 1.0 to 51.1).

Figure 4. In Kaplan-Meier survival curve for those achieving anatomic repair after PA banding, similar outcomes were achieved compared with those undergoing anatomic repair but not requiring training (eg, ccTGA with VSD; P=.30). Abbreviations are as defined in text.
achieve anatomic repair if mLV training commenced after age 16. Over time, it is apparent that we have attempted to train patients earlier, when they are in better general condition. This may also relate to increasing confidence in the safety and efficacy of definitive anatomic repair at our institution.

Our results reinforce the importance and difficulties inherent in assessing mLV function at all points from banding to anatomic repair. Ventricular damage may result from a band being applied too tightly or for too long. Our management protocol was mostly dependent on observing changes in pressure developed by the mLV compared with the systemic ventricle. Echocardiography and cineangiography are useful tools in demonstrating mLV function but remain highly subjective. With current techniques, titrating afterload to adequately but safely train the mLV is largely a matter of surgical judgment and experience. Hemodynamic changes related to the band may take up to 7 minutes to fully evolve, and excessive tightening may lead to ventricular fibrosis in humans as well as animal models. Others have used a protocol involving multiple early rebandings and earlier anatomic repair to minimize the risk of perioperative myocardial injury associated with subendocardial ischemia.

In the longer term, it is difficult to estimate how long the window is between sufficient training and onset of ventricular dysfunction due to banding. Because the response to banding in children beyond the infant age group is hypertrophy rather than hyperplasia, diastolic dysfunction may rapidly develop and seriously compromise outcome. In several instances, we observed poor outcomes in those who had been banded for a long period, wherein the LV generates good systemic pressures but does not contract or relax well. Because mLV dysfunction was a poor prognostic factor at every time point in the treatment protocol, it may be that many of those undergoing anatomic repair in our series could have safely and perhaps optimally undergone this operation earlier. The development of remotely adjustable PA bands may minimize detrimental effects of acutely banding patients if an adequate narrowing can be achieved over a period of days to weeks.

Our study has a number of limitations. First, this was a retrospective, noninterventional study designed to evaluate outcomes of an established clinical program. All patients were managed as individuals and not according to a treatment protocol, which would have improved our ability to analyze outcomes. Second, echocardiographic assessment of ventricular function was qualitative. Most mLVs were not of the standard geometric LV shape secondary to raised RV pressure. Thus, they were not amenable to standard 2D echocardiographic evaluation of function. Nevertheless, all echocardiograms were reviewed in a blinded fashion by a single cardiologist in 4 sessions to eliminate interobserver error and bias that might have been introduced over time. Third, clinical assumptions about LV mass are based on assessments of LV PWd and assume that posterior wall changes mirror global changes in the mLV, which may not be true. Magnetic resonance–based techniques may provide a better assessment of LV mass in this context. Finally, objective assessment of functional performance was not carried out in this study.

Conclusions
PA banding is a safe procedure and should be considered in all patients with a systemic mRV and an untrained subpulmonary mLV who develop mRV dilation, dysfunction, or AV valve incompetence. In young patients, this should be performed early in the natural history, because severe mRV dilation and dysfunction are associated with a worse outcome. Later in life, when the likelihood of eventual anatomic repair is small, a significant proportion of patients derive benefit from the procedure that may delay the onset of more severe symptoms. In those who can be effectively trained, the outcome is not worse than in those who undergo anatomic repair where the mLV is maintained by a coexisting pressure or volume load. Outcomes may be further improved by better assessment of mLV function during the training protocol.

References
Intention-to-Treat Analysis of Pulmonary Artery Banding in Conditions With a Morphological Right Ventricle in the Systemic Circulation With a View to Anatomic Biventricular Repair


_Circulation_. 2005;111:405-411
doi: 10.1161/01.CIR.0000153355.92687.FA

_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2005 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/111/4/405

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in _Circulation_ can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to _Circulation_ is online at:
http://circ.ahajournals.org//subscriptions/