

Pulmonary artery banding: Rationale and possible indications in the current era

Rajesh Sharma

Department of Pediatric Cardiac Surgery, Fortis Escorts Heart Institute, New Delhi, India

Muller and Dammann described pulmonary artery banding (PAB) for palliation of congenital heart disease (CHD) with pulmonary hypertension (PAH) secondary to increased pulmonary blood flow in 1952, a time when intra cardiac repair was virtually nonexistent.^[1] When it did come about, PAB continued to occupy an important place in surgical treatment of CHD, due to increased risk of cardiopulmonary bypass (CPB) in the very young infant. The purpose of PAB was to protect lungs from unrestricted pulmonary flow while allowing corrective surgery to be postponed to a safer future, when the cardiac structures of the child was bigger. Pulmonary artery banding itself has not been an entirely benign procedure, being associated with a not insignificant morbidity and mortality. Branch pulmonary artery distortion secondary to band migration, band erosion into pulmonary artery lumen, pulmonary valve distortion, subannular ventricular hypertrophy are all described complications. Trusler, *et al.*, proposed guidelines for pulmonary artery banding for cyanotic and acyanotic situations.^[2] The target distal pulmonary artery pressure (PAP) is conventionally 30-50% of systemic for a biventricular heart, while it should be as close to the Fontan pressure (mean of 15 mm Hg), as possible, in a univentricular situation.^[3]

With time, CPB-associated risks have been reduced to a minimum, while mortality of PAB has still remained higher (these groups are no longer comparable as the straightforward septal defect would undergo one stage primary correction, while possibly only the higher risk group would be submitted for a PAB). In addition, separate risks of two procedures with an interim waiting period with possibility of intercurrent attrition increase the cumulative risk to above that for primary closure of a routine septal defect. Pulmonary artery banding is therefore today referred to more as a historical procedure in the surgical treatment of the straightforward septal defect.

PAB exists in its original role today for the small infants with

1. Swiss cheese type of multiple ventricular septal defects

2. Postponing the choice between biventricular and univentricular repair in atrioventricular or ventricular septal defects (VSD) with ventricular imbalance.
3. Defects with complex anatomy, often associated with a common atrioventricular connection or a criss-cross heart, where a biventricular repair of better quality may be possible later in life when the cardiac structures are bigger.
4. Univentricular physiology with unrestricted pulmonary blood flow to drop PAP and pulmonary vascular resistance to levels suitable for future univentricular palliation.
5. The extremely marasmic, septic infant, or in one who presents with a contraindication for CPB, eg., recent intracranial bleed.

Newer indications that have joined the above list, aim at the following:

1. Left ventricular preparation for an involuted subpulmonary left ventricle of late-presenting simple D-transposition of the great arteries (TGA), in preparation for an arterial switch operation.
2. Left ventricular preparation and/or reduction of tricuspid regurgitation (TR) in congenitally corrected transposition of the great arteries (CCTGA) with tricuspid incompetence without sizeable VSD.
3. PAB as an adjunct to another procedure, eg., 1. bidirectional Glenn shunt to maintain antegrade flow with acceptable superior caval pressures, 2. as part of a palliative arterial switch operation.

Controversial indications would include

1. Routine initial PAB for CCTGA VSD in a proposed strategy towards preserving left ventricular function following anatomic repair.
2. Adjunctive PAB along with physiological correction of late presenting simple TGA for future conversion to anatomic repair.
3. PAB for reversing the advanced pulmonary vasculopathy of unoperated CHD with Eisenmenger's syndrome.

In this paper, Dehaki *et al.* present their five-years cumulative experience with PAB till April, 2010.^[4] All

Address for correspondence: Dr. Rajesh Sharma, Director, Pediatric Cardiac Surgery, Fortis Escorts Heart Institute, Okhla Road, New Delhi - 110 025, India.
E-mail: rsharmacvs@hotmail.com

PABs were performed for PAH secondary to increased pulmonary blood flow situations.

Out of a total of 305 PABs in their series, 57.4% were for VSDs (large or multiple), 20.6% for atrioventricular septal defects, 3.9% for VSD with coarctation, 4.3% for double outlet of the right ventricle, 10.2% for TGAVSD, and 2.6% for single ventricle situations. In their own words, 20% had complex heart disease and sepsis while 45% had a weight below 4 kg. The mean age and weight at the time of PAB were 7.12 ± 5.2 months and 5.5 ± 1.6 kg, respectively.

PAB was classified as anatomically effective if it reduced the MPA diameter to 50% in the banded segment (achieved in 97%) and functionally effective if there was reduction of PAP to 50% of systemic pressure or below (achieved in 92%). This was achieved in equal measure by any of the three described methods of PAB that they mention, i.e., guided by direct pressure measurement, by Trusler's formula, or by relying only on O_2 saturations without any pressure measurement, without any difference in efficacy.

In hospital PAB mortality was only 2%. The complication rate was also very small.

Completion of repair of 78% of the banded patients was carried out at a mean gap of 23 ± 10 months. The rest were on follow up. There was no attrition in the waiting period for the entire group (a total mean follow up of 39 ± 20 months). In-hospital mortality for the corrective procedure was 3%. Among the patients who underwent correction, most of them had removal of their bands and only 20% received a pulmonary artery augmentation by a patch. There was no difference in gradient in the MPA in the patched or nonpatched patients, postoperatively.

The authors conclude that PAB is still a low risk temporising option especially in centers ill-equipped for open cardiac surgery in the small child and a useful procedure for the group where CPB may be a high risk strategy even for centers routinely performing primary repairs.

The following discussion deals only with PAB for the indication which the above paper has dealt with.

The most notable feature of this publication is the very low complication rate following initial PAB, right through the interval period and including the second stage complete repair. For this, the authors needs to be congratulated for, achieving such low rates of negative outcomes, in a milieu which is less than ideal (as they themselves state), is no mean task.

I have selected some aspects of the paper and I mention my own thoughts on these:

PAB - HOW TIGHT?

Adequate banding has traditionally aimed to result in a reduction in distal pulmonary artery pressures to 30-50% of systemic level. As a guide, the Trusler formulae are followed as a first step with finer adjustments being made keeping in mind systemic O_2 saturation, distal PAP, and visual cardiac action. Additional criteria that groups have variously advocated are central venous pressure, mixed venous O_2 tension and end tidal carbon dioxide in the expired gases, compared to these values prior to band placement. In general, the higher the systemic O_2 saturation after PAB (the safety margin), with the lower the distal PAP (the pulmonary protection), the better.

The Trusler formula aims for adequate PAB in the normally related great artery (NRGA) group but in the TGA and related group with admixture defect, it cedes effectivity to O_2 saturation. Hence it brings in an extra 4 mm of circumference for the same weight (20+ wt in kg vs 24 + wt in kg). This extra 4 mm is placed in the formula to prevent the aggravation of preexistent hypoxia in the admixture lesion. In my own experience, PA banding by Trusler's formula in a TGA situation is a poor way of reducing PAP. The end result is usually unacceptable hypoxia with persisting high PAP. In this situation, even concomitant atrial septectomy with its theoretical salutary effect on mixing helps little by way of better saturations or a more effective band. Therefore, in a TGA situation, complete repair is without question a safer and more effective strategy. In a univentricular situation with TGA, or an intracardiac anatomy not immediately conducive to septation, we would prefer to convert the TGA to a NRGA (by means of an arterial switch) and then band the MPA on merits-a palliative ASO, i.e., This may sound like overkill, but in our experience, it is a far less morbid (and effective) strategy than a simple PAB!

I have issue with the authors describing a 50% drop in pressure as adequate banding. In a patient with NRGA with normal lungs, even with PAB with distal pressure down to 30%, the resulting O_2 saturation is above 90%, which means a $Q_p/Q_s > 2:1$. Routinely accepting 50% of systemic pressures in the pulmonary arteries after PAB would imply a Q_p far in excess of Q_s . This would go against one of the aims of PAB, i.e., relief of respiratory issues and would also permit continued large steal of cardiac output to the pulmonary circulation, perpetuating the pre-existent abnormal physiology.

There is no mention of whether the single ventricle group has been treated in any different ways from the remainder. A 50% distal PAP is completely an inappropriate PAP for future univentricular repair.

AGE AND WEIGHT AT PAB

The age range of both weight and age shows that the oldest child was 12 months and the heaviest 7 kg at time of PAB. This is far in excess of the age where one would expect CPB to still be an increased risk. The justification of PAB in children at this end of the age and weight range is unclear.

SECONDARY REPAIR

In the paper, 80% of PAB could just be removed without need for patching without any excessive residual gradients. In general, a pulmonary artery in our experience would spring back to its original dimension till an outer limit of six months from the time of initial banding. Beyond this time, with a good band, the pulmonary artery will not allow the minimum full size Hegar dilator through, thus necessitating some form of arterioplasty (resection and end to end anastomosis or patching). The one situation where one may get away without any additional procedure would be if the banded segment was large enough, i.e., if the band was loose. This would seem to correlate with the 50% aimed reduction of distal PAP.

ROLE OF PAB IN SEPSIS, LOW BIRTH WEIGHT, INTRACRANIAL BLEED

In general our approach has been to treat the sepsis and then go for complete repair. In situations where the child is on positive pressure ventilation (IPPR) with pulmonary infection, we would wait for the infection to show a response by way of reduction in leucocytosis, C reactive protein, or procalcitonin level, for clinical lung signs to disappear, to go for complete repair without waiting for complete resolution. Recovery from open intracardiac repair in these situations is admittedly slow, but the great advantage in postoperative management is that there is no residual pulmonary overflow.

The problem with PAB in the presence of pulmonary dysfunction is that it may be inadequate, because of preexistent hypoxemia and, therefore, inability to tighten the band to the desired level, which would imply greater pulmonary overflow despite banding. This would subsequently come in the way of separating from IPPR. Often, one would need to complete the repair after several unsuccessful attempts at weaning from ventilator.

In the low birth weight group, end points of PA banding are not defined, measurement of PAP is inaccurate and unhelpful secondary to small size and low systemic pressures under anesthesia and thus a complete repair

with its implied restoration of normal physiology may once again be the best way out.

Recent intracranial hemorrhage would probably be the only noncontroversial indication for PAB if a procedure is required urgently.

PA BANDING WITHOUT PRESSURE MEASUREMENT

While Trusler's formula does provide a starting point, it is not uncommon to have to modify the tightness of the band based on direct pressure measurements in the pulmonary artery distal to the band and systemic O₂ saturations. It is common knowledge that even minor changes in band tightness will result in major changes in pulmonary artery pressure. If one is to aim for ideal band tightness (close to 30% or a mean pressure of 15 mm Hg depending on whether we are dealing with a biventricular or a univentricular heart), direct pressure measurement, I feel, is critical.

PA BANDING - A BENIGN PROCEDURE?

For a child with increased pulmonary blood flow but without respiratory symptoms, a band on the loose side (50% reduction in PAP) would probably suffice. The more a child presents with respiratory symptoms, the more one would have to concentrate on reducing the Q_p. The tighter the PAB gets, the lesser the safety margin (and hence the inherent safety of simply correcting the underlying defect instead of taking the circuitous route via a PAB). Hence, if one desires the full benefit from a PAB, the difference between a perfect and a too tight band is very small. If we were to band loosely, one is in a way not changing the preoperative loading conditions significantly, and a child who was not in major respiratory compromise secondary to excessive pulmonary blood flow would have a high probability of surviving the banding procedure. If one were to band loosely routinely, one is likely to have a low early risk of the banding procedure. Whether he benefits from the PAB (vis a vis Q_p/Q_s reduction, increase in systemic output, prevention of future respiratory complications, gain in weight, etc) would not be evident straightaway.

The two basic hemodynamic differences between PAB and closure of defect are: 1. The right ventricle remains hypertensive after PAB, while it becomes low pressure following defect closure, and 2. as already stated, PAB can never be safely so tight as to reduce Q_p/Q_s to 1:1, an endpoint that is always achieved with defect closure. Following effective PAB, even with an unrestrictive interventricular communication and an

unobstructed systemic outlet, one sometimes does see right ventricular dysfunction. This is a temporary phenomenon, but can make a patient quite sick. Such a child following PAB can sometimes require supportive care in the intensive care unit, often in excess of a completely repaired physiology.

The argument that PAB can be safely performed in centers that are not equipped for infant cardiac surgery may not, therefore, be entirely true.

One last point, despite all of the above rationale, one cannot detract from the fact that the results published here are some of the best results with PAB that are to be found in the literature. The banded children have recorded good weight gain on follow up, have not succumbed to intercurrent illnesses (as one would have expected in some if the bands were too loose), and have presented for their second stage repairs in good condition. In short, they have behaved like those with good bands. Obviously, then there is a take home message here from a group that has a huge continuing experience with this two stage approach. And that is that for all biventricular repair candidates, we need not aim to band everybody to 30% systemic pressure. And that is why the forefathers gave us the range: 30-50%!

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