Tricuspid regurgitation in single ventricular palliation for corrected transposition

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Keywords: Congenitally corrected transposition of the great arteries • Tricuspid regurgitation • Single ventricle palliation

We have read the interesting article from Hsu et al. [1], reporting the surgical outcomes for patients with corrected transposition of the great arteries (CTGA) who underwent physiological repair, anatomical repair and single ventricular palliation (SVP). With regard to lower mortality and morbidity after Fontan palliation in their cohort, the authors concluded that they prefer SVP in patients with complex anatomy.

As Karl [2] from Brisbane listed in his review in 2011, potential advantages of Fontan palliation managing CTGA included technical simplicity, less arrhythmia, absence of a Raselli conduit and less left ventricular outflow tract obstruction. More importantly, the left and right ventricle (RV) are both working as the systemic ventricle, theoretically eliminating the development of heart failure. In 2005, Hraska et al. [3] also proposed the role of Fontan palliation in complex CTGA. In the case with remote VSD, chordae straddling and anomalous pulmonary venous connection, this approach is fully justified with favourable short- to mid-term mortality and morbidity which was confirmed by the current study. However, there were some significant issues influencing the surgical outcomes.

A potential confounding factor was that no patients who underwent SVP presented with preoperative tricuspid regurgitation which was the major manifestation of morphological RV dysfunction. As the authors in this study and Hörer et al. [4] in his previous study analysed, patients with significant tricuspid valve regurgitation showed the worse outcome after surgery, raising the question of whether the morphological RV function was a true risk factor rather than the surgical strategy? And why was there no tricuspid regurgitation in this group? Was tricuspid regurgitation a contra indication of SVP in the authors’ strategy? A study from Moscow [5] suggested that atrioventricular regurgitation increased the risk of poor outcome after SVP. Tricuspid insufficiency could limit Fontan function in the long term as it has in the physiological strategies as this valve is still subjected to systemic pressure.

There is a lack of consensus when it comes to the timing of SVP. When was SVP or anatomical repair performed if a CTGA patient presented with no tricuspid regurgitation and no severe cyanosis? Moreover, clinicians are frequently impressed by the fact that patients with CTGA and pulmonary outflow tract obstruction fare better than those without. They can even survive decades with minimal or no symptoms and require no intervention. Interestingly, 78% (18/23) of patients in the SVP group had significant pulmonary obstruction. The pressure gradients and indication for SVP are required in detail to propose SVP as a preferred strategy.

Of course, the authors should be congratulated for their excellent surgical outcomes and excellent analysis which transferred knowledge to us. As Karl [2] stated in his review, Fontan timing and technique might have a different influence on outcomes for CCTGA patients than for other true univentricular patients. Physicians may benefit from their detailed surgical strategy including the timing and indication for various approaches.

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Reply to Ma et al.

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We were glad to receive the comments from Ma et al. [1], regarding our recently published article [2]. This is a retrospective study with chart review using our patients’ medical records. In our data, there was no moderate to severe tricuspid regurgitation (TR) in our patients receiving single ventricle palliation (SVP) [2]. We chose to perform SVP in this group of patients due to the presence of severe pulmonary stenosis or pulmonary atresia combined with other anatomical restrictions (e.g. remote type ventricular septal defect (VSD), chordae straddling, unusual coronary artery pattern and unbalanced ventricle) [2]. There was only 1 patient with moderate TR receiving valve repair before the SVP, and the postoperative echo showed no aggravation of valve regurgitation [3]. Why was there no patient receiving SVP with severe TR in our study? We believed severe tricuspid valve regurgitation is not an indication for SVP, and anatomical repair might be helpful, despite the suboptimal long-term result [4]. We think the progression of TR severity is slow and insidious. When TR becomes apparent, it might be due to structural defects (i.e. Ebsteinoid anomalies) or be regarded as a sign of morphological RV dysfunction. We performed SVP in the early years of the patients’ lives (mean age of 8.6 years) to prevent TR progression in the near future and preserve ventricular function [2, 4–6]. Our article presented only the short- and mid-term results; the long-term follow-up results are still unknown.

Although there is no consensus for the timing of performing surgical repair in patients without severe TR or cyanosis, we preferred to perform the operation earlier, before right ventricular failure [2, 5, 6].

Our patients with congenitally corrected transposition of the great arteries typically presented with cyanosis or other heart failure symptoms. Patients without symptoms usually do not seek medical help and are diagnosed accidentally for other medical diseases. If there were no major cardiac anatomical anomalies such as large VSD, severe pulmonary stenosis or atresia, and atrioventricular valve defects with severe valve regurgitation that would lead to heart failure in the future, surgical intervention would not be beneficial for these patients [6].
Which animal model is proper for evaluation of a muscular ventricular septal defect closure device?

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We have read the great article by Lang et al. [1], which showed the feasibility of closure of a ventricular septal defect (VSD) with bacterial nanocellulose (BNC) patches and the biocompatibility of the BNC. They used a muscular VSD pig model which created an iatrogenic muscular VSD before performing VSD closure, because their device was aiming to close the muscular VSD. In the case of a perimembranous VSD, Yucatan minipigs are a suitable animal model because of their naturally occurring VSD [2]. However, choosing an animal model for muscular VSD is a challenge. If the VSD was created iatrogenically, then acute myocardial injury related with puncture could affect the healing response following the implantation of an occlusion device [3]. We believe that the iatrogenic muscular VSD animal model is an apt model for a feasibility test study of a VSD closure device; but we would like to know whether the iatrogenic muscular VSD model is good enough to evaluate the long-term effects such as the healing process or inflammation related with BNC patches.

The authors compared the results of the BNC patch with those of the polyester patch in their previous study [4]. They observed more pronounced tissue overgrowth on top of the polyester patch compared with the BNC patch [1]. They used two different breeds of pigs in the present study and the previous one: German landrace pigs and Göttingen minipig. Would it be possible to compare the tissue reaction of these two patches in the same animal or at least in the same breed.

We would like to thank Son et al. [1] for their interest in our study [2]. We agree that there is a paucity of suitable animal models for closure of muscular ventricular septal defects (VSDs) in contrast to animal models for perimembranous VSDs [3]. There are a few reports which describe a similar technique. There are also reports which describe creation of VSDs by balloon dilatation [4]. We believe that a substantial defect in the muscular interventricular septum is essential for a meaningful animal model. Therefore, we decided to create a muscular VSD with a specially designed core device [5].

We further agree that tissue reactions to the myocardial damage caused by creation of the VSD might affect the healing response. However, we have not seen extended necrosis or significant inflammation at the edges of the defect on histology. In addition, this animal model might even negatively influence the results of biocompatibility testing of our device as the reaction to the myocardial injury adds to the reaction to the device. Thus, the biocompatibility of the patches might even be more favourable than demonstrated by our study.

Amin et al. [6] developed a slightly different animal model of muscular VSDs: After creation of the VSDs, the animals were observed for 3–6 weeks and closure of the VSDs was performed in a second step. During this observation period, edges of the VSDs can heal and histology might be more representative for the healing process of the VSDs. We decided against this approach.

We agree that using two different breeds of pigs might lead to different tissue reactions which limit the comparison between these two different patches. To have a valid comparison, it would have been more appropriate to compare the tissue reaction of these two patches in the same animal or at least in the same breed.

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Reply to Son et al.

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Keywords: Ventricular septal defects • Biocompatibility • Animal model • Closure devices

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We agree that using two different breeds of pigs might lead to different tissue reactions which limit the comparison between these two different patches. To have a valid comparison, it would have been more appropriate to compare the tissue reaction of these two patches in the same animal or at least in the same breed.

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