Repair of complete atrioventricular septal defect: close to the moon? From giant leap for the medical community to small steps for our patients

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Keywords: Atrioventricular septal defect • Surgery • Outcomes

Outstanding low mortality has been achieved in the last 10 years for almost all types of congenital cardiac repair. If the 60’s, 70’s and 80’s represented times of exceptional innovation, with a giant leap from palliative surgery to the attainment of successful complex repairs, then the current era is a time of perfectionism, with goals of achieving very low morbidity and mortality, in incremental small steps, through technically ideal surgery and advances in overall intensive care and patient management.

This era of advancement through ‘small steps’ is probably less impressive and less rewarding for surgeons but just as important for our patients as well as from a global health management point of view.

In this issue of the European Journal of Cardio-Thoracic Surgery, the article from the Melbourne group (Outcomes of repair of complete atrioventricular septal defect in the current era) [1] perfectly illustrates the tremendous improvement that has been achieved in the modern era. Their data, according to the latest literature, support the idea that low mortality is now a goal that has been attained by the most experienced centres.

There still remain, however, challenging questions, perfectly emphasized by Xie et al. [1], that need to be addressed in order to improve the outcome of complete atrioventricular septal defect (cAVSD) repair: timing of surgery; choice of technique for repair of the left atrioventricular valve (LAVV) and influence of postoperative LAVV function on long-term outcomes.

The authors have tried to define the ideal time for repair based on an analysis of postoperative morbidity, postoperative LAVV function and need for later reintervention. We completely agree with the technical difficulties of manipulating delicate valvular tissue that may be encountered in the youngest patients (<3 months of age) and also with the potential progressive preoperative atrioventricular valve deterioration over time (patients >6 months). One has to keep in mind, however, that for a significant proportion of cAVSD patients, timing of repair is not the surgeon’s choice but is determined by the patient’s clinical status (which in turn partly correlates with the cAVSD anatomical variants). In practice, repair may sometimes be clinically necessary with timing dictated by these circumstances, irrespective of what the optimal theoretical timing of LAVV repair might be.

Moreover, defining the best time for surgery should also take into consideration preoperative morbidity and mortality which are often far from negligible, especially when compared with operative mortality.

We are convinced that these symptomatic patients definitively benefit from early repair (1–3 months), which is thus a better option than pulmonary artery banding or delayed repair. The surgical community currently makes impressive and continuous technical improvements, evolving from basic surgery to almost microsurgical techniques that can be used for the repair of the most fragile and tiniest valve, something that ‘the 5-0 sutures repair era’ could not offer.

We, thus, progressively come to the key point of complete AVSD surgery, which is the quality of atrioventricular valves repair [2, 3], probably a more important factor than the timing of surgery (though the latter consideration can influence the former). In their article, Xie et al. emphasize the difficulty of achieving such a goal, with a significant rate of moderate-to-severe postoperative LAVV regurgitation (16% at discharge), probably very disappointing when taking into consideration the correlation between postoperative LAVV regurgitation and the need for reoperations. It is our firm belief that to answer the question of ‘how to improve outcomes in these patients’ is in fact to answer the question of ‘how to achieve the best surgical repair, especially of the LAVV’.

Again, we are convinced that cAVSD repair is probably a more demanding procedure than a standard arterial switch and that the surgeon is more important than the technique itself, confirmed by the observation that different surgical techniques are not associated with different outcomes. Nevertheless, there are common principles required to achieve an optimal repair, with whichever technique is used, which are determined by an understanding of the mechanisms of postoperative AVV regurgitation.

If no regurgitation is noted prior to surgery but is then found afterwards, one can accept the idea that the surgical repair itself can result in regurgitation: something that could explain the absence of a real correlation between preoperative and AVV
postoperative regurgitation. These unexpected early postoperative leaks are mainly the consequence of two mechanisms which, in an ideal repair, are minimized or eliminated by the most experienced and delicate surgeons:

(i) Postoperative restrictive leaflet motion due to either chordae restriction by the VSD patch or leaflet tissue reduction due to attachment to the VSD patch.

(ii) Distortion of the zone of apposition by stitching the two hemi- valves to the VSD and ostium primum patches (anteroposterior and right-to-left partition).

Even if the water test does not show any regurgitation, the height of the zone of apposition can be dramatically reduced compared with the preoperative situation due to minor distortion and tissue reduction. The subsequent evolution of this zone of apposition is, therefore, modified by surgery, compared with the slow evolution in non-operated patients.

Closing the cleft at initial repair is, in our mind, a way to avoid the possibility of developing later regurgitation at the zone of apposition, particularly for patients with underdeveloped leaflet tissue. The current report from Melbourne, highlighting that patients in whom the zone of apposition had been closed had a worse outcome (more postoperative regurgitation and more reoperations), is thus extremely confusing because the authors only decided to close the cleft when the water test was not satisfactory. In these cases, early mechanisms of regurgitation (restrictive leaflet motion and distortion of the zone of apposition) might be involved and it is probably more accurate to first reposition or to resize the VSD patch before closing the cleft; just as in mitral valve repair, it is mandatory to achieve mitral competence prior to implanting the annular ring.

If we look now at the long-term evolution of a competent postoperative LAVV, the appearance of regurgitation is usually seen at the zone of apposition if not closed earlier (something that has been described for non-operated partial AVSD) or in cases of cleft dehiscence. Secondary or iterative cleft closure is the treatment of choice, highlighting its role in obtaining LAVV competence. This fact should emphasize the necessity of cleft closure at initial repair without being focused on the potential stenosis that can rarely occur in specific patients (e.g., those with anomalous subvalvular apparatus).

Another mechanism of late central regurgitation is the LAVV annular remodelling with a significant increase in LAVV diameter by lateral wall displacement [4] rather than septal shift, which might justify the frequent use of commissural or posterior annuloplasty to avoid this specific type of later regurgitation; this translates into the philosophy at our unit of: ‘immediate postoperative mild stenosis with no regurgitation is better than initial mild regurgitation’.

The LAVV is the heart of cAVSD repair, though controversy over cleft closure persists. We believe in the beneficial effects of systematic cleft closure with very limited risks. As such, its closure represents a small step for the surgeon but a giant leap for LAVV competence!

REFERENCES