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ORIGINAL ARTICLE

Permanent complete heart block following surgical closure of isolated ventricular septal defect

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KEYWORDS

Congenital heart defect;
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Abstract A serious complication after surgical closure of ventricular septal defect (VSD) is complete heart block (CHB). It continues to be a leading cause of long-term postoperative cardiac morbidity despite all surgical technical improvements, especially with performance of more surgical procedures in increasingly younger patients.

Objective: This study was undertaken to determine the incidence of early postoperative CHB requiring pacemaker implantation following surgical repair of isolated ventricular septal defect, and try to identify possible procedural risk factors.

Design: Prospective study design.

Setting: Multicenter study: Ain-Shams University Hospital, Aboul Reesh Student Hospital and The National Heart Institute.

Method: We reviewed four hundred patients who had a surgical repair of isolated VSD from 2009 to 2011.

Results: 14 out of 400 patients (3.5%) developed permanent post-operative CHB. All; but one; underwent closure of large perimembranous VSD. CHB patients had a significant lower body weight (8.36 vs. 12.68 kg, $p < 0.01$), longer ACC time (42.6 vs. 36.4 min, $p < 0.01$), longer CPB time (75.4 vs. 67.4 min, $p < 0.01$) and longer hospital stay (19 vs. 8.3 days, $p < 0.01$). Tricuspid valve detachment was performed in 28 patients (7%) and was associated with a higher incidence of CHB (7% vs. 3.75%, $p = 0.6$).

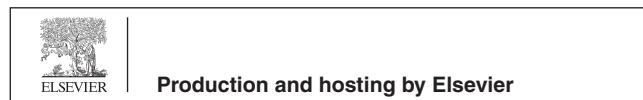
Conclusion: Large perimembranous VSD and lower body weight appear to be independent risk factors for permanent CHB following the surgical closure of isolated VSD.

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Introduction

Permanent complete heart block (CHB) is a significant complication of intracardiac repair for congenital heart disease. It refers to post-operative heart block that does not spontaneously revert to the pre-operative rhythm (usually

within 10 days of the operation). Treatment entails lifelong dependence on an artificial pacing system requiring replacement at least once every decade. The relevant part of the cardiac conduction system in this setting is the atrioventricular (AV) node and the bundle of His. The AV node is located in the triangle of Koch within the floor of the right atrium and continues as the His bundle which penetrates the right fibrous trigone to emerge at the base of the non-coronary aortic cusp in the upper interventricular septum. The bundle of His (and its divisions) is located within the ventricular septum and is thus vulnerable to injury during surgical procedures on the ventricular septum. CHB has been reported after repair of lesions such as isolated ventricular septal defect (VSD), atrioventricular septal defects (AVSD), VSD in the setting of conotruncal anomalies {tetralogy of Fallot (TOF), double outlet right ventricle (DORV), and others} and subaortic stenosis (SAS).

The risk of post-operative permanent CHB has decreased since Lev and others [1–3] delineated the course of the conduction tissue in various types of congenital cardiac malformations of the ventricular and adjacent atrial septa. In earlier reports [4] of 1971, the risk of surgical CHB was as high as 25%. Improved surgical techniques and better understanding of the anatomy of the conduction tissue in various congenital cardiac anomalies have reduced this risk to 1–4% in the current era [5]. Anderson et al. reported a risk of 0.7% for closure of isolated VSD [6]. It is however a real risk that both the surgeon and his potential patient must confront.

Unlike other centers worldwide, the incidence of early post-operative heart block for congenital heart disease in Egypt has not been reported in a large study. This study was undertaken to determine the incidence of early postoperative CHB requiring pacemaker implantation following surgical repair of isolated ventricular septal defect, and try to identify the possible procedural risk factors.

Patients and methods

This is a multicenter prospective observational study of 400 consecutive patients who underwent elective open heart operations for isolated ventricular septal defect between December 2009 and December 2011 at 3 centers: Ain-Shams University Hospital, Aboul Reesh Student Hospital and The National Heart Institute. Patients with associated complex cardiac anomalies, previous VSD surgery, and those with chronic arrhythmias were excluded. Data collection included preoperative age and body weight, history of arrhythmia, preoperative 12 lead electrocardiogram and echocardiogram, operative details including surgical details, ischemic time and cardiopulmonary bypass time and postoperative invasive arterial blood pressure continuous monitoring, central venous pressure measurement, and peripheral and core temperature continuous monitoring. Continuous ECG monitoring was performed during the entire ICU stay with Drager Infinity Vista XL monitors. When arrhythmias were detected on the ECG monitor, this was also documented with the standard 12 lead ECG. In case temporary pacing was required, the patient's own rhythm was checked at 12-h intervals by shortly turning off the pacemaker and recording the patient's ECG for decision regarding the continuation of

pacing therapy. Before hospital discharge, a 12-lead ECG was routinely done. In the case of arrhythmia in the postoperative course, the patient also had a 24-h Holter recording before discharge.

Results

During the period under review, 400 patients underwent a surgical closure of isolated VSD. There were 236 (59%) males and 164 (41%) females. Their age ranged from 0.2 to 14 years (mean of 3 ± 3.29 years). The body weight ranged from 3 to 53 kg (mean of 12.14 ± 8.46 kg). According to echocardiogram, VSD type distribution was 328 (82%) perimembranous (PM), 36 (9%) muscular (MS), 4 (1%) PM + MS and 32 (8%) subarterial. VSD closure was done through trans-atrial approach in 390 (97.5%) cases, trans-pulmonary in 6 cases, combined trans-atrial trans-pulmonary in 2 cases and trans-aortic in 2 cases.

Tricuspid valve detachment (TVD) was done in 28 (7%) patients according to surgeon's preference. All VSDs were closed with patch (pericardial or synthetic). Patch stitching was continuous, interrupted, or combined (continuous and interrupted). Mean aortic cross clamp (ACC) time was 37 ± 5 min (range 27–55 min) and mean cardiopulmonary bypass (CPB) time was 68 ± 6 min (range 58–90 min). Mean ICU stay was 3 ± 2 days (range 1–22 days) and mean hospital stay 9 ± 3 days (range 6–30 days). 10 (2.5%) patients died, the most common cause of mortality was LCOS.

14 patients (3.5%) developed postoperative CHB for more than 14 days ending in insertion of PPM. None of them showed clinical or electrocardiographic (ECG) evidence of complete heart block before surgery. All were in sinus rhythm pre-operatively. Intra-operative rhythm was sinus as shown by continuous ECG monitoring until the institution of cardiopulmonary bypass. The clinical data and the intracardiac anatomy of their defects confirmed at operation are shown in Table 1. All but one of the VSDs of patients developing post-operative permanent CHB were perimembranous in location (only one was muscular type) and all were large defects (>50% of the patient's aortic root).

Temporary epicardial pacing was routinely employed in all patients after separation from cardiopulmonary bypass before the heart fully regained normal rhythm and chronotropy from the effects of cardioplegia and hypothermia. For most patients this was necessary for less than 24 h. For patients with post-operative CHB, attempted cessation of this temporary pacing revealed bradycardia and hypotension. Continuous ECG monitoring in the Intensive Care Unit post-operatively showed CHB. Temporary pacing was then continued with the expectation of spontaneous resolution within 7–10 days.

CHB patients had a significant lower body weight (8.36 vs. 12.68 kg, $p < 0.01$), longer ACC time (42.6 vs. 36.4 min, $p < 0.01$), longer CPB time (75.4 vs. 67.4 min, $p < 0.01$) and longer hospital stay (19 vs. 8.3 days, $p < 0.01$). None of the CHB patients with PPM regained AV conduction during the hospital stay of a mean of 19 days (range 16–23 days). We also noticed a higher incidence of CHB with TVD vs. non-TVD group (7% vs. 3.75%, $p = 0.6$) Table 2.

Table 1 Clinical data for CHB patients with PPM insertion.

| | Age (yrs) | Weight (kg) | VSD Type | TVD | ACC (min) | CPB (min) | ICU stay (days) | Hospital stay (days) |
|----|-----------|-------------|-----------|-----|-----------|-----------|-----------------|----------------------|
| 1 | 0.7 | 6 | PM Inlet | – | 40 | 75 | 3 | 21 |
| 2 | 0.7 | 6.5 | Ms In/Out | – | 45 | 80 | 3 | 20 |
| 3 | 1 | 5.5 | PM | – | 38 | 70 | 3 | 16 |
| 4 | 1 | 5 | PM In/Out | – | 40 | 70 | 4 | 19 |
| 5 | 1.5 | 7 | PM | – | 45 | 75 | 3 | 17 |
| 6 | 2 | 7.5 | PM | – | 42 | 70 | 3 | 18 |
| 7 | 2.4 | 8 | PM Inlet | – | 40 | 70 | 4 | 21 |
| 8 | 2.4 | 7.5 | PM | + | 48 | 90 | 3 | 20 |
| 9 | 2.5 | 9 | PM Inlet | – | 35 | 70 | 3 | 19 |
| 10 | 3 | 9.5 | PM In Out | – | 48 | 75 | 4 | 16 |
| 11 | 3.5 | 10 | PM | + | 45 | 85 | 3 | 20 |
| 12 | 5 | 10 | PM | – | 45 | 75 | 3 | 17 |
| 13 | 5 | 10 | PM | – | 40 | 75 | 4 | 23 |
| 14 | 7 | 15.5 | PM Inlet | – | 45 | 75 | 3 | 18 |

Table 2 Comparison between CHB and non-HB patients.

| Variable | | Age | Weight | ACC (min) | CPB (min) | ICU Stay (days) | Hospital stay (days) |
|----------------|------|-------|--------|-----------|-----------|-----------------|----------------------|
| Permanent CHB | Mean | 3.69 | 8.36 | 42.57 | 75.36 | 3.29 | 18.93 |
| | SD | 1.88 | 2.67 | 3.86 | 6.03 | 0.47 | 2.3 |
| Non-HB | Mean | 3.23 | 12.68 | 36.41 | 67.4 | 2.69 | 8.28 |
| | SD | 3.51 | 9.07 | 4.64 | 5.71 | 1.98 | 1.73 |
| <i>p</i> Value | | 0.329 | 0.0000 | 0.0000 | 0.0000 | 0.168 | 0.0005 |

Discussion

It is salutary to note that, when Lillehei first performed the surgical closure of VSDs, conventional wisdom suggested that there was no single axis responsible for atrioventricular conduction, despite the earlier exemplary description of the atrioventricular bundle provided by Tawara. It was Lev, in fact, who clarified the course of the atrioventricular bundle in hearts with the typical VSD, with Copenhaver and Truex shortly thereafter showing the distinction of the course of the conduction axis in the setting of what we now call perimembranous as opposed to muscular defects opening to the inlet of the right ventricle. Indeed, on the basis of these early studies, the disposition of the atrioventricular conduction axis has been well established for all the various types of VSDs, and surgeons are well aware of the significance of these anatomical findings [6].

Tremendous advances in the surgical management of congenital heart disease (CHD) have been achieved over the past half century. Nevertheless, conduction system injury continues to be a leading cause of long-term postoperative cardiac morbidity, especially with performance of more surgical procedures in increasingly younger patients. The cause of the block is either placement of the suture through the conduction system or hemorrhage from the suturing in the vicinity of the conduction system [7]. In the earlier reports of 1971, the risk of surgical CHB was as high as 25%. Improved surgical techniques and better understanding of the anatomy of the conduction tissue in various congenital cardiac anomalies have reduced this risk to 1–4% in the current era [5]. Anderson et al. suggest that in light of existing knowledge of the atrioventricular conduction axis, the risk of iatrogenic complete heart block with VSD closure should be less than 1% [8].

CHB following surgical repair may be transient or permanent. Transient CHB in this setting generally reverts to the sinus rhythm within the first 7–10 days after surgery though recovery several months later is not unknown. By definition then, permanent CHB is not expected to exhibit spontaneous recovery. Weindling et al. have shown that 63% of patients with CHB after surgery regained atrioventricular conduction in the first post-operative month; in the majority (97%) this occurred in the first 9 days after surgery [9]. Belated recoveries after permanent pacemaker implantation occur in a small but significant number of patients. Batra et al. noted that after insertion of a pacemaker, recovery of atrioventricular conduction occurred in 7 of 72 patients (9.6%) at a median of 41 days (range 18–113 days) after the initial cardiac operation [10].

This report focused on the permanent post-operative CHB which has a significant post-operative impact in terms of morbidity and cost. This study shows the commonest indication for surgery on the ventricular septum relates to the presence of an isolated VSD. In our experience, permanent complete heart block occurred in 14 of 400 (3.5%) patients which compares favorably with others [5,11,12]. All the patients who developed complete heart block had large perimembranous VSDs, except one with muscular (inlet/outlet) type. In a perimembranous VSD, the His bundle is intimately related to the posterior and inferior margins of the defect making it vulnerable to operative trauma; the margin of error can be very small indeed. It is generally held that surgical heart block is due to direct operative trauma to the conduction tissue. In all 14 instances, the QRS morphology was of broad-complex type, indicating possible injury to the distal His bundle or the bundle branches.

It is also noteworthy that all the patients who suffered surgical heart block in our study had ventricular septal defects

that were larger than 50% of the aortic orifice. The designation of a VSD as large or small is arbitrary but useful. In this report, a large VSD is arbitrarily taken to be greater than 50% of the size of the aortic root. It may be inferred that the larger the defect, the more intimate and extensive is the relation between the conduction tissue and the VSD margin. Titus et al. [13] noted that when the defect is small relative to the size of the heart, no part of the conduction system was intimately related to the defect, apparently because the defect is too small to impinge on the tissues normally carrying conduction fibers.

Perimembranous defects on the other hand, especially those involving the inlet are more vulnerable to CHB after repair. Because conclusive differentiation between perimembranous and muscular outlet defects is not always possible on gross morphological examination, it is recommended that the crest of the muscular septum and fibrous tissue in the posterior rim of the VSD should be avoided during closure of the defect [14,15]. Our technique of closing these defects follow established surgical principles [16] based on the anatomy of these defects using relatively oversized patches, suturing to the right ventricular septal surface at least 2 mm away from the rim of the VSD, especially around the postero-inferior border of the defect with placement of superficial sutures in this area.

Several studies have shown that the risk factors for early postoperative arrhythmias were lower body weight, younger age, longer cardiopulmonary bypass time, higher surgical complexity, and residual defect, yet these studies included a wide range of congenital defects and arrhythmias [17,18]. In our study, lower body weight was a significant predictor of PPM insertion for CHB. Younger age failed to show significance in patients who needed PPM insertion, where this complication was reported at 5 and 7 years old. Tucker E et al. showed that neither the younger age in months as a continuous variable, nor the lower weight for age meet significance as an independent risk factor for PPM placement after repair of PM VSD [19].

Anderson et al. at Great Ormond Street Hospital for Children; who reported 0.7% iatrogenic permanent CHB; showed no attribution for age and body weight, and commented that despite the knowledge of disposition of the atrioventricular conduction axis has been well established for all the various types of VSDs, and surgeons are well aware of the significance of these anatomical findings., occasional instances of iatrogenic complete heart block continue to occur after surgical closure of VSD, either because of unexpected biological variations, or because of unawareness of the known disposition of the atrioventricular conduction axis in particular circumstances; reported in his cases; in combined perimembranous and muscular defects, and straddling and overriding of the tricuspid valve [6].

Despite our results seem to agree with the literature with both aortic clamp time and cardiopulmonary bypass time slightly longer in patients with CHB, yet in a relatively simple procedure as isolated VSD the difference was in the range of few minutes, suggesting other surgical contributing factors and manipulation around the AV node area with instruments, which are reported to cause CHB [20].

Successful transatrial closure of a VSD requires adequate visualization of the margins of the defect in order to avoid residual VSD, creation of heart block, and distortion of the tricuspid valve. Clinical application of temporary tricuspid valve

detachment for complete visualization of ventricular septal defect through right atriotomy is not new. In 1961, Hudsepeth and associates first described this adjunct [21]. Opinions vary among surgeons regarding usefulness of TVD as an adjunctive for transatrial closure of VSD. Consequently, its frequency of use varies widely, from never being used to always being used. TVD allows complete visualization of VSD at the cost of destroying valve architecture, and skepticism involves the balance between this benefit and the potential risks of complications. Others have expressed concern at the potential risk of postoperative TR and heart block due to atrioventricular node injury caused by mechanical stress or incidental tear with retraction of the incised leaflet [22].

In our study, TVD was performed only in 28 patients (7%) at the surgeon's discretion. Although PPM insertion was higher in the TVD group, yet that was not statistically significant, which puts us in a neutral position with literature reports. Gaynor et al. reported the use of TVD in 21% in isolated VSDs that did not result in an increased surgically induced heart block. Actually, there was a trend toward a lower incidence of reoperation for residual VSD and less severe TR in the patients in whom TVD was used suggesting that the improved visualization of the defect after detachment of the septal leaflet results in more accurate suture placement with less distortion of the tricuspid valve apparatus [21]. Sasson et al.; using TVD in 23% of isolated VSD repair; showed that patients who had indications for tricuspid valve detachment who actually had detachment performed during repair had fewer postoperative surgical complications; including heart block; as opposed to patients who fulfilled the criteria but did not undergo detachment [23].

TVD percentage in our study is low compared to both studies, reflecting relative conservative use of TVD in our practice that would need a larger study to focus on all its aspects including criteria for use, technique of detachment and reinsertion of the leaflet, associated residual shunting and tricuspid regurgitation. As expected, our patients with CHB had a prolonged hospital stay, but that did not affect their ICU stay time, as patients with CHB were hemodynamically stable with a temporary pacemaker and could be transferred to the regular ward on a monitored bed awaiting either resolution of HB or PPM insertion.

Study limitations

The main objective of our study was to evaluate early post operative CHB rather than follow up. Our follow-up period was only during the period of hospitalization. The preliminary results and possible delayed heart block, need to be confirmed by a larger trial and by long-term follow-up. To limit our population, we included only patients with isolated VSD with no associated complex anomalies. Selection of patients for tricuspid leaflet detachment was at the surgeon's discretion, depending on the anatomy perceived on the operating table, without objectively defining the indications for employing the technique. The major limitation of the present evaluation was that by far not all variables occurring during or early after pediatric cardiac surgery could be assessed. There are inherent individual differences that could not be addressed, even in operations for the same defect done by the same surgeon. We recommended longer periods of follow up, larger study group, establishing a registry of all operative cases.

Conclusion

Conduction system injury continues to be a leading cause of long-term postoperative cardiac morbidity despite all surgical technical improvements, especially with performance of more surgical procedures in increasingly younger patients. We reported permanent complete heart block in 3.5% patients, which compares favorably with the current era (1–4%). CHB was neither a primary nor a contributing factor for mortality in our study, yet it has a significant impact on morbidity involving psychological, procedural and economic, presented by PPM insertion, prolonged hospital stay, parental concern regarding the need for repeated pulse generator replacements and the risk of LV cardiomyopathy requiring a close long term follow-up not only of their cardiac rate and rhythm, but also the ventricular function. Lower body weight appears to be an independent risk factor for early postoperative CHB and PPM insertion. The dominant risk factor for PPM insertion was large perimembranous VSD. Instances of iatrogenic complete heart block continue to occur after surgical VSD closure, either because of unexpected biological variations or because of unawareness of the disposition of the atrioventricular conduction axis in particular circumstances.

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