When Pacing a Heart is No Longer "Just Another Option"!

To Editor,

Importance of pacing in postoperative pediatric cardiac patients is well-known and epicardial wires are usually left at the end of cardiac surgery; albeit this practice is neither mandatory nor necessarily useful.

We recently encountered a clinical scenario which best fits a variant of atrial standstill.^[1]

A 5-month-old male child presented with congestive failure. He had situs solitus, levocardia, atrioventricular concordance, large muscular ventricular septal defect amounting to single ventricle, double outlet right ventricle, anterior malposition of outlet septum, and normal ventricular function. The preoperative investigations were normal, including a 12 lead electrocardiogram (ECG).

He underwent pulmonary artery banding. After staying stable initially, he gradually slipped into profound low cardiac output syndrome (LCOS). The systemic arterial blood pressure (SBP) was low with rising blood lactates, metabolic acidosis, and acute oliguria necessitating multiple vasopressors (dopamine, noradrenaline, vasopressin, and adrenaline) to maintain perfusion. Peculiarly, as the LCOS developed and progressed, the heart rate remained inappropriately low for age (90–104/min).

Echocardiography (ECHO) revealed mildly depressed ejection of the left ventricle, flow acceleration across the band (gradient around 40 mmHg), and dilated atria with no evidence of "atrial kick" on recordings of the mitral valve waveforms indicating the absence of atrial activity. ECG done concomitantly showed relative bradycardia and absent P waves with a regular R-R interval.

Despite aggressive strategy, including empirical Eltroxin and Hydrocortisone, perfusion remained poor. Given the relative bradycardia and ECHO impression of an "atrial standstill," it was decided to re-open the sternum and pace the heart or to proceed to mechanical circulatory support. After initiation of atrial pacing, gradually, the SBP improved and filling pressure fell; the sternum was closed, and medical management was continued. The hemodynamics stabilized over the next 48 h with a simultaneous improvement in renal function. Over the next 24 h, ECG reverted to normal sinus rhythm, and pacing was discontinued. The child eventually recovered and was discharged.

The atrial contribution to ventricular filling may account for up to 40% of ventricular filling in adverse scenarios^[2] and loss of this atrial kick may result in profoundly low cardiac output.^[3] The diagnosis of atrial standstill entails absence of P waves, absence of A waves, supraventricular type QRS, immobility of atria, and inability to pace the atria.^[1] Usually classified as partial or total and as intermittent or permanent, a temporary variant has also been described where the atria can be paced.^[2] Drugs (digitalis and quinidine), hyperkalemia, acute myocardial infarction, familial inheritance, and open-heart surgery are well-known factors associated with atrial standstill.^[3,4]

Our case presented an extremely rare scenario; certainly, to reopen the sternum following a closed heart procedure merely to place epicardial wires may seem like resorting to extraordinary measures! Nonetheless, it worked. The etiology of initial deterioration of our case is not obvious and labeling it as an "atrial standstill" is arguably conjectural.

Our case highlights the atrial contribution in ventricular filling and therefore, critical dependence of the cardiac output on the heart rate.

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Conflicts of interest

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