Cardiac Resynchronization Therapy in the Young Patient: Current Status and Future Directions

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Editorial Comment

Cardiac resynchronization therapy (CRT) in young patients with congenital cardiac disease is in its infancy and is just now beginning to be employed. The article by Moak et al.[1] points out a growing problem and, potentially, a dilemma in this group of patients—when and how should CRT be employed? This article raises many pertinent questions that need to be addressed. To the present time, articles addressing the use of resynchronization therapy in children have all been retrospective studies and for the most part involved a small number of somewhat diverse patients. The largest study to date was reported by Dubin and colleagues.[2] They retrospectively reviewed the records of 103 patients at multiple institutions, who had undergone placement of resynchronization devices. Unfortunately, the centers used multiple and diverse criteria for deciding when resynchronization therapy was to be employed and multiple means of assessing outcomes. Both articles provide an excellent beginning from which to develop this type of therapy in children, but should not be looked upon as providing definitive criteria for such use.

The current major issues with the use of CRT in this patient population are similar to those posed in the adult population and relate to indications for resynchronization therapy, specific measures of ventricular dyssynchrony, and implant methodology as it relates to this unique group of patients. However, the answers may be very different. To date, indications for consideration of ventricular resynchronization have included advanced heart failure unresponsive to medication together with some indication for left ventricular dyssynchrony with or without a wide QRS complex. Patient symptomatology has been variable, ranging from being listed for transplantation to NYHA class I.[2] Use of ejection fraction alone may not be a good indicator for CRT need. The degree of symptoms and the likelihood of patient improvement must be balanced by the risk of CRT placement,[2] and quantitative indicators of a declining clinical state should be used.

In all series reported to date, there has been a significant number of nonresponders. The question then arises as to the difference between responders and nonresponders. Dubin et al.[2] have suggested that perhaps the nonresponders were children who had less advanced dysfunction and higher initial ejection fractions, and therefore failed to show much improvement. Better patient selection criteria hopefully will improve this.

Measures of dyssynchrony employed are also highly variable. Some authors have looked only at QRS duration,[2,3] which may not be indicative of ventricular mechanical dyssynchrony. Moak et al.[1] utilized only a single m-mode echocardiographic measure of the time from peak posterior motion of the interventricular septum to the peak anterior motion of the left ventricular free wall. Unfortunately, such a measure only evaluates one small area of the heart and may or may not be representative of overall ventricular dyssynchrony. Newer methods utilizing 2- and 3-dimensional echocardiography and use of Doppler tissue imaging techniques evaluate multiple areas of the ventricular myocardium and may give a clearer picture of the overall degree of dyssynchrony.[4] Use of QRS duration alone should probably not be used as conduction disturbances produced by chamber enlargement, and surgical procedures may affect QRS duration with a variable impact on ventricular dyssynchrony. Clearly, additional investigation into the measurement and quantification of left ventricular and, potentially right ventricular dyssynchrony are needed in this young patient population who may or may not have characteristics similar to the adult population.

Implant methodology and the selection of an appropriate left and right ventricular pacing site have not been well studied. In most series reported, placement of the left ventricular electrode either transvenously or epicardially has for the most part been nonspecific. Additionally, there has been little investigation at the time of implant of the effect of different electrode sites. To improve CRT results, one must be much more specific as to the site of left ventricular stimulation and as recent work has shown, perhaps utilize techniques during implant to assess the appropriateness of the electrode placement, thus allowing potential movement of the electrode at the time of implant to fully optimize the degree of resynchronization.[5] Finally, appropriate timing intervals including AV delays and RV versus LV delays have also not been well studied, with little information presented in the current published series.

While not directly discussed in the article by Moak et al.,[1] the concept of primary utilization of biventricular pacing to prevent ventricular dyssynchrony with resultant potential dysfunction arises. At the current time, there does not appear to be any substantial evidence to support the use of this approach. While some work addresses myocardial changes following right ventricular-only pacing,[6] extrapolation from histologic changes to myocardial function is speculative at best. If one confines the patient population to only those patients with congenital complete heart block that require pacing, only a small number will ultimately develop myocardial dysfunction.[7] In addition, it appears that patients who ultimately develop ventricular dysfunction are those who require early pacing, which would imply an early and more severe degree of cardiac compromise leading to early

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ventricular pacing. In most series, the number of patients with congenital complete heart block who will develop ventricular dysfunction appears to be less than 10%.[7] It is potentially possible that young patients with congenital complete heart block and early signs of ventricular dysfunction might benefit from early biventricular pacing, but this is currently also speculative.

The use of CRT in children and young patients is an entirely new treatment modality that requires a very different approach. Its use is just beginning and it is through work such as that presented by Moak and colleagues that our knowledge of this therapy and intelligent selection of patients who would benefit from it have improved. Detailed, prospective studies evaluating the relationship between ventricular dysfunction, dyssynchrony, and the use of resynchronization therapy are clearly needed. In addition, one must be cautious when combining patients with differing disease states into a single group, as the answers to the questions posed may well be quite different for diverse disease states. This is an exciting new therapeutic area in patients with congenital heart disease. Either inappropriate overuse or underuse of this modality would be unfortunate, and it is hoped that well-designed prospective clinical trials will prevent this.

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