

CORRESPONDENCE

Letters to the Editor

Cardiac Resynchronization Therapy

Two recent studies published in *JACC* (1,2) suggest that the indication for cardiac resynchronization therapy (CRT) may be extended to heart failure patients with normal QRS width (<120 ms) and evidence of mechanical dyssynchrony identified by tissue Doppler imaging.

Cardiac resynchronization therapy is an electrical therapeutic approach to treat an electrical conduction delay that in turn causes mechanical failure, mainly within the left ventricle. This pathophysiologic concept has been elaborated and proven in numerous well-designed and well-controlled animal experiments, in hemodynamic studies in humans, and in mathematical models (3–6) but it is now challenged by a new approach that completely disregards the traditional electrical basis for resynchronization therapy and focuses solely on mechanical dyssynchrony.

Although it seems logical to look closer for the presence of mechanical dyssynchrony, by means of echocardiography or other imaging modalities, before recommending resynchronization therapy, I believe that we cannot completely ignore the electrocardiogram (ECG) at this time. Some important issues have not been addressed by both studies: how to deliver ventricular pacing in patients with normal electrical conduction and how to achieve sufficient ventricular capture to resynchronize ventricular contraction. It would be interesting to learn more about the intrinsic PR interval before pacemaker implantation and how the atrioventricular delays were set to ensure ventricular capture without interruption of atrial filling. How many patients had evidence of complete ventricular capture by the ECG at rest and in how many patients was mechanical resynchronization achieved by fusion between intrinsic activation and the pacing stimuli?

Both trials (1,2) are certainly very intriguing and stimulating, but they are also clearly limited by their design, mainly due to the lack of a control group with inactivated pacing. Thus, the 2 studies can only serve to initiate larger randomized multicenter trials before the ECG can be completely abandoned as a selection criterion for resynchronization therapy.

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REFERENCES

1. Yu CM, Chan YS, Zhang Q, et al. Benefits of cardiac resynchronization therapy for heart failure patients with narrow QRS complexes and coexisting systolic asynchrony by echocardiography. *J Am Coll Cardiol* 2006;48:2251–7.
2. Bleeker GB, Holman ER, Steendijk P, et al. Cardiac resynchronization therapy in patients with a narrow QRS complex. *J Am Coll Cardiol* 2006;48:2243–50.
3. Kass DA, Chen CH, Curry C, et al. Improved left ventricular mechanics from acute VDD pacing in patients with dilated cardiomyopathy and ventricular conduction delay. *Circulation* 1999;99:1567–73.
4. Auricchio A, Stellbrink C, Block M, et al. Effect of pacing chamber and atrioventricular delay on acute systolic function of paced patients with congestive heart failure. *Circulation* 1999;99:2993–3001.
5. Blanc JJ, Etienne Y, Gilard M, et al. Evaluation of different ventricular pacing sites in patients with severe heart failure: results of an acute hemodynamic study. *Circulation* 1997;96:3273–7.
6. Claus P, Bijmens B, Breithardt OA, Herbots L, Sutherland GR. Why ischemic hearts respond less to cardiac resynchronization therapy. A modeling study. In: Magnin IE, Montagnat J, Clarysse P, Nenonen J, Katila T, editors. *Functional Imaging and Modeling of the Heart*. Berlin: Springer, 2003:287–94.

Reply

We thank Dr. Breithardt for his interest in our study on cardiac resynchronization therapy (CRT) in patients with a narrow QRS complex (<120 ms). Our report demonstrates that CRT appears to be beneficial in 33 patients with narrow QRS complex and severe left ventricular (LV) dyssynchrony on tissue Doppler imaging, with similar improvement in symptoms and comparable LV reverse remodeling as in patients with wide QRS complexes (1). Our results are confirmed by the data of Yu et al. (2) in the same issue of the *Journal* and are in line with 2 earlier studies (3,4), bringing the total of included patients to 118 (2–4). However, none of the studies to date included a control group of narrow QRS patients with inactive pacing, which can be considered as a limitation. We totally agree with Dr. Breithardt that the promising results of CRT in narrow QRS patients in these initial studies will now need confirmation in larger multicenter randomized studies before the current selection criteria can be refined.

A second point raised by Dr. Breithardt is that patients with a narrow QRS may potentially have a shorter PR interval compared to patients with a wide QRS, which may lead to a higher number of patients with fusion of intrinsic activation and the pacing stimuli. However, a narrow QRS complex in these patients may not necessarily be associated with a short PR interval. The (degree of) fusion during CRT is often difficult to assess, and the incidence of fusion in patients with a wide QRS complex is also unknown. As indicated, we cannot exclude that some patients may have had fusion between intrinsic activation and pacing stimuli, but still the application of CRT resulted in a significant improvement in clinical and echocardiographic parameters comparable to patients with a wide QRS complex.

In conclusion, we agree with Dr. Breithardt that there is a clear need for multicenter randomized trials to confirm the promising initial results of CRT in heart failure patients with a narrow QRS complex.