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LETTERS TO THE EDITOR

Regional Ventricular Wall Motion Abnormalities in Tricuspid Atresia After the Fontan Procedure: Flawed Methodology May Lead to a Spurious Finding of Hypokinesia

We read the report of Akagi et al. (1) with interest. Their analysis of ventricular wall motion shows apparent systolic hypokinesia in patients with tricuspid atresia after the Fontan procedure. They kindly refer to some of our previous work and point out the inability of their two-frame method to study sequential changes throughout the cardiac cycle. It is this fundamental methodologic weakness that we believe needs to be discussed in more detail before their data can be interpreted.

In our study (2), we performed frame by frame analysis of segmental wall motion throughout the whole of the cardiac cycle. Our results showed that patients develop grossly *incoordinate* wall motion after the Fontan procedure. These abnormalities were particularly marked during the period of isovolumetric relaxation. Indeed, in some areas of the ventricle, inward wall motion continued throughout the period of isovolumetric relaxation, reaching a peak only after the atrioventricular (AV) valve opened. In other regions, peak inward wall motion occurred well before the time of global minimal cavity dimension, and outward motion was complete before the AV valve opened. Truly hypokinetic areas were seen in only 20%. We have subsequently demonstrated that abnormal intraventricular flow measured using pulsed wave Doppler echocardiography commonly occurs during isovolumetric relaxation in these patients, and this is almost certainly related to intraventricular pressure transients consequent to this incoordinate movement of the ventricular wall (3).

The impact of incoordinate relaxation is seen both immediately and in the longer term. In our study of the changes occurring during the immediate transition to the Fontan circulation (4), we found that in addition to a marked reduction in cavity dimension and increased wall thickness, there was similar Doppler evidence of incoordination and a marked prolongation of the time constant of isovolumetric relaxation, a well described sequel of incoordinate wall motion (5). Slowed relaxation will prolong the period of isovolumetric relaxation, and we were subsequently able to show a highly significant direct relation between prolongation of isovolumetric relaxation time and reduction in early rapid filling in the later postoperative patients (6).

These data shed light on the concern that we have with regard to the methodology used in the Akagi et al. study (1). The two-frame method is unreliable in the presence of temporally nonuniform wall motion. We presume that end-diastole was measured at the time of the Q wave of the electrocardiogram. The definition of end-systole is not given and is clearly difficult to precisely define in patients with systolic and diastolic incoordination. As we mentioned earlier, end-systole in some segments may precede end-systole in other segments by 100 to 200 ms (10 or more angiocardiographic frames). It is predictable therefore that a single-frame analysis may demonstrate areas of *apparent* hypokinesia. Depending on the defined point of end-systole, these segments may represent areas that have yet to reach their peak inward position or, conversely, represent areas that have already begun to move out, peak inward wall motion having been achieved earlier. Clearly, such apparent hypokinesia in turn affects the measurement of global indexes of ventricular performance, such as ejection fraction, and it is not surprising that they were related in the patients whom Akagi et al. studied.

Summary. We believe that the two-frame method described by Akagi et al. (1) cannot adequately describe the highly abnormal wall motion characteristics of these post-Fontan ventricles, and the systolic hypokinesia they describe may be spurious. Our data show that the predominant abnormality is *incoordinate* relaxation of the ventricular wall, which in turn prolongs the time constant of relaxation and the isovolumetric relaxation time and leads to reduced early rapid filling. Indeed, it was these abnormalities of diastolic, not systolic, function that were the strongest predictor of poor exercise performance in our study of patients late after the Fontan procedure (7). We strongly believe that the analysis of ventricular wall motion requires sequential data throughout the cardiac cycle, with well defined reference points concerning the timing of cardiac events, so that misinterpretation can be avoided.

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The Ischemic Threshold Does Vary Under Differing Exercise Conditions

In their recent article, Benhorin et al. (1) conclude that the ischemic threshold during exercise is fixed and does not vary with the type of exercise. Although their data are supportive on the basis of the two exercise protocols utilized, inadequate consideration is given to other published studies (2–5).

Four published studies from our laboratory demonstrate variability of the ischemic threshold during exercise (2–5). In two of these studies, exercise was combined with radionuclide imaging that confirmed the presence of myocardial ischemia even in the absence of ST segment depression (4,5). The purpose of the studies was to

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