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LETTERS TO THE EDITOR

Angled Interventricular Septum on Echocardiography

Bernstein et al. (1) convincingly demonstrate that the angled interventricular septum on echocardiography is really dependent on one's "point of view." We would like to amplify their observations by our findings and approaches to this issue. 1) In most cases, septal angulation is predictable from the M-mode trace when: a) the septum is positioned more anteriorly than is the anterior aortic wall (rather than being equidistant from the echo source), and b) septal echoes are discontinuous (due to tangential motion of the septum across, rather than along, the axis of the M-mode beam). These signs serve as reliable clues to septal angulation and the necessity for two-dimensional echocardiographic correlation. 2) As shown in the authors' Figure 1, not only are septal thicknesses and ratios distorted, but the left ventricular dimensions are also routinely overestimated. 3) Although use of the most cephalad interspace retaining the perpendicular transducer position is the logical conclusion to obviate this problem, one is frequently forced to utilize a more inferior interspace than is ideal. We have found such to be the case in many elderly and pulmonary patients, in whom retrosternal air restricts observations to a lower acoustical window, and septal angulation is frequently observed (or produced, as the case may be). Finally, this work underscores the necessity to view M-mode and two-dimensional echocardiography as joint, complementary diagnostic modalities as are the bell and diaphragm of the stethoscope. Combining the strengths of these modalities certainly allows for the most complete cardiac diagnosis and best chance to avoid errors in M-mode interpretation (1,2).

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References

1. Bernstein RF, Child JS, Shah PM. Angled interventricular septum on echocardiography: anatomic anomaly or technical artifact? J Am Coll Cardiol 1983;2:297–304.
2. Kessler KM, Pefkaros K, Sequeira R, Myerburg RJ. Quantitation and significance of horizontal cardiac motion in M-mode and two dimensional echocardiography. Am J Cardiol 1982;50:520–34.

Correction

In the article entitled "Parasympathetic Effects on Electrophysiologic Properties of Cardiac Ventricular Tissue" by Rardon and Bailey (J Am Coll Cardiol 1983;2:1200–9), the last line on page 1204, "treated and control animals (Fig. 5 and 6). The negative," was inadvertently printed at the bottom of the left hand column. It should have been printed after the last line in the right hand column, "was similar in resperine-pretreated, tyramine-". This error causes considerable confusion in understanding these two paragraphs, corrected versions of which follow here.

Thus in the intact heart, evidence exists both for cholinergic inhibition of norepinephrine release from sympathetic terminals and a myocardial cell receptor-mediated muscarinic cholinergic effect. Multiple sites for myocardial cellular effects are postulated. Muscarinic cholinergic agonists inhibit adenylate cyclase activity and increase cyclic GMP. Additional intracellular effects of muscarinic cholinergic stimulation are currently being investigated, but their physiologic importance is uncertain.

Rardon and Bailey (71) demonstrated that muscarinic cholinergic stimulation produced a direct (adrenergic-independent) negative chronotropic response in isolated guinea pig ventricular strips. The chronotropic response to physostigmine was similar in reserpine-pretreated, tyramine-treated and control animals (Fig. 5 and 6). The negative chronotropic response was not diminished by reserpine pretreatment or reserpine pretreatment plus propranolol, nor was it accentuated during simultaneous treatment with tyramine. Carmeliet and Ramon (28) demonstrated that the electrophysiologic effects of acetylcholine in sheep cardiac Purkinje fibers were not antagonized by the simultaneous treatment of the preparations with propranolol or phentolamine.