

transiently entrains the tachycardia must necessarily enter via the excitable gap. Otherwise, the reentrant circuit will act as a protected focus. Clearly, then “. . . access is not blocked by the wave front emerging from the circuit.”

The argument that transient entrainment indicates a macroreentrant circuit was considered by us early on. However, we (4) and others (5) have now demonstrated transient and interrupted atrioventricular nodal reentrant tachycardia. Clearly, this is not an anatomically large or macroreentrant circuit. Therefore, transient entrainment does not imply, ipso facto, that the reentrant circuit is large. Perhaps, as per the above point, the presence of fusion beats in the electrocardiogram demonstrated by two of the three criteria (constant fusion beats during rapid pacing except for the last captured beat and progressive fusion) may imply the presence of an anatomically large or macroreentrant circuit.

Finally, we agree that “. . . the occurrence of entrainment of ventricular tachycardia proves that not all ventricular tachycardia is caused by ‘protected localized reentry’.” In fact, MacLean et al. (6) have previously stated this.

ALBERT L. WALDO, MD, FACC
RICHARD W. HENTHORN, MD
VANCE J. PLUMB, MD, FACC
WILLIAM A. H. MACLEAN, MD, FACC
Birmingham, Alabama

HEIN J.J. WELLENS, MD, FACC
PEDRO BRUGADA, MD
*Clinical Electrophysiology Laboratory
University of Limburg
Department of Cardiology
Annadal Hospital
Maastricht, The Netherlands*

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Exercise-Induced Atrioventricular Block

Woelfel et al. (1) claim that exercise-induced ischemia was unlikely to have caused heart block in their patients. Our recent article (2) on exercise-induced bundle branch block (versus atrioven-

tricular [AV] block) indicates that the majority of these patients have coronary artery disease, some with demonstrable ischemia concurrent with the development of conduction disturbance. The fact that there was no electrocardiographic evidence of ischemia in the patients with AV block does not necessarily rule this out. It is conceivable that ischemia could be present without apparent electrocardiographic evidence (apart from conduction disturbance!), since ST segment changes generally become evident at higher ventricular rates—hence the 85 to 90% of maximal predicted heart rate criterion for diagnostic exercise tests. Yet in two of their patients, diagnostic ventricular rates were not achieved because of the development of AV block. Thus, ischemia could have affected the proximal conducting system, without being manifested by ST segment changes because of the minimization of ischemia by ventricular rate protection, but with the ischemia manifesting as AV block. In Case 2, perhaps a thallium perfusion exercise test would therefore have been preferable to the gated blood pool scan that was cited as normal.

The difference in onset rates of AV block during exercise versus atrial pacing in Patient 3 is discussed by the authors, who conclude that the reason is unclear. Chapman's report (3) on an athlete with intermittent left bundle branch block occurring at differing onset rates during exercise versus atrial pacing suggested autonomic modulation of intraventricular conduction, with both adrenergic and cholinergic influences accounting for this phenomenon. While the authors suggest that the His-Purkinje system is relatively insensitive to autonomic modulation, there is evidence to the contrary (4-6) so this explanation is plausible.

Finally, a minor point regarding the authors' comment in connection with this rate onset difference. Their statement, that variability in onset rates of rate-dependent aberrancy with serial tests has been noted, is unrelated to their discussion at that point, which deals with variability in onset rates of AV block between differing modes of rate increase (that is, pacing and exercise) rather than serial exercise tests.

VICTOR S. WAYNE, MBBS (HON), FRACP
*16 Dunraven Avenue
Toorak (Melbourne)
Victoria 3142, Australia*

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Reply

We agree that the absence of chest pain or ST segment change does not exclude the presence of myocardial ischemia. However,