it ever occurs. Second, no mapping of the ventricles was performed to provide any data which could be used to analyze this possibility. Third, it is really not relevant to the points of the report regarding transient entrainment and interruption of a tachycardia with rapid pacing.

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## Transient Entrainment and Interruption of Ventricular Tachycardia With Rapid Atrial Pacing—II

We read with great interest the report by Waldo et al. and the accompanying editorial about entrainment of ventricular tachycardia (1,2). Several additional points about this phenomenon merit emphasis because of their importance to the mechanisms of ventricular tachycardia. We agree with Brugada and Wellens (2) that entrainment per se does not imply reentry. However, if focal impulse formation were hypothesized to be the mechanism responsible for an entrainable ventricular tachycardia with fusion, a sector of unidirectional block out of the focus would have to be present for the entraining wave to access the site of abnormal impulse formation and accelerate the next beat. Reentry with an excitable gap, as indicated by Waldo and his colleagues, is a simpler and better explanation for ventricular tachycardia in the patient they describe.

More can be said about the reentry mechanism in their case. The point or points at which the wave of excitation from the reentrant circuit engages the rest of the myocardium must be separate from the path over which the entraining wave front gains access to the circuit, so that access is not blocked by the wave front that just emerged from the circuit. Furthermore, a large area of physiologic or anatomic block between the muscle depolarized by the entraining stimulus and that depolarized by the wave front emerging from the site of reentry is required for fusion to be manifest during entrainment. If this were not the case, a contribution to the activation sequence by the entrained impulse would be inapparent.

The simplest explanation for the findings of Waldo et al. is reentry around a large anatomic or physiologic barrier, or macroreentry. This mechanism would provide both for facile engagement of the reentry circuit by an entraining impulse and for fusion. We believe that ventricular tachycardia subject to entrainment with fusion constitutes strong evidence of macroreentry (3). Other explanations for ventricular tachycardia of this nature are contrived and complicated. Finally, the occurrence of entrainment of ventricular tachycardia proves that not all ventricular tachycardia is caused by "protected localized reentry" (4).

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## *Reply*

Waldo et al. have emphasized that transient entrainment can only be established by fulfilling one of three criteria: 1) constant fusion beats during rapid pacing at a constant rate except for the last captured beat; 2) progressive fusion (constant fusion beats at different rapid pacing rates but different degrees of fusion at the different rate); and 3) interruption of the tachycardia by rapid pacing associated with localized conduction block to a site followed by activation of that site from a different direction and with a shorter conduction time by the next pacing impulse. If one of these criteria can be fulfilled, Waldo et al. (1,2) have suggested this is best explained by reentry. Similarly, Brugada and Wellens (3) stated that "unless otherwise proved, demonstration of transient entrainment of tachycardia using the criteria of Waldo et al. is a very easy way to demonstrate that reentry is the underlying mechanism of the arrhythmia" (3). Thus, both Waldo et al. (1,2) and Brugada and Wellens (3) emphasize the point that one must be able to demonstrate one of the proposed criteria in order to demonstrate transient entrainment and, therefore, reentry. Furthermore, they have emphasized that with available data, only reentry can satisfactorily explain the observations that fulfill any of the three proposed criteria.

We agree it is likely that ". . . a large area of physiologic or anatomic block between the muscle depolarized by the entraining stimulus and that depolarized by the wave front emerging from the site of reentry is required for fusion to be manifest during entrainment." However, we do not understand clearly their statement that "the point or points at which the wave of excitation from the reentrant circuit engages the rest of the myocardium must be separate from the path over which the entraining wave front gains access to the circuit, so that access is not blocked by the wave front that just emerged from the circuit." The point is that to obtain transient entrainment, there must be an excitable gap in the reentrant circuit. The wave front from the pacing impulse which

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.

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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 atrioventricular [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.

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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,