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Letters to the Editor:

Dual Septal Pathways

To the Editor:

The case reported by Gonzalez et al.[1] describes a patient with atrioventricular reentrant tachycardia utilizing the AV node of the antegrade limb and a left sided posteroseptal accessory pathway as the retrograde limb of a reentrant circuit. The participation of this accessory pathway as the retrograde limb of the circuit is well established by the retrograde atrial activation sequence during tachycardia and by the change in the ventriculoatrial intervals during left bundle branch block aberration. However, the authors suggest that a second anteroseptal accessory atrioventricular pathway participates as a bystander during reciprocating tachycardia, functioning in the antegrade direction. Their most compelling argument for the presence of this accessory pathway is the observation of a typical "concertina" progression during spontaneous sinus rhythm as illustrated in their Fig. 6. The typical concertina affect is characterized by a gradual prolongation of the AH interval, usually observed during spontaneous increase in the sinus rate or incremental atrial pacing while at the same time the interval from the onset of the atrial electrogram to the onset of the delta wave remains constant. Their Fig. 6 shows a relatively constant AH interval associated with a shortening of the interval from the His bundle atrial electrogram to the onset of QRS depolarization. This finding would be more compatible with fortuitous ventricular fusion.

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1. GONZALES, R, SCHEINMAN, M M, DESAI, J, KERSH, E AND PETERS, R W: Enhanced atrioventricular nodal conduction in a patient with dual extranodal pathways. J Electrocardiol 13:85, 1980

The Author's Reply

To the Editor:

We thank Dr. Klein for his interest and comments regarding our recent manuscript.[1] His comments with regard to the "concertina" effect are well taken and although we cannot exclude the possibility of fortuitously placed PVC's, we think it unlikely that these would occur in precisely this fashion on several occasions. In addition, please note from Fig. 3 that similar type complexes were recorded during the tachycardia. Moreover, patients with WPW may have intermittent preexcitation without obvious change in sinus rate.[2] The reasons for the above are not well understood but could result in a concertina effect with constant AH, shortened HQ and preexcitation.

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1. GONZALEZ, R, SCHEINMAN, M M, DESAI, J, KERSH, E, PETERS, R W: Enhanced atrioventricular nodal conduction in a patient with dual extranodal pathways. J Electrocardiol 13:85, 1980
2. SCHAMROTH, L: The Disorders of Cardiac Rhythm. Blackwell Scientific Publication, Oxford, 1971, p. 238

Anterior Displacement of QRS Loops

To the Editor:

The paper by Piccolo et al.[1] establishes without doubt that anterior displacement of QRS loops may occur as a result of right ventricular conduction disturbances. Anteriorly displaced QRS loops have been a problem in vectorcardiography since its inception in the 1950's. Many individuals without evidence of coronary disease or of right ventricular hypertrophy have presented with anterior loops and have been incorrectly labeled as "dorsal infarction".

It seems likely, however, that left sided conduction disturbances might also, in some circumstances, lead to anterior QRS loop displacement. In our paper[2], two individuals with coronary artery disease limited to the left anterior descending artery presented with prominent anterior forces on ECG and VCG study. Both patients underwent left anterior descending bypass graft procedures for severe angina, and both developed post-operative anterior wall myocardial infarction. In both cases, the prominent anterior forces completely disappeared and were replaced by Q waves.

If the prominent anterior loops in these two individuals had been the result of right ventricular conduction delay, some evidence of such delay should have persisted after anterior infarction. It seems quite possible that the anterior wall of the left ventricle was the site of the conduction disorder, perhaps due to delayed conduction in the mid-septal branch of the left bundle system, and that, following infarction, this myocardium be-