Dual A-V Nodal Pathways and Preexitation

To the Editor:

We have read with interest the article by Zipes and coworkers, "Unusual properties of accessory pathways."

We were particularly intrigued by case 2, where Zipes postulates sustained A-V nodal reentry as one of the mechanisms of paroxysmal supraventricular tachycardia (Type I SVT). The Type 2 SVT in case 2 was felt to reflect antegrade normal pathway, and retrograde Kent bundle conduction.

With extra-stimulus technique, the authors noted that A2-H2 coupling intervals inducing Type 1 SVT were longer than A2-H2 inducing Type 2 SVT. Examination of table 2 reveals that at similar cycle lengths (480 and 490 msec), and at identical A1-A2 coupling intervals (230 msec), two H1-H2 (and of necessity A2-H2) were obtained, which differed by 100 msec.

The longer A2-H2 was associated with induction of Type 1 (A-V nodal reentrant SVT), while the shorter was associated with induction of Type 2 SVT. These findings resemble those recently described in patients with dual A-V nodal pathways and SVT. The sudden increase in H1-H2 with little or no change in A1-A2 is consistent with failure of a fast A-V nodal pathway, with conduction via a slow pathway. Slow pathway conduction allows the fast pathway to recover for retrograde conduction, and sustained A-V nodal reentry results. Type 1 SVT in this patient is consistent with the above findings. We would postulate that Type 2 SVT in this patient depends upon antegrade fast pathway conduction and retrograde Kent bundle conduction. The postulated dual pathways are supported by the demonstration of A-H of 260-300 during Type 1 SVT, and A-H of 75-85 during Type 2 SVT.

We are suggesting that Zipes' case 2 had dual A-V nodal pathways in addition to an anomalous pathway, and that the former were responsible for sustained A-V nodal reentry. Although A-V nodal reentry has been previously reported in patients with preexcitation, this would be the first case with strong documentation of additional dual A-V nodal pathways. The postulated fast A-V nodal pathway could be extra-nodal (James Tract).

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The author replies:

To the Editor:

Based on an analysis of table 2 from our recent study, Denes and Rosen correctly point out that the curve relating A1-A2 and H1-H2 intervals for case 2 (fig. 1) demonstrates the sudden lengthening of H1-H2 intervals previously described and interpreted by them to represent the response of dual A-V nodal pathways. This concept conveys the idea that either the fast pathway or the slow pathway is used for propagation and that A-V nodal conduction time therefore reflects conduction in one or the other, but not both, pathways. Thus, the dual pathway thesis differs from the established concept, functional longitudinal dissociation, because the latter, supported by experimental data, implies that late responses engage both pathways. Certainly, pathways with different functional properties must exist for re-entry to be explained by either hypothesis, but the duality treatise advanced by Denes and Rosen suggests permanent duality not found in the functional longitudinal dissociation concept.

The question must be raised whether the abrupt shift in A-V nodal conduction time may be explained on another basis. It is possible that the effects of summation may account, in part, for sudden lengthening of A-V nodal conduction. At longer cycles, the most rapid A-V nodal conduction may be due to a propagating wavefront engaging the entire A-V node uniformly. If summation plays an important role in human A-V nodal conduction, then block in one pathway

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Pulmonary Diffusing Capacity

To the Editor:

The recent article on 'Pulmonary diffusing capacity in disorders of the pulmonary circulation' by Dr. J. H. Burgess published in the March issue of this Journal prompts us to make a few observations, since our findings on the measurements of Dm and Vc in patients with mitral valve disease are somewhat conflicting with their report. First of all, it is unfortunate that Dm and Vc measurements were not made on all of their patients. It is difficult to accept the statement that 'the values of Vc are regularly normal when Dco is normal or increased when Dco is high.' As a matter of fact, Dr. Burgess himself points out that patients with mitral valve disease are well known to have high Vc in spite of a normal Dco because of a concomitant reduction in Dm. The actual values of Dm and Vc in 10 of their patients in group 2 (table 2) are rather surprising. Although the results of Dm and Vc estimations in mitral valve disease have been extremely conflicting, almost all the studies reported so far show a decrease in Dm as a consistent abnormality. The only exception is the study by Yuba. The Vc has been variously reported as being decreased, normal or increased. Unfortunately, Dr. Burgess does not give the normal values of Dm and Vc in his laboratory, but to us the Dm in 10 patients in group 2 appears to be normal or increased. This is a group of patients with severe mitral stenosis and high PVR where one would have expected the Dm to be considerably decreased.

Almost all the studies reported in literature show that the Vc/Dm ratio is considerably increased in patients with mitral valve disease as compared to normal subjects. It has not been less than 1 in any of the reported studies. An increase in this ratio may occur as a result of increased resistance to diffusion by the alveolar-capillary membrane. Vc/Dm ratio would also be directly proportional to the radius of the capillaries and independent of their number or length. In presence of capillaries of different radii, the Vc/Dm ratio would vary as the geometrical mean of the radius of the capillaries. In all the patients in group 3 and most of the patients in group 2 one would expect the radius of the capillaries to be considerably increased. This would be reflected in an increased Vc/Dm ratio as is borne out by the studies reported earlier. Therefore, the observation of an extremely low Vc/Dm ratio (0.3 approximately) in group 2 patients of Dr. Burgess is rather difficult to explain. Such a low Vc/Dm ratio has never before been reported even in normal subjects.

References

Dual A-V Nodal Pathways and Preexcitation: The author replies:
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