accurate conducted in the field of cardiac electrophysiology, and we accept them as such now. It is only some of the interpretations which we may debate.

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The authors reply:
To the Editor:

Some of the remarks made by Drs. Sherf and James do not accurately reflect our ideas on the Wolff-Parkinson-White syndrome.1–4

1. In our opinion this syndrome can be the result of several possibilities, summarized in our article “Pre-excitation revisited.”3 The heterogeneous rather than uniform causes of this syndrome were stated explicitly in this article.

2. There is no acceptable electrophysiologic evidence presently available to convince us that malfunction of the normally located conduction system can give rise to a WPW complex. Nor do we think that there is electrophysiologic proof that the direction of input of the impulse from the atrium into the AV conduction system determines the spread of excitation through the ventricular conduction system. This is in contrast to the spread of excitation in the isolated rabbit AV junction.5 As for the ventricular conduction system, we referred in our present paper to several articles and to our own experiments in patients with atrial septal defects. In our patients, without the preexcitation syndrome, we never found any differences in QRS-complex configurations in many leads, after right and left atrial stimulation from many endocardial atrial sites, during the standardized procedure.

3. We used “Kent bundle” for semantic reasons and because of its brevity. For us it simply means an AV connection lateral to the normal AV conduction system, not implying a fixed anatomic location. We agree this is not the bundle described by Kent (and which in the hearts he examined may not even have been present6). It is a generalization, which may, therefore, be correctly criticized on historical grounds.

We do not know (and never said we did) the exact location of the anomalous AV connection in patients with WPW, type A. We have postulated in one of our patients a location at the posterior basal part of the ventricular septum.9 Our results obtained during surgery will be published soon. In our present paper we stated that only left atrial premature beats influenced the time relations during the tachycardia and offered the possibility that this could mean a location of the anomalous AV connection close to the AV junction. It is likely that the anomalous AV connections differ in conduction velocity and refractoriness, just as in distance to the AV junction. This is supported by the different frequencies found among our patients during their tachycardias. It is obviously impossible to be accurately informed about the exact proximity of our stimulating electrode to normal and abnormal AV conduction system. It is not surprising, therefore, that no uniform results were obtained in all five patients. The location of the anomalous bundle could be solved by electrophysiologic studies during surgery or of an isolated heart with WPW, type A, and, quoting Hudson, “not by looking at it,” however experienced one might be.7

4. As we have said under point 2, we did not find evidence in the intact heart that the direction of input into the AV junction influences the spread of excitation through the ventricular conduction system. Therefore, at present, we do not think that the presence or absence of specialized conduction in the atria is relevant to the configuration of the WPW complex.

5. We favoured the explanation given for the initiation of tachycardias in patient B because both ways of initiation resulted in tachycardias of exactly the same frequency, suggesting identical tachycardia pathways.

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