Nonparoxysmal Junctional Tachycardia Complicating Acute Myocardial Infarction

To the Editor:

I enjoyed reading the article by Drs. Konecke and Knoebel (Circulation 45: 367, 1972). I would like to raise two questions pertaining to figures 4 and 5. The top of figure 4 shows inverted P waves with a P-R interval exceeding 0.12 in lead II. This rhythm is interpreted by the authors as indicating an A-V junctional rhythm. Although this is certainly a possibility, the other possibility cannot be excluded, namely, that the ectopic focus is actually situated in the lower right or left atrium (lower atrial ectopic focus). The presence of fusion beats by no means excludes this latter possibility. It has now been amply demonstrated by electrophysiologic experiments that lower ectopic atrial rhythms can give inverted P waves with a normal P-R interval in the inferiorly oriented leads. This has now been demonstrated in the dog, primate (unpublished observations by the writer), and in the human.1

The interpretation given for figure 5 is even more difficult to accept without reservation. Again, although P waves with inverted polarity in leads II, III, and aVF usually indicate A-V junctional origin, the statements made above apply equally to this figure. Furthermore, it is more difficult to accept the concept of intranodal dissociation than to accept the likelihood that the inverted P wave is actually originating from the lower part of the atrium.

I am aware that the authors prefer to refer to the impulses originating in the “coronary sinus” as junctional rhythms as mentioned in their reference 5. However, it should be emphasized that ectopic pacemakers exist elsewhere in the lower parts of the atria besides the coronary sinus, and that stimulation of part of the atrium away from the coronary sinus can also give rise to inverted P waves in II, III, and aVF. These ectopic atrial rhythms cannot be obviously called junctional rhythms.

Incidentally, the reference 6 given by the authors must be an error, since it does not seem to be relevant to the paragraph in which it is quoted. Interestingly, the same reference describes inversion of the polarity of the P wave even if atrial activity is initiated in the region of the sinus node, provided intraatrial conduction is disturbed.

Therefore, although I am not disputing the main impact of the conclusions of the authors, I would have liked to see some reservation in the form of a question mark or in the form of an explanation appended to figures 4 and 5 and in the discussion of the article, which is otherwise excellent.

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Reference

His Bundle Electrogram

To the Editor:

The critique of His bundle pacing by Scherlag et al. (Circulation 46: 601, 1972) was interesting and helpful. However, I question the authors’ recommendation that His bundle pacing is necessary for validation of recorded H potentials.

I have the following reservations about His bundle pacing: (1) Bipolar electrodes positioned at the tricuspid valve may record several electrophysiologic events on the same tracing, including atrial, ventricular, His bundle, and right bundle-branch activation.1,2 Pacing of one of these structures does not negate the recording of the others from the same electrodes. Failure to pace one of these structures also does not negate its having been recorded. It is possible to record an H potential without being able to His bundle pace from the same electrodes (Schuilenburg and Durrer’s case 2).3 (2) The technic of His bundle pacing for validation of H potentials necessitates
a stimulus–V interval equivalent to the H-V interval. Pacing of a structure or chamber may produce a latency period between the stimulus and the recorded onset of depolarization in the structure being paced. This latency period varies, depending upon the properties of the electrode-tissue interface. Pacing the His bundle with free-floating electrodes could produce a long latency period. Since the His bundle electrogram is not recorded during His bundle pacing, this latency period cannot be measured. It seems unreasonable to require that stimulus–V interval be equivalent to H-V interval. (3) Other laboratories have reported that pacing through H recording electrodes produces supraventricular beats, ventricular beats, and fusion beats. This also has been our own experience. It is generally possible to select and display those beats suggestive of His bundle pacing. We are doubtful whether this constitutes true validation. (4) Narula suggests that slight catheter rotation or movement may be necessary to obtain selective His bundle pacing. Slight changes of position of the recording electrodes may markedly change the nature of electrograms being recorded. Therefore, a slight change in electrode position in order to obtain His bundle pacing would seem to invalidate the validation.

We agree with Scherlag that single or coupled atrial pacing is useful in the delineation of atrial and His bundle activation. However, we prefer a similar means of delineating H and right bundle-branch (RB) activation. Coupled atrial pacing may produce delays between H and RB potentials, often with appropriate ECG changes. It is accepted that functional block may occur in the distal right bundle branch prohibiting this means of validation. The relative amplitudes of recorded atrial and ventricular electrograms may also be helpful in delineating H and RB potentials. Absence of an atrial electrogram with a prominent ventricular electrogram suggests an intraventricular electrogram position, and a likelihood of recording RB activation. Prominence of both atrial and ventricular electrograms suggests an atrioventricular electrogram position and a likelihood of recording His bundle activation. It can also be noted that H potentials occur clearly earlier than RB potentials in normally conducted beats, simplifying delineation of these two potentials. Simultaneous recording of left bundle-branch (LB) potentials may also be useful in selected cases for separating H and RB potentials since proximal LB and RB electrograms are usually simultaneous. The relationship of H, RB, and LB, must of course be correlated with the presence of conduction defects on the surface electrocardiogram.

We agree that His bundle pacing provides useful physiologic information. We would disagree strongly with insistence upon this as a necessary means of validation of His bundle recording.

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